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Dear Reader,

Welcome to the April 2026 issue. As spring blossoms around us, so too does the field of medical science, bringing fresh insights and innovations that shape the future of health and well-being.

In our Feature Story, we mark World Autism Awareness Day on 2 April, a day dedicated to promoting acceptance, support, and inclusion for autistic people worldwide. This article explores mechanisms, diagnosis, and the future of treatment, with a special emphasis on care coordination—an essential pillar in advancing holistic support.

Our Focus Section turns to World Kidney Day, observed this year on 12 March. Celebrating its 20th anniversary, this global movement continues to champion kidney health. The 2026 theme, "Kidney Health for All: Caring for People, Protecting the Planet," underscores the profound connection between human health and environmental sustainability.

In the Epoch Section, we delve into thought-provoking topics such as the off-label use of GLP-1 medications for cosmetic weight loss, exploring the balance between the benefits and risks of this growing trend.

As always, our mission is to inform, inspire, and ignite meaningful conversations about the evolving landscape of medicine and health.

Warm regards,

Dr. Feng Xue
MPH, PhD
Managing Editor, V.Pulse

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by Dr. Feng Xue

Autism Spectrum Disorder: Mechanisms, Diagnosis, and the Future of Treatment

—For the World Autism Awareness Day 2026

Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition characterised by persistent challenges in social communication, restricted interests, and repetitive behaviours. Affecting individuals across all cultures and socioeconomic backgrounds, autism represents a broad spectrum of strengths and difficulties rather than a single uniform condition. Over the past two decades, rapid advances in genetics, neuroscience, digital health, and artificial intelligence have transformed scientific understanding of ASD and reshaped approaches to diagnosis and intervention. This article provides a comprehensive overview of autism, including its definition and epidemiology, underlying biological mechanisms, and the latest developments in diagnosis and treatment as of 2024–2026. Emphasis is placed on the multifactorial nature of ASD, the shift toward earlier and more precise detection, and the growing movement toward personalised and lifespan-oriented care.

Introduction to Autism Spectrum Disorder

Autism Spectrum Disorder (ASD) is defined as a neurodevelopmental condition that emerges early in life and persists across the lifespan. According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), core features include difficulties in social interaction and communication, alongside restricted, repetitive patterns of behaviour, interests, or activities. The term "spectrum" reflects the wide variability in symptom presentation, cognitive ability, language skills, and support needs among autistic individuals.[1]

Globally, the prevalence of autism has risen steadily over the past two decades. In the United States, the Centers for Disease Control and Prevention (CDC) reported that approximately 1 in 31 children aged 8 years were identified with ASD in 2022, compared with 1 in 150 in the year 2000 (**Figure 1**). Similar trends have been observed in many other regions, largely attributed to increased awareness, broader diagnostic criteria, improved screening, and better access to services rather than a single environmental cause.[2,3]

Autism is not a disease in the traditional sense but a neurodevelopmental condition and a form of neurodiversity. Many autistic individuals view their traits as integral aspects of identity, emphasising the importance of respectful, strengths-based approaches to research and clinical practice.[4] Nevertheless, significant challenges remain, particularly for individuals

with co-occurring intellectual disability, limited language, epilepsy, anxiety, or gastrointestinal problems, underscoring the need for continued scientific and clinical progress.[4]

Epidemiology and Clinical Features

ASD occurs in all racial, ethnic, and socioeconomic groups and is diagnosed more frequently in males than females, with a ratio of approximately 3–4:1. However, recent research suggests that autism in females and gender-diverse individuals may be underdiagnosed or diagnosed later in life due to different symptom profiles and compensatory behaviours such as social “masking” or "camouflaging".[2,5,6,7]

The core clinical features include:[8]

- Social communication differences: difficulties with reciprocal conversation, understanding nonverbal cues, and developing peer relationships.
- Restricted and repetitive behaviours: repetitive movements, insistence on sameness, intense interests, or sensory sensitivities.
- Developmental variability: language abilities range from fluent speech to minimal or absent verbal communication.

Many individuals with ASD also experience co-occurring conditions, including attention-deficit/hyperactivity disorder (ADHD), anxiety disorders, depression, sleep disturbances, and epilepsy. This clinical heterogeneity has been a major driver of contemporary research into mechanisms and personalised care.[9]

Biological and Neurodevelopmental Mechanisms of Autism

Genetic Architecture

Autism has a strong genetic basis, with heritability estimates ranging from 50% to 90% across studies. Modern genomic technologies, including whole-exome and whole-genome sequencing, have identified hundreds of genes associated with ASD risk. These include both rare, high-impact mutations and common genetic variants that collectively contribute to polygenic risk.[10]

Current clinical genetic testing can identify a likely genetic cause in approximately 20–25% of individuals with ASD, particularly those with intellectual disability or syndromic features. Importantly, autism is not caused by a single “autism gene” but by the interaction of many genetic factors affecting early brain development.[11]

Brain Development and Neural Circuits

At the neurobiological level, ASD is associated with atypical brain development beginning during prenatal and early postnatal periods. Research has highlighted alterations in synapse formation, neuronal migration, and excitation–inhibition balance within cortical circuits.

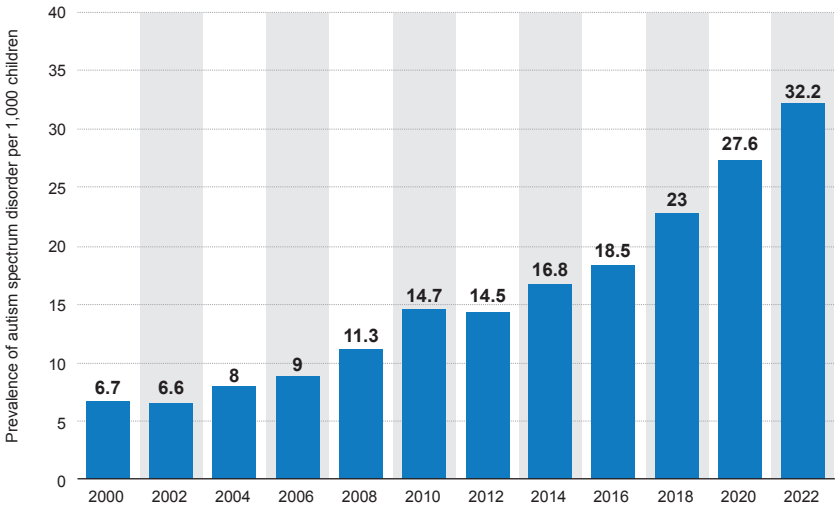

Figure 1. *Prevalence of autism spectrum disorder in the U.S. from 2000 to 2022*[2]

These changes are thought to disrupt information processing relevant to social cognition, sensory integration, and behavioural regulation.[9]

Postmortem and neuroimaging studies have also identified differences in brain regions involved in social processing, such as the prefrontal cortex, temporal lobes, amygdala, and cerebellum, although findings vary across individuals.[12]

Molecular Pathways and Neuroinflammation

Converging evidence suggests that multiple molecular pathways contribute to ASD, including those involved in synaptic signalling, transcriptional regulation, and immune function. Dysregulation of inflammatory and immune-related genes has been observed in both blood and brain tissue from autistic individuals, supporting a role for neuroinflammation in at least a subgroup of cases.[11,13]

The Gut–Brain Axis

An emerging area of research focuses on the gut–brain axis. Many autistic individuals experience gastrointestinal symptoms, and studies have reported altered gut microbiota composition and increased intestinal permeability in subsets of ASD. These changes may influence brain development and behaviour through immune, metabolic, and neurotransmitter-related pathways, although causality remains under investigation.[14]

Advances in Autism Diagnosis

Traditional Diagnostic Approaches

Currently, autism diagnosis relies on comprehensive clinical evaluation, including developmental history, direct behavioural observation, and standardised instruments such as the Autism Diagnostic Observation Schedule (ADOS) and Autism Diagnostic Interview–Revised (ADI-R). While these methods are effective, they are time-consuming, require specialised expertise, and may contribute to delays in diagnosis.[15]

Early Screening and Digital Tools

Routine developmental screening at 18 and 24 months is widely recommended, enabling diagnosis as early as 18 months in some children. Recent years have seen rapid growth in digital health tools designed to support earlier and more accessible screening.[16]

The gaze patterns of individuals with autism can significantly diverge from those of neurotypical individuals. For instance, many autistic individuals might engage in prolonged staring or avoid direct eye contact, behaviours that can be misinterpreted by others as rudeness or disinterest.[17] Artificial intelligence (AI) and machine-learning approaches using eye-tracking, facial analysis, voice patterns, and video-based behavioural data have shown promising accuracy in distinguishing autistic from typically developing children. A 2026 systematic review reported pooled diagnostic accuracy of approximately 85% for machine-learning models based on eye-tracking data, highlighting their potential as objective screening aids.[18,19]

Genetics and Biomarkers

Genetic testing is increasingly incorporated into the diagnostic workup, providing etiological insights and informing medical management. However, despite intensive research, no validated biological biomarkers are currently available for routine autism diagnosis, and experts caution against premature clinical adoption of experimental markers.[11,15]

Current and Emerging Treatments for Autism

Behavioural and Developmental Interventions

Evidence-based behavioural and developmental interventions remain the cornerstone of autism care. Approaches such as Applied Behaviour Analysis (ABA), naturalistic developmental behavioural interventions, speech and language therapy, and occupational therapy have demonstrated benefits in communication, adaptive functioning, and quality of life, particularly when initiated early.[20]

Pharmacological Management

At present, no medication treats the core features of autism. Pharmacotherapy is primarily used to manage co-occurring symptoms such as irritability, aggression, anxiety, ADHD, and sleep disturbances. Recent FDA approval of trofinetide for Rett syndrome—a genetic condition with autistic features—highlights progress in targeted treatments for specific subtypes.[9, 11]

Precision Medicine and Gene-Targeted Therapies

One of the most significant recent shifts in autism research is towards precision medicine. Advances in genomics, brain organoids, and molecular neuroscience are enabling the development of gene-specific and pathway-targeted therapies, particularly for rare monogenic forms of ASD. While these approaches are largely experimental, they represent a major step toward individualised treatment strategies.[11,21]

Microbiome-Based and Novel Interventions

Interventions targeting the gut microbiome—such as probiotics, dietary modifications, and faecal microbiota transplantation—have shown preliminary promise for improving gastrointestinal and, in some cases, behavioural symptoms. However, clinical evidence remains limited, and large, controlled trials are needed before widespread clinical adoption.[14]

ASD Care Coordination

Because of the complexity of autism, the assessment and management should be done with the help of a multidisciplinary team, which consists of a developmental paediatrician, psychiatrist, psychologist, audiologist, occupational therapist, speech therapist, special educator, and social worker (**Figure 2**).[22]

The expanding practice of multidisciplinary care to address the complex nature of ASD suggests that there is a need for a means of coordinating care that transcends the disciplinary distinctions of relevant ASD treatment providers. As ASD services become more specialised, there is a growing need for effective care coordination with providers across the systems of care.[23]

Autism care coordination involves organising services across health, education, and community sectors to support individuals with ASD. It focuses on creating a personalised, family-centred plan to improve quality of life, streamline access to specialists (e.g., occupational therapy and speech therapy), and reduce caregiver stress.[24]

The core components of autism care coordination include: [24]

- Family-Centred Approach: Focuses on family priorities, strengthening partnerships between parents and providers.
- Individualised Planning: Creating a comprehensive plan that acts as a roadmap, addressing unique medical, educational, and social needs.
- Centralised Communication: Ensuring all providers (specialists, schools, therapists) are aligned on the same goals.
- Transition Support: Assisting with transitions, particularly from early intervention to school, or from paediatric to adult care.
- Resource Navigation: Identifying and accessing community resources, financial assistance, and, if necessary, Special Child Care Institutions.

Figure 2. *Multidisciplinary approach managing autism spectrum disorder*[22]

ASD care coordination offers profound benefits for families and providers alike. By streamlining communication among healthcare professionals, therapists, and educators, coordination significantly reduces stress for families who would otherwise face the overwhelming task of navigating complex care systems on their own. This relief allows parents to focus more on supporting their child rather than managing fragmented services. It also drives improved outcomes, ensuring that children receive timely access to therapies and interventions. With a coordinated approach, care plans are more consistent, progress is monitored effectively, and children are less likely to experience delays in receiving essential support. Furthermore, care coordination strengthens continuity of care, preventing gaps in services that can disrupt a child's development. By maintaining a steady flow of communication and planning across providers, children benefit from stable, long-term support that adapts to their evolving needs. In essence, ASD care coordination is not just about logistics—it is about creating a smoother, more reliable path toward growth and well-being for both children and their families.[23]

Ethical Considerations and Future Directions

As scientific understanding of autism advances, ethical considerations are increasingly central. Autistic self-advocates emphasise the importance of research that respects neurodiversity, avoids stigmatisation, and prioritises quality of life and autonomy. Future research directions include improved support across the lifespan, better services for adults and aging populations, and greater inclusion of under-represented groups in studies.[5]

Large international collaborations and multimodal datasets are expected to accelerate discovery of clinically useful biomarkers, refine subtyping, and enhance translation from laboratory findings to real-world care.[15]

Conclusion

Autism Spectrum Disorder is a highly heterogeneous neurodevelopmental condition shaped by complex interactions among genetic, biological, and environmental factors. Over the past decade, and particularly from 2024 onwards, remarkable progress has been made in elucidating its mechanisms, improving diagnostic practices, and developing more personalised interventions. While no cure exists, early identification, evidence-based support, and emerging precision medicine approaches offer growing opportunities to enhance outcomes and quality of life for autistic individuals and their families. Continued interdisciplinary research, ethical engagement, and inclusive policy development will be essential as the field moves forward.

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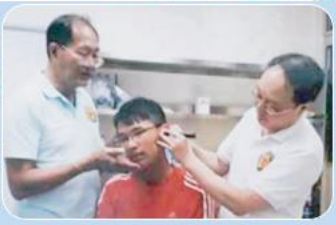

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Member activities

by Dr. Feng Xue

Kidney Health for All: Caring for People, Protecting the Planet

The World Kidney Day 2026 marks the 20th anniversary of a global movement dedicated to advancing kidney health. The 2026 theme, "Kidney Health for All: Caring for People, Protecting the Planet," highlights the inseparable relationship between human kidney health and environmental sustainability. Chronic kidney disease (CKD) affects nearly one in ten people worldwide and is increasingly influenced by environmental degradation, climate change, and inequitable healthcare systems. At the same time, kidney care—especially dialysis—places a substantial burden on natural resources and contributes to greenhouse gas emissions. This article explores the global burden of kidney disease, the impact of environmental factors on kidney health, the environmental footprint of kidney care, and emerging strategies for sustainable, people-centred kidney health systems that benefit both individuals and the planet.[1]

World Kidney Day at 20 Years

World Kidney Day (WKD), established in 2006 by the International Society of Nephrology (ISN) and the International Federation of Kidney Foundations – World Kidney Alliance, has grown into one of the largest global health awareness campaigns. Observed annually on the second Thursday of March, WKD aims to raise awareness of kidney health and reduce the growing global burden of kidney disease.

In 2026, WKD celebrates its 20th anniversary, offering a timely opportunity to reflect on progress while addressing emerging threats. The chosen theme—"Kidney Health for All: Caring for People, Protecting the Planet"—reflects a growing understanding that kidney health cannot be separated from planetary health, social equity, and sustainable healthcare systems.[2]

The Global Burden of Kidney Disease

CKD is one of the most common non-communicable diseases globally, affecting over 850 million people worldwide. CKD significantly increases the risk of cardiovascular disease, premature mortality, reduced quality of life, and economic hardship. Despite its prevalence, CKD often progresses silently, with many individuals remaining undiagnosed until advanced stages.[1,3]

The burden of kidney disease is not evenly distributed. Disadvantaged populations, low- and middle-income

countries, and marginalised communities face higher risks due to limited access to clean water, healthcare services, early screening, and affordable treatments. These inequities directly challenge the goal of "Kidney Health for All."[1,3]

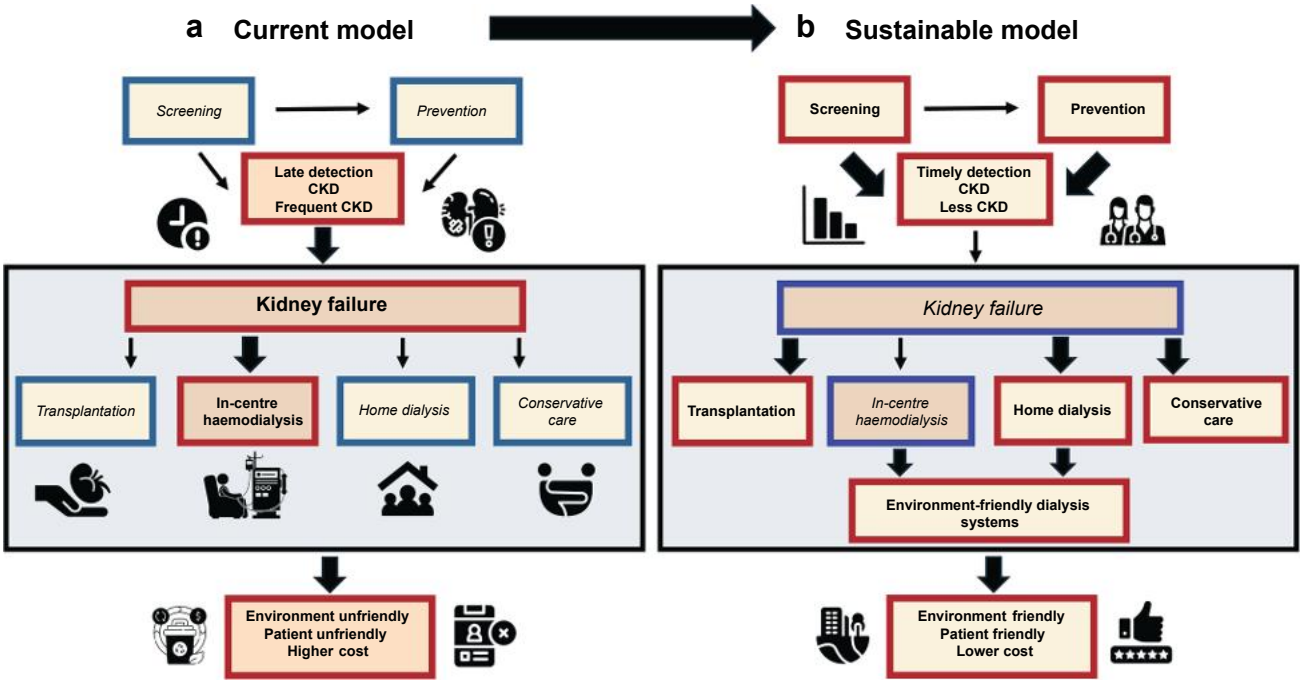
Environmental Determinants of Health

Environmental change is increasingly recognised as a major driver of kidney disease. Rising global temperatures, air pollution, unsafe drinking water, and occupational heat stress contribute to both acute kidney injury and chronic kidney disease.[4]

Extreme heat and dehydration are strongly linked to kidney damage, particularly among outdoor workers in agriculture and construction. Air pollutants such as particulate matter and heavy metals have been associated with declining kidney function, while water contamination exposes populations to nephrotoxic substances. Climate change thus acts as a multiplier of kidney disease risk, particularly in resource-limited settings.[5]

The Environmental Footprint of Kidney Care

Ironically, while environmental degradation worsens kidney health, kidney care itself can harm the environment. Dialysis is among the most resource-intensive medical therapies, requiring large volumes of water and energy while producing significant plastic waste and greenhouse gas emissions.



Adapted from: Vanholder R, et al. Kidney Int. 2026[1]

Figure 1. *(a) Current model of kidney care. (b) Sustainable model of kidney care.[1] Elements that are less or insufficiently prominent in that specific model are in italics with a blue frame; elements that are prominent in the specific model are in bold with a red frame. Yellow background indicates "beneficial for sustainability", and orange background indicates "disadvantageous". Light blue–shaded box with black frame is used for kidney replacement therapy. The thickness of the arrows indicates the degree of impact on the given elements. CKD: chronic kidney disease; home dialysis: peritoneal dialysis and/or home haemodialysis*

The healthcare sector overall contributes more than 5% of global carbon emissions, with dialysis recognised as a high-impact contributor.[1,6]

As the global demand for kidney replacement therapy rises, especially in low- and middle-income countries, the current care model is increasingly unsustainable. This reality underscores the urgent need for eco-friendly, low-carbon kidney care without compromising patient outcomes.[7]

Re-imagining Kidney Care: Prevention and Early Detection

A central message of the 2026 theme is that prevention protects both people and the planet. Early detection of kidney disease—through blood pressure checks, diabetes control, and simple urine and blood tests—can slow disease progression and reduce the need for resource-intensive treatments.[1]

Public health strategies that prioritise healthy diets, physical activity, reduced salt intake, and access to primary care can significantly lower the incidence and progression of CKD. By focusing upstream on prevention rather than downstream on end-stage care, healthcare systems can reduce costs, environmental impact, and patient suffering simultaneously.[1]

Sustainable Kidney Care Models

Sustainable kidney care involves rethinking how services are delivered. Home-based dialysis, transplantation, and conservative kidney management offer benefits for patients and the environment. Home therapies reduce transportation emissions, while kidney transplantation provides superior quality of life with a substantially smaller environmental footprint compared to long-term dialysis (**Figure 1**).[1]

Innovations in "green nephrology," such as water-saving dialysis technologies, recycling programs, renewable energy use in dialysis units, and reduced single-use plastics, demonstrate that environmental responsibility can align with high-quality patient care. Scaling these

solutions globally will require coordinated action among clinicians, policymakers, industry, and patients.[8]

Equity, People-Centred Care, and Planetary Responsibility

"Kidney Health for All" emphasises that sustainable kidney care must also be equitable and people-centred. Access to early diagnosis, essential medicines, dialysis, transplantation, and supportive care remains highly unequal across regions. Environmental sustainability should not become a barrier to care but rather a pathway to more resilient and inclusive systems.[1]

Empowering patients through education, shared decision-making, and community engagement strengthens kidney health outcomes while fostering environmental stewardship. Protecting vulnerable populations from environmental harm is both a health and moral imperative.[2]

Conclusion

The 2026 World Kidney Day theme, "Kidney Health for All: Caring for People, Protecting the Planet," reflects a powerful and necessary shift in global health thinking. Kidney health, environmental sustainability, and social equity are deeply interconnected. Protecting kidney health means addressing climate change, reducing environmental harm, preventing disease, and ensuring fair access to care.

As World Kidney Day marks its 20th anniversary, the global kidney community is called upon to act—by prioritising prevention, embracing sustainable care models, and advocating for policies that safeguard both human health and the planet. The future of kidney health depends not only on medical advances, but on our collective commitment to caring for people while protecting the world we all share.

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by Dr. Feng Xue

Cognitive Reserve: Building Brain Resilience Across the Lifespan

Cognitive reserve describes the brain's capacity to adapt, compensate, and find alternate neural strategies in the face of pathology, stress, or aging. Individuals with greater cognitive reserve can maintain cognitive function despite significant neurodegenerative changes, delaying or reducing the clinical expression of diseases such as Alzheimer's disease, Parkinson's disease, and other neurological conditions. First described in the late 1980s through neuropathological observations, cognitive reserve has since become a central concept in brain health and preventive neurology. Accumulating evidence demonstrates that lifestyle behaviours—including diet, physical activity, sleep, stress management, social engagement, and intellectual stimulation—collectively strengthen cognitive reserve across the lifespan. This article reviews the origins and mechanisms of cognitive reserve, examines its relevance for neurodegenerative disease and life stressors, and discusses the six cornerstone lifestyle interventions identified by Harvard Medical School as essential for maintaining cognitive fitness. For healthcare professionals (HCPs), understanding and translating these principles into patient counselling offers a powerful, non-pharmacological approach to preserving brain health and functional independence.

What Is Cognitive Reserve?

Cognitive reserve can be understood as the brain's ability to "improvise"—to recruit alternative networks, cognitive strategies, or compensatory mechanisms to maintain performance when confronted with damage or increased demand. Rather than reflecting brain size or neuron count alone, cognitive reserve represents a functional adaptability developed over a lifetime of learning, curiosity, problem-solving, and engagement with the environment.[1]

Clinically, the importance of cognitive reserve lies in its ability to explain why individuals with similar degrees of neuropathology can experience markedly different cognitive outcomes. Two patients may show comparable amyloid plaques or vascular lesions on imaging or at autopsy, yet one remains functionally intact while the other develops overt dementia. Cognitive reserve provides a framework for understanding this variability and offers a target for preventive strategies.[2]

Historical Origins of the Cognitive Reserve Concept

The concept of cognitive reserve emerged in the late 1980s, when neuropathologists reported a paradoxical finding: some individuals who had no documented cognitive impairment during life were found at autopsy to have extensive brain changes consistent with advanced Alzheimer's disease. These included significant amyloid deposition, neurofibrillary tangles, and cortical atrophy.[3]

Early work by Katzman and later formalised by Stern and colleagues proposed that education, occupational complexity, and intellectual engagement allowed certain individuals to tolerate a greater burden of pathology before clinical symptoms emerged. Cognitive reserve, as distinct from "brain reserve" (a purely structural notion), emphasised flexible and efficient neural processing rather than passive protection. Since then, cognitive reserve has been supported by epidemiological, neuroimaging, and clinical studies, becoming a cornerstone concept in aging and dementia research.[4,5]

Mechanisms Underlying Cognitive Reserve

Cognitive reserve is not a single biological entity but a multifaceted process involving several overlapping mechanisms:[6]

- Neural Efficiency: Individuals with higher cognitive reserve often show more efficient use of neural

networks, requiring less activation to perform the same cognitive tasks.
- Neural Compensation: When primary networks are compromised, alternate or secondary networks can be recruited to maintain function.
- Cognitive Strategy Flexibility: Higher reserve allows individuals to adopt different problem-solving strategies when familiar ones fail.
- Synaptic Plasticity: Lifelong learning enhances synaptic density and connectivity, supporting adaptability in the face of injury or degeneration.

Importantly, cognitive reserve is dynamic. It can be strengthened or eroded throughout life depending on health behaviours, environmental exposures, and psychosocial factors.[5]

Cognitive Reserve and Neurodegenerative Disease

Dementia and Alzheimer's Disease

The strongest evidence for cognitive reserve comes from Alzheimer's disease research. Numerous cohort studies demonstrate that higher educational attainment, intellectually demanding occupations, and active lifestyles are associated with delayed onset of clinical dementia—even when neuropathological burden is high.[7]

However, an important clinical nuance is that once symptoms do appear in individuals with high cognitive reserve, decline may be more rapid (**Figure 1**). This reflects the fact that pathology has already progressed substantially before clinical thresholds are crossed.[8]

Other Neurological Conditions

Cognitive reserve is not limited to Alzheimer's disease. A robust reserve has been associated with:[9]

- Better functional outcomes after stroke
- Delayed cognitive impairment in Parkinson's disease
- Improved coping and preserved function in multiple sclerosis
- Greater resilience following traumatic brain injury

In each case, cognitive reserve acts as a buffer, allowing patients to maintain independence for longer despite neurological insult.[9]

Cognitive Reserve and Life Stressors

Beyond chronic neurodegeneration, cognitive reserve plays a critical role in responding to acute or subacute challenges. Major stressors—such as surgery, hospitalisation, toxin exposure, or prolonged psychological stress—place increased demands on cognitive systems. In these situations, the brain may need to "shift gears," deploying additional cognitive resources to maintain normal function.[10]

Patients with higher cognitive reserve often show:[10]

- Lower risk of postoperative cognitive dysfunction
- Better stress tolerance and emotional regulation
- Enhanced recovery following illness or injury

For HCPs, this underscores the importance of assessing lifestyle and cognitive engagement when evaluating risk and resilience.[1]

Lifestyle as the Foundation of Cognitive Reserve

While genetics contribute to cognitive reserve, lifestyle factors are the most modifiable and clinically actionable components. Researchers at Harvard Medical School have identified six interdependent cornerstones of brain health and cognitive fitness. Importantly, these elements work synergistically; no single intervention is sufficient in isolation:[11]

Step 1: Eat a Plant-Based Diet

A diet emphasising fruits, vegetables, whole grains, legumes, nuts, and healthy fats supports cognitive reserve through multiple mechanisms:

- Reduction of oxidative stress and inflammation
- Improved vascular health
- Enhanced gut–brain axis signalling

Mediterranean and MIND (Mediterranean-DASH diet Intervention for Neurodegenerative Delay) dietary patterns have been consistently associated with slower cognitive decline and reduced dementia risk. These diets support neuronal health while minimising metabolic and vascular insults that erode reserve over time.

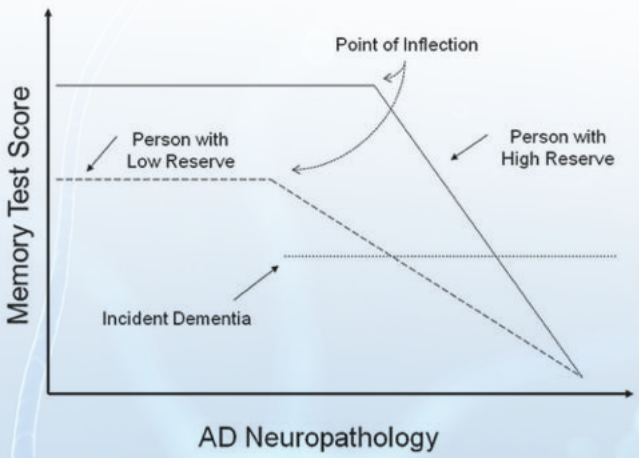

Figure 1: *Theoretical illustration of how cognitive reserve may mediate between AD pathology and its clinical expression.[8] AD: Alzheimer's disease*

Step 2: Exercise Regularly

Physical activity is one of the most powerful modulators of brain health. Aerobic and resistance exercise:

- Increase cerebral blood flow
- Promote neurogenesis in the hippocampus
- Upregulate brain-derived neurotrophic factor (BDNF)
- Improve executive function and memory

Regular exercise not only builds cognitive reserve but also amplifies the benefits of other lifestyle interventions, such as improved sleep and stress reduction.

Step 3: Get Enough Sleep

Sleep is essential for memory consolidation, synaptic homeostasis, and metabolic waste clearance via the glymphatic system. Chronic sleep deprivation is associated with increased amyloid accumulation and impaired attention, learning, and executive function.

For adults, consistent sleep duration and quality are critical for preserving cognitive reserve and preventing long-term cognitive vulnerability.

Step 4: Manage Stress

Chronic psychological stress negatively affects cognitive reserve through sustained cortisol exposure, hippocampal atrophy, and impaired neuroplasticity. Stress management techniques—including mindfulness, relaxation training, and cognitive behavioural strategies—help protect neural integrity.

Clinicians should recognise stress not only as a mental health issue but as a modifiable risk factor for cognitive decline.

Step 5: Nurture Social Contacts

Social engagement stimulates cognitive, emotional, and language networks simultaneously. Strong social ties are associated with reduced dementia risk and better cognitive performance in aging populations.

Isolation and loneliness, in contrast, are linked to accelerated cognitive decline, depression, and increased mortality. Encouraging social interaction is therefore a critical, yet often overlooked, component of cognitive reserve building.

Step 6: Continue to Challenge the Brain

Lifelong learning remains central to cognitive reserve. Activities that challenge the brain—such as learning new skills, engaging in complex tasks, reading, or problem-solving—promote neural flexibility.

Importantly, novelty and difficulty matter. Passive activities that do not require active engagement confer fewer benefits than those that demand sustained attention and adaptation.

A Cohesive, Integrated Strategy

These six cornerstones form a cohesive plan rather than a checklist of independent actions. Improving diet without addressing sleep, or exercising without managing stress, yields limited benefit. Cognitive reserve is built through cumulative, interacting behaviours that collectively enhance brain resilience.[11]

For HCPs, this integrated framework supports a shift from reactive disease management to proactive cognitive health promotion.

Conclusion

Cognitive reserve provides a unifying concept for understanding resilience in brain aging, neurological disease, and life stress. Rooted in decades of clinical observation and research, it explains why some individuals maintain cognitive function despite significant pathology and why lifestyle interventions can meaningfully alter cognitive trajectories.

For HCPs, cognitive reserve highlights the importance of early, sustained lifestyle counselling as a core component of preventive care. By promoting plant-based nutrition, regular physical activity, adequate sleep, stress management, social engagement, and lifelong learning, clinicians can help patients build and preserve cognitive reserve—supporting functional independence and quality of life well into older age.

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by Jasmine Lai

Latent Yet Chronic: Can Herpes Zoster Encourage a Change to the Vaccine Paradigm For a New Age?

Public vaccination efforts have historically focused on infectious diseases against child mortality, and more recently on cancer prevention.[1] Herpes zoster (HZ, commonly known as shingles) primarily affects older adults and immunocompromised individuals, and its vaccine is currently being evaluated for cost-effectiveness and public immunisation policy in Hong Kong.[2] Though traditional vaccination strategy in public health follows single disease focus, accumulating research has revealed how this latent infection has chronic disease implications,[3-7] suggesting potential new ways of understanding the role of vaccines in public health.

An Uncomplicated, Yet Complex Target: 'Single' Disease Perspectives of HZ Vaccination

HZ occurs when cell-mediated immunity falls due to ageing or immunosuppression, allowing latent varicella-zoster virus (VZV) infections to reactivate.[8] Nearly all cases of HZ result in painful skin lesions, and if not treated immediately, it often can lead to post-herpetic neuralgia amongst other potential longer-term complications. Indeed, 95% of individuals over 50 years of age have had prior VZV exposure, and individuals who live to 85 years old have an approximately 50% lifetime risk of developing HZ.[9]

Currently, two vaccines against HZ exist: 1) the live, attenuated zoster vaccine, and 2) the non-live, recombinant zoster vaccine, with the latter showing higher and longer-lasting estimates of efficacy. Yet studies on cost-benefit analyses have varied results, potentially since HZ has low mortality and incidence in later life (equating to lower economic productivity).[10,11] Nevertheless, the rise in HZ likely owes much to ageing populations, increasing chronic disease, the use of immunosuppressive medications, as well as changes in health-seeking behaviour and more comprehensive disease surveillance.[12] Lower VZV circulation from widespread varicella vaccination in childhood immunisation programmes also reduces the exogenous boosting, immunological mechanisms that encourage HZ immunity. That being said, evidence of increased HZ incidence predates mass varicella vaccination, including in Hong Kong (**Figure 1**).[13]

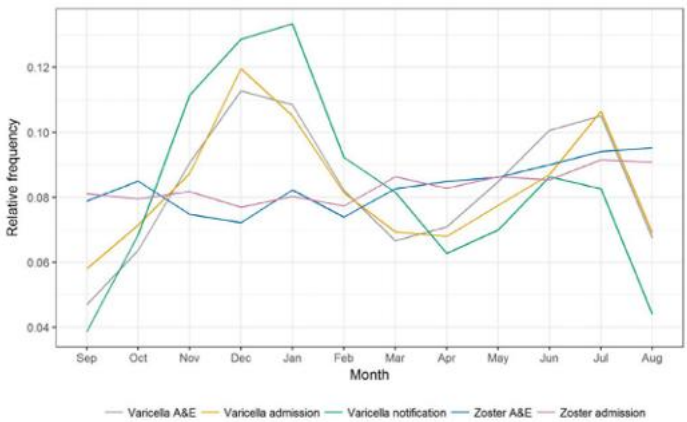

Figure 1: Relative frequency distribution for varicella and herpes zoster cases of all ages by month throughout September 1999 to August 2014 in Hong Kong.[13]

Expanding Vaccination Targets: 'Multi-' Disease Perspectives of HZ Vaccination

An increasing body of work is recognising that the immunological/protective benefits of HZ vaccination, as well as other vaccines, exist beyond their intended target. This suggests the value of moving beyond the single disease perspective, to consider the broader, multi-disease and overall public health benefits of vaccines. HZ affects the nervous system, and studies in mouse and human tissue models have linked herpesviruses to the hallmarks of Alzheimer's disease: amyloid beta production, tau phosphorylation, and neuroinflammation, that arise as a protective response to acute infection, but becomes maladaptive during chronic infections.[3,4,8]

Other studies have compared individuals who chose to be vaccinated or not, to understand the potential link between HZ vaccination and dementia prevention, but are often limited by potential confounding factors such as diet, socioeconomics, physical activity and more.[4] Meanwhile, on top of their previous natural experiments of HZ vaccination and dementia in Wales and Australia, Pomirchy *et al.* (2026) recently published their analysis on their natural experiment of live, attenuated HZ vaccination and dementia onset in Canada, to strengthen the evidence base on any causal links.[4]

Natural Experiment Methodology

Randomised controlled trials (RCTs) remain the gold standard of pharmaceutical interventions, but such design may not be ethical, feasible, or appropriate to investigate policy changes, programme introductions, or structural interventions common to public health initiatives. In particular, confounding factors that are 'controlled' for in RCTs become important contextual factors in the real-world. Natural experiments are more aligned to public health research — tracing back to John Snow's work in understanding London's cholera epidemics in the mid-nineteenth century; they now sit in the realm of 'evidence-based' research, and are popular in other fields such as economics, education, agriculture, social work, engineering, and urban planning. Broadly speaking, natural experiments are on an event (or when 'early adopters' implement an intervention) that is not under the control of a researcher, that divide a population into exposed and unexposed groups.[14]

In their natural experiment study, Pomirchy *et al.* (2026) looked at the introduction of publicly funded HZ vaccination in adults aged between 65–70 years on September 15, 2016 in Ontario, Canada. They also offered HZ vaccination between September 15 to December 31, 2016 to people born in 1945 (71 years old that year). This naturally created three groups: 1) eligible (aged 65–70 years on Sept 15, 2016), 2) eligible for 3.5 months (aged 71 in 2016); and 3) ineligible (born before 1945). The primary analysis focused on comparing those eligible for at least 1 year and 3.5 months to those ineligible, assuming that other than HZ vaccination, individuals are similar/balanced in observed and unobserved characteristics. The outcome of new dementia diagnoses in the 5.5-year follow-up period was identified based on a pan-Canadian electronic health record database for primary care.[4]

Pomirchy *et al.* (2026) found that 4.3% of vaccine-eligible patients (n=8,795) and 5.3% of vaccine-ineligible patients (n=6,997) had a new dementia diagnosis during follow-up, with vaccine eligibility decreasing the probability of receiving a new dementia diagnosis by an absolute difference of 2.0 percentage points (95% CI 0.4–3.5, p=0.012). A regression discontinuity analysis and multiple hypothesis testing for the quasi-randomised design found no biases in the data. Similarly, when controlling for diagnoses of the 15 most common clinical conditions at baseline, vaccine eligibility still reduced new diagnoses of dementia over the 5.5-year

follow-up by 1.9 percentage points (95% CI 0.4–3.4, *p*=0.016). Interestingly, the effect of vaccine on new diagnoses of dementia was significant among female but not among male individuals, shedding further light on the immunological differences between males and females.[4]

The authors also compared the vaccine-eligible cohort in Ontario to other provinces in Canada. Not only did they find near-identical trends in dementia incidence before the vaccination programme was introduced, but incidence of new dementia diagnoses was significantly lower among the vaccine-eligible birth cohort (born between Jan 1, 1945, and Dec 31, 1946) in Ontario than in the same birth cohort in other provinces after the vaccination programme (**Figure 2**). Similar results with the secondary analysis (comparing those eligible for only 3.5 months) support the causal effect.[4]

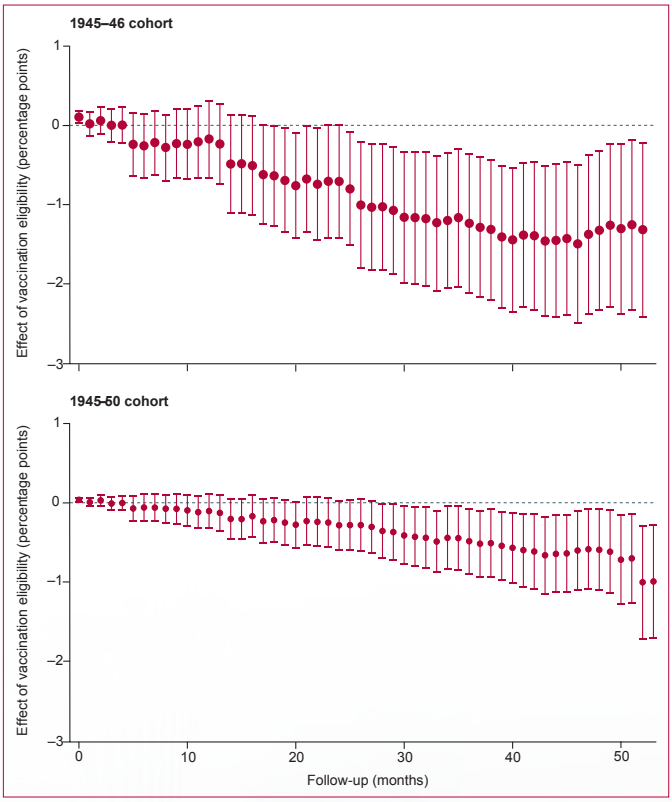

Figure 2: Differences in the probability of dementia between patients from Ontario (HZ vaccine-eligible) and in other provinces. The graphs shows the 1945–46 cohort (born between Jan 1, 1945, and Dec 31, 1946) and the 1945–50 cohort (born between Jan 1, 1945, and Dec 31, 1950).[4]

In a similar vein, the effects of HZ vaccination have been studied in biological ageing. A population-based cohort study of 3,884 adults >70 years with previous HZ vaccination in the US showed improvements in inflammation, innate and adaptive immunity, cardiovascular haemodynamics, neurodegeneration, epigenetic and transcriptomic ageing, as well as composite biological ageing score. The effects were most pronounced within the three-years after vaccination, but had unclear longitudinal effects.[5]

HZ infection and HZ vaccination with the live, attenuated virus have also been linked to cardiovascular health. A retrospective cohort study of 27,093 adults in the US with chronic disease demonstrated a lower risk of stroke (1.61% vs 2.20%; $p<0.05$) and myocardial infarction (1.29% vs 1.82%; $p<0.05$) in vaccinated versus unvaccinated individuals, even when controlling for comorbidities, although differences resulting from socioeconomic status could not be determined.[6] Another longer-term, nationwide study in South Korea of 1,271,922 individuals aged ≥50 years showed reduced cardiovascular disease risk (HR=0.77, 95% CI 0.76–0.78). They also found that the greatest reduction was observed 2–3 years after vaccination, similar to the study on biological ageing, and persisted up to eight years.[5,7] The Korean study also found that the cardiovascular benefits were greater in those from low-income households and rural residents. While the evidence is still unfolding, the effects of HZ vaccination beyond HZ morbidity are worth considering.[7]

A New Vaccine Paradigm: Public Health Perspectives of HZ Vaccination

An active area of discussion in the public health literature involves calling for a new vaccine paradigm: one that considers the wider effects vaccines have on the overall population, rather than the traditional approach of a single vaccine for each infectious target.[3] Benns *et al.* (2020) cited the epidemiological research on childhood vaccines, as well as immunology studies on live vaccines specifically.[3] For example, studies of the measles, mumps, and rubella vaccine from high-income settings demonstrated reductions in respiratory infections amongst others, and that in low-income countries, the reductions in all-cause mortality attributed to measles vaccination were much larger (>50%) than anticipated. Live vaccines also induced innate immune training, producing stronger proinflammatory responses to unrelated antigens; and RCTs comparing live and inactivated vaccines in Bangladesh and Finland found

that the live oral polio vaccine was associated with lower risks of diarrhoea and otitis media than the inactivated polio vaccines.[3]

HZ vaccination's role in public health can be understood with the analogy of the rotavirus vaccine, which has lower efficacy in low-income settings, likely owing to co-infecting pathogens and malnutrition.[15] An RCT showed lower vaccine effectiveness in Malawi than in South Africa (49.4% vs. 72%), yet the number of prevented episodes of severe rotavirus gastroenteritis was greater in Malawi, suggesting that vaccines have extra public health value where healthcare services are more limited.[16] For rotavirus, the interrelated burden of diarrhoea, stunting, and metabolic disease encourages a shift-in perspective to long-term disease prevention.[15] Similarly, HZ's links to pathological ageing and cardiovascular conditions may encourage recognising the role HZ vaccination may play against non-communicable conditions and in addressing health disparities—a public health priority of the past decade with a significance that cannot be understated.[17]

However, usage of the non-live, recombinant zoster vaccine is increasing in Hong Kong and other countries.[8] A modelling study based in Hong Kong by Chan *et al.* (2023) found that mass vaccination with the non-live, recombinant zoster vaccine is expected to considerably reduce public health burden of HZ among individuals ≥50 years compared with no vaccination or the live vaccine, as well as in preventing complications (**Figure 3**).[8] It also supported earlier vaccinations from age 50 rather than later, with the most cases avoided between the ages of 50-59.[8] Whether non-live HZ vaccines have similar off-target health effects remains to be seen.

Can Vaccination Influence Public Health?

A more holistic vaccine paradigm would affect how vaccines are evaluated and how vaccination policy decisions are made.[3] Indeed, attempts have been made to revamp vaccine assessment strategies to capture their tangible and intangible value.[1] It has also opened the realm of vaccinology development to innate immunity-training vaccines.[3] Ultimately, vaccines must be effective for their primary target, but considering their multitude of health effects may lead the way on new public health strategies and potentially influence both societal attitudes and vaccine acceptance.

a)

	ZVL	RZV
Shingles	↓ 47,477	↓ 204,875
Nerve pain	↓ 7,701	↓ 31,949
Shingles around the eye	↓ 1,769	↓ 8,471

b)

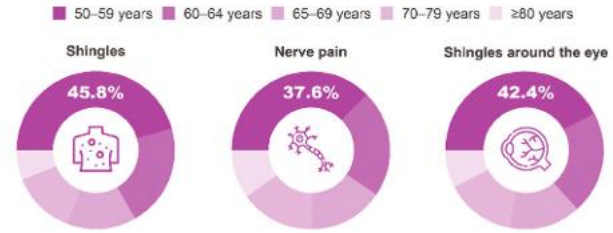

Figure 3: a) Reduction in herpes zoster cases and complications b) Effect of recombinant zoster vaccine on cases and complications in different age groups.[8]

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Abbreviations: A&E, accident & emergency; CI, confidence interval; HR, hazard ratio; HZ, herpes zoster; RCT, randomised-controlled trial; RZV, recombinant zoster vaccine; VZV, varicella-zoster virus; ZVL, zoster vaccine live

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by Dr. Feng Xue

Global Burden and Future Trajectory of Female Breast Cancer: Evidence from the GBD 2023 Study

Breast cancer remains the most common malignancy among females worldwide and a major contributor to morbidity and mortality.[1] Using findings synthesised from the Global Burden of Disease Study 2023 (GBD 2023), an article recently published in the Lancet Oncology reviewed global, regional, and national trends in breast cancer incidence, mortality, and disability-adjusted life years (DALYs) from 1990 to 2023, alongside forecasts through 2050.[2] The data reveal widening disparities between high-income countries—where mortality rates have steadily declined—and low-income and lower-middle-income countries, where both incidence and mortality continue to rise.[3,4,5] Risk factor analysis shows that lifestyle, metabolic, and behavioural determinants together accounted for nearly one-third of breast cancer DALYs globally.[2] Forecasts suggest that incident cases may rise to 3.56 million annually by 2050, driven primarily by population aging and growth.[2] Without strategic investment in early detection, treatment capacity, and equitable health systems—particularly in resource-limited settings—the global breast cancer burden will intensify further.[2] This article highlights the urgent need for coordinated, evidence-based action to address persistent disparities and to strengthen prevention, diagnosis, and treatment pathways worldwide.

Introduction

Breast cancer has long been recognised as the most frequently diagnosed cancer among females.[1] In 2023 alone, an estimated 2.30 million new cases and over 764,000 deaths were recorded worldwide, marking breast cancer as the leading cause of cancer-related DALYs among women.[1,2] Despite improvements in high-income countries, global progress remains uneven.[4,5]

The Global Burden of Disease 2023 Study provides the most comprehensive epidemiological assessment to date, analysing incidence, mortality, and risk factors across 204 countries over a 34-year period and projecting future trends up to 2050. These findings reveal profound inequities in outcomes, shaped by socioeconomic development, health system capacity, risk factor exposure, and demographic change.[2]

Global Trends in Incidence and Mortality (1990–2023)

Rising Incidence Worldwide

Between 1990 and 2023, the global age-standardised incidence rate (ASIR) increased by approximately 16%. In 2023, the ASIR reached 49.3 cases per 100,000 females. However, the global picture conceals deep disparities:[2,4]

- High-income countries had the highest ASIR (75.7 per 100,000), largely reflecting widespread screening and early detection.
- Low-income and lower-middle-income countries (LICs and LMICs) experienced the largest proportional increases in ASIR—147% and 128% respectively—driven by health system deficits, changing reproductive patterns, and rising obesity.

Divergent Mortality Patterns

While incidence has risen across most of the world, mortality trends diverge sharply:[2]

- High-income countries saw a 30% reduction in age-standardised mortality rates (ASMR) since 1990 due to improvements in screening, diagnosis, and therapy.
- Low-income countries, by contrast, experienced a near doubling of mortality, with ASMR reaching 24.1 per 100,000—the largest change among all income groups.

Key drivers for these differences include delayed diagnosis, limited access to treatment, inadequate pathology and radiotherapy services, and affordability barriers.[2]

Disability-Adjusted Life Years (DALYs)

In 2023, breast cancer resulted in 24.1 million DALYs, with more than 90% attributable to premature death (measured as years of life lost [YLLs]). Although high-income countries still account for the largest share of incident cases, nearly half of global DALYs occurred in low- and lower-middle-income settings, illustrating the disproportionate impact of late-stage diagnosis and insufficient treatment capacity (**Figure 1**).[2]

Regional Disparities and Country-Level Patterns

High-Income Regions: Progress with Remaining Gaps

High-income countries demonstrate sustained reductions in ASMR and DALYs.[2] Five-year survival rates in many of these regions exceed 85–90%.[6] Nevertheless, disparities persist within countries:[7,8,9]

- In the US, mortality among Black women remains 1.4 times higher than among White women.
- Indigenous women in Australia and Māori and Pacific women in New Zealand experience significantly poorer survival.

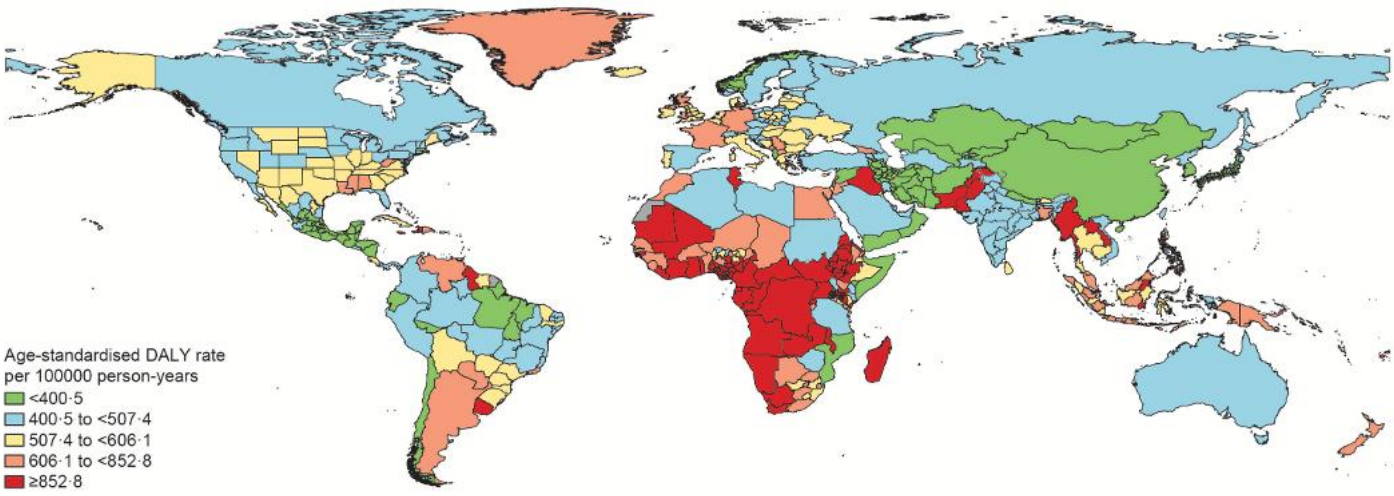
These differences highlight the importance of addressing structural and social determinants of health even in settings with advanced cancer care infrastructure.

Sub-Saharan Africa: Rapidly Growing Burden

Sub-Saharan Africa recorded the highest mortality rates globally.[2] Health system constraints and sociocultural and financial barriers impede timely access to life-saving screenings and diagnosis.[10,11,12] In some African countries, fewer than 30% of cases are detected at early stages,[4] and survival at three years can be as low as 50%.[13]

Middle-Income Countries: Expanding Burden Amid Transition

Upper- and lower-middle-income countries (e.g., India, Brazil, Iran) have witnessed sharp increases in incidence. While some progress has been made in building screening and treatment capacity, gaps persist between urban and rural areas, and between insured and uninsured populations. Economic burden has also surged, with countries such as India projecting a near-doubling of breast cancer–related costs by 2030.[14,15,16]



Adapted from: GBD 2023 Breast Cancer Collaborators. Lancet Oncol. 2026[2]

Figure 1. Global map of age-standardised DALY rate quintiles for breast cancer in 2023.[2] Values presented are for all ages combined. Quintiles are based on DALYs per 100,000 person-years. DALYs: disability-adjusted life-years

Risk Factors and Attributable Burden

Lifestyle and Metabolic Risks

The GBD 2023 study identified six key risk factors contributing to breast cancer DALYs:[2]

- Dietary risks (specifically a diet high in red meat)
- Tobacco use (smoking and second-hand smoke)
- High fasting plasma glucose
- High body mass index (BMI) in adults
- High alcohol consumption
- Low physical activity

In total, these accounted for 28.3% of global breast cancer DALYs in 2023.[2]

Premenopausal vs. Postmenopausal Burden

Risk factor impacts varied by menopausal status:[2,17]

- Postmenopausal women had 33.6% of DALYs attributable to risk factors, with a high BMI contributing strongly.
- Premenopausal women showed a complex relationship with BMI, where an elevated BMI appeared to reduce risk marginally—consistent with established epidemiological patterns.

Forecast to 2050: A Mounting Challenge

Increasing Cases and Deaths

By 2050, global breast cancer incidence is projected to reach:[2]

- 3.56 million cases annually
- 1.37 million deaths annually

These increases will occur even though ASIR and ASMR are expected to remain relatively stable. The primary drivers are population growth, population aging, and increasing exposure to risk factors in transitioning countries.[2,17]

Regional Forecasts

Sub-Saharan Africa is expected to continue having the highest mortality burden, with ASMR projected to increase from 29.8 to 32.8 per 100,000 by 2050—far above the global average. Conversely, high-income regions are expected to maintain relatively low and stable mortality rates.[2]

Policy Implications and Health System Priorities

Early Detection and Timely Diagnosis

The World Health Organization's Global Breast Cancer Initiative emphasises three pillars: education and awareness, timely diagnosis, and comprehensive

treatment.[18] Evidence from Rwanda, Pakistan, and Tajikistan illustrates that low-cost strategies—such as community education and clinical breast examination training—can meaningfully improve early detection rates.[19,20]

Expanding Treatment Capacity

Many low-resource regions lack essential components of breast cancer care:[10,12,21]

- radiotherapy machines
- pathology services
- specialised surgery
- hormone therapy and chemotherapy availability

To address these gaps, it is critical to expand capacity—investing in infrastructure, training healthcare professionals, and ensuring reliable access to medicines and technologies—so that comprehensive breast cancer treatment becomes feasible and sustainable in these settings.

Reducing Financial Toxicity

Cancer treatment costs can devastate households, particularly in LMICs. Expanding universal health coverage, increasing insurance coverage for cancer, and reducing the cost of essential medicines (e.g., through generic production and pooled procurement) are crucial steps. Targeted investment, technology transfer, procurement transparency, and international partnerships will be critical.[22]

Conclusion

Despite impressive global advancements, breast cancer continues to impose an expanding and unequal burden. High-income countries have achieved substantial reductions in mortality through robust screening programs, early diagnosis, and high-quality treatment. Meanwhile, low- and middle-income countries face rising incidence and worsening mortality, fuelled by demographic transition, limited access to timely care, and escalating risk factor exposure.

Forecasts to 2050 underscore that without decisive action, breast cancer will increasingly strain health systems and widen global health inequities. A comprehensive, equity-oriented strategy—bolstering early detection, strengthening treatment infrastructure, mitigating risk factors, and reducing financial barriers—is essential to safeguard global progress in women's health and ensure that all women, regardless of geography or income, have the opportunity to survive and thrive after breast cancer.

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by Dr. Feng Xue

GLP-1 Medications for Cosmetic Weight Loss: Social Pressures, Off-Label Use, and Health Risks

Glucagon-like peptide-1 (GLP-1) receptor agonists, including semaglutide and tirzepatide, have transformed the medical management of obesity. Originally developed for type 2 diabetes and later approved for chronic weight management in people with obesity or overweight plus comorbidities, these medications are increasingly used for cosmetic weight loss by individuals with a body mass index (BMI) below 27 kg/m^2. Such use is considered off-label and raises important medical, ethical, and social questions. This article examines the scientific basis of GLP-1 therapy, the drivers of cosmetic use—particularly peer pressure and social media influence—the known and potential health risks, and how clinicians and patients should approach the benefit–risk balance when GLP-1 medications are used outside approved indications.

From Metabolic Therapy to Aesthetic Tool

GLP-1 receptor agonists were developed to mimic endogenous incretin hormones that regulate appetite, insulin secretion, and gastric emptying. Their ability to produce double-digit percentage weight loss has led to widespread adoption in obesity management. However, public attention, celebrity endorsements, and online wellness clinics have propelled these drugs into the cosmetic sphere, where they are sought for relatively modest weight reduction rather than for treatment of obesity-related disease. According to a Google trends analysis, the relative search volume (RSV) in "Ozempic" grew exponentially, at a rate of $y = (2 \times 10^{-36})e^{0.0019x}$. At the start of the 5-year period, the RSV remained steady from March 2018 until a small peak in June 2021, plateauing again until December 2021. Subsequently, the RSV increased, at first slowly until October 2022, then significantly from December 2022 onwards (**Figure 1**).[1,2]

This shift has blurred the line between medical necessity and aesthetic desire, prompting debate about safety, fairness, and appropriate prescribing.

Approved Indications and Off-Label Use in BMI < 27

Regulatory agencies such as the US Food and Drug Administration approve GLP-1 medications for chronic weight management only in:[3]

- Adults with BMI ≥ 30 kg/m^2, or
- Adults with BMI ≥ 27 kg/m^2 plus at least one weight-related comorbidity (e.g., hypertension, type 2 diabetes, dyslipidaemia).

Use in individuals with BMI < 27 kg/m^2 does not meet approved criteria and is therefore off-label. While off-label prescribing is legal, it relies on clinical judgment rather than robust evidence in this population. Importantly, safety and long-term benefit data for normal-weight or mildly overweight individuals remain limited.[4]

Peer Pressure, Social Media, and the "GLP-1 Aesthetic"

One of the strongest drivers of cosmetic GLP-1 use is social influence. Social media platforms amplify

before-and-after transformations, often without disclosure of side effects or the need for ongoing therapy. Peer pressure—particularly in image-focused professions and social circles—normalises pharmacologic weight loss even when health risks are rare but serious.[2,5]

Studies of off-label GLP-1 use in aesthetic and plastic surgery settings suggest that some patients pursue these drugs primarily to conform to perceived beauty standards rather than to improve metabolic health. This environment may undermine informed consent and inflate expectations of effortless, risk-free weight loss.[5]

Potential Health Risks of Cosmetic GLP-1 Use

Common Adverse Effects

The most frequent side effects of GLP-1 receptor agonists are gastrointestinal, including nausea, vomiting, diarrhoea, constipation, and abdominal discomfort. These effects are dose-dependent and may limit adherence.[6]

Serious and Emerging Risks

Although uncommon, more serious adverse events have been reported:

- Pancreatitis (including rare fatal cases)[7]
- Gallbladder disease, partly related to rapid weight loss[8]
- Gastroparesis and bowel obstruction, particularly with higher doses or prolonged use[9]

Concerns also exist about loss of lean muscle mass, nutritional deficiencies, and weight regain after

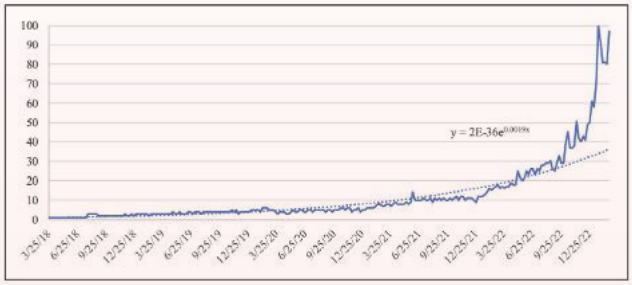

Figure 1. Relative search volume of the term "Ozempic"—March 2018 to February 2023[2]

discontinuation, especially when lifestyle interventions are not well integrated.[10]

Risk Amplification in Low-BMI Individuals

For individuals without obesity-related disease, the absolute health benefit is smaller, while exposure to pharmacologic risk remains. This alters the benefit–risk equation, making even low-probability adverse events more ethically significant.[11]

Benefits: Are There Any for Cosmetic Users?

GLP-1 medications can produce meaningful short-term weight loss and appetite control, even in non-diabetic populations. Some users report improved body image and psychological well-being. However, these benefits are largely subjective and depend on continued treatment; cessation often leads to weight regain.[4]

Critically, there is little evidence that cosmetic use confers long-term health advantages in people without obesity or metabolic risk factors.

Ethical and Clinical Considerations

Ethical analyses emphasise concerns about:[1,12]

- Medicalisation of normal weight variation
- Inequitable access, where individuals with medical need face drug shortages
- Commercial influences overshadowing evidence-based care

Clinicians are encouraged to prioritise medical necessity, conduct thorough risk assessments, and provide transparent counselling about uncertainties and alternatives. Patients, in turn, should critically evaluate whether pharmacologic weight loss aligns with their long-term health goals rather than short-term aesthetic pressures.

Balancing Benefit and Risk

For patients with obesity and related diseases, the benefits of GLP-1 therapy clearly outweigh the risks. In contrast, for individuals with BMI < 27 using these drugs for cosmetic purposes, the balance is far less favourable. The potential harms—however rare—may outweigh modest aesthetic benefits, especially when non-pharmacologic approaches could achieve similar outcomes with fewer risks.[1,11]

Conclusion

GLP-1 receptor agonists represent a major advance in obesity medicine, but their growing use for cosmetic weight loss highlights the tension between medical innovation and societal pressure. Off-label use in individuals with BMI < 27 occurs in a context of limited evidence, potential health risks, and powerful social influences. A cautious, ethically grounded approach—focused on informed consent, individualised risk assessment, and prioritisation of medical need—is essential. Ultimately, sustainable health should outweigh transient aesthetic ideals.

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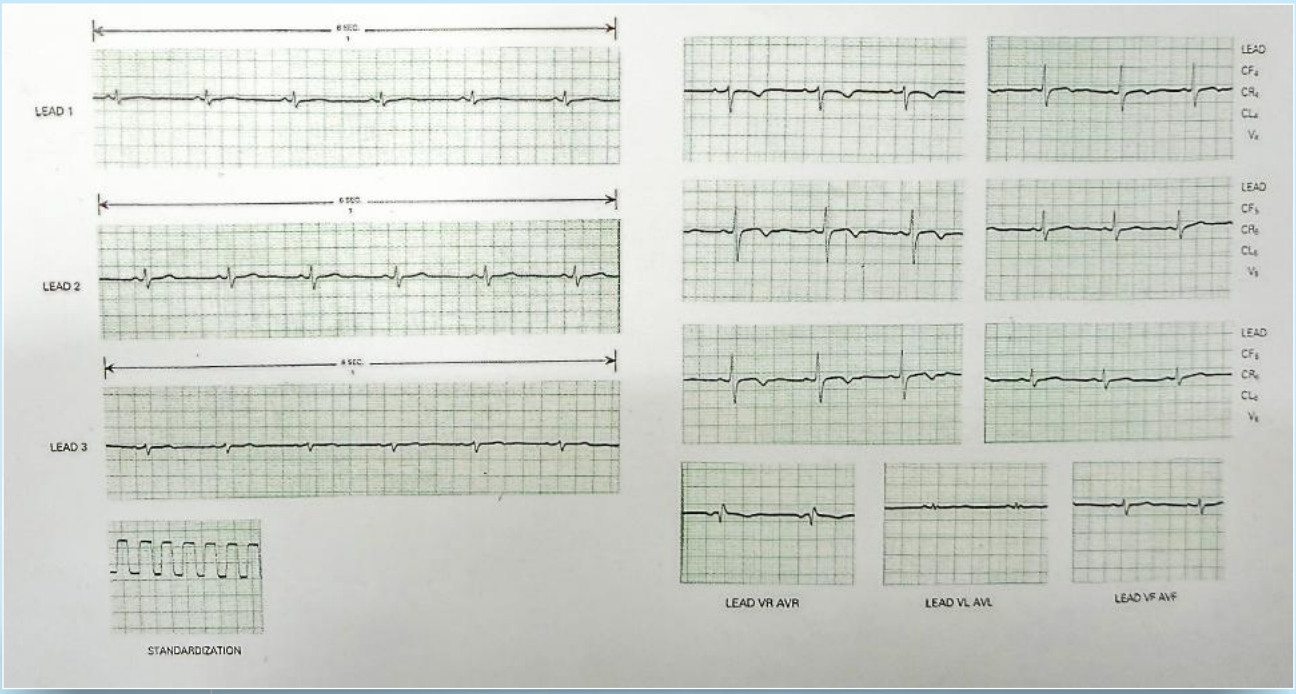
ECG CME APRIL 2026 (0.5 CME POINTS)

Dr. Pun Chiu On, Specialist in Cardiology

History:

This ECG belonged to a 37 year old female. She came to see you for routine medical checkup. Examination revealed she was obese. Blood pressure was 115/65 mmHg. This was her ECG.



Question: **What is the ECG diagnosis?**

(Please tick one)

- ❑ Normal ECG
- ❑ Sinus tachycardia
- ❑ Acute myocardial infarction
- ❑ Incomplete right bundle branch block
- ❑ Generalised low voltage

This ECG CME was prepared by Dr. Pun Chiu On, Specialist in Cardiology.

Please complete the Self-Study by visiting our website: https://cmevideo.hkdu.org/ or scan the QR code to submit your answers on or before ***30-APRIL-2026***

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Answer to February 2026 Issue's CME

ECG diagnosis: Left ventricular hypertrophy.

Reason for the ECG diagnosis:

Criteria for left ventricular hypertrophy:
(1) R wave in V5 or V6 >26 mm.
(2) R wave in V5 or V6 + S wave in V1 >35 mm.
(3) Tallest R wave in any of leads V4, V5, V6 plus deepest S wave in any of leads V1, V2 or V3 exceeds 40 mm.
(4) S wave in V1, V2 or V3 exceeds 30 mm.
(5) R wave in aVL >11 mm.
(6) R wave in aVF > 20 mm.
(7) Ventricular activation time (i.e. the duration from onset of QRS complex to peak of R wave) exceeds 0.04 seconds.
(8) Abnormal ST segment depression in any leads facing the left ventricle (i.e. in leads V4, V5, or V6).
(9) T wave inversion in leads facing left ventricle.

Please note that left ventricular hypertrophy (LVH) is a graded abnormality. Not all the above criteria need to be present in making the diagnosis.

In this ECG, R wave in V5 is 40 mm, S in V1 is 27 mm, and S in V1 plus R in V6 is 67 mm.

Thus, it fulfils the voltage criteria for LVH.

Also, the ventricular activation time (the duration of time from beginning of QRS to peak of R wave) is 0.04 seconds. There are ST segment downslope depression and abnormal T inversion in leads V3-V6, I, II, aVL.

If the only criterion present is tall R wave or deep S wave, it would be known as "left ventricular hypertrophy by voltage criteria". Just tall R wave in ECG could be due to many other causes, and may not be due to real left ventricular hypertrophy.

Possible aetiology:

(1) Hypertension (commonest cause). (2) Aortic stenosis. (3) Hypertrophic cardiomyopathy. (4) Coarctation of aorta.
(5) Occasionally aortic regurgitation, or mitral regurgitation may produce similar ECG.

Significance:

It depends on the underlying aetiology.

Principle of management:

Look for underlying aetiology from history and physical examination. Treat accordingly if found.

Management of this patient:

Echocardiography confirmed apical hypertrophic cardiomyopathy (apical HCM) which accounts for his ECG changes. Apical HCM is more often seen in orientals than westerners. The course is more benign than the classical hypertrophic obstructive cardiomyopathy.

Note the symmetrical giant T wave inversion is often found in apical HCM, as in this ECG. As this patient is asymptomatic, no medication is required. He is advised to observe for any cardiovascular symptom, and to have his echo repeated in 1-2 years.

by Michelle Lee

On-the-Pulse

Rehabilitation Medicine

Discover Nordic Walking[1]

A systematic review of 14 randomised controlled trials suggests that Nordic walking offers a promising non-pharmacological approach for managing chronic pain and fatigue. The research, which included participants with conditions ranging from Parkinson's disease and fibromyalgia to chronic back pain and post-COVID recovery, found that six of nine studies reported beneficial effects on pain perception, while six of eight studies showed improvements in fatigue following Nordic walking interventions. Programs typically lasted 6-24 weeks with 2-4 sessions weekly. Notably, Nordic walking proved accessible and easy to learn, with benefits achievable even in unsupervised settings, though its effects were not consistently superior to regular walking. The findings support Nordic walking as a viable, low-barrier exercise option that may improve quality of life for individuals living with chronic conditions.

Nutritional Health

Mala Flavour Linked to Excessive Pregnancy Weight Gain[2]

A cohort study of 495 pregnant women in Chongqing examined whether taste preferences and dietary patterns were linked to excessive gestational weight gain. The researchers found that women who preferred Mala flavour were more likely to follow a high-carbohydrate diet, characterised by elevated cereal intake, and this pattern was associated with a higher risk of gaining too much weight during pregnancy. Mediation analysis suggested that Mala preference did not act mainly on its own; instead, its effect on excessive gestational weight gain was largely explained through high-carbohydrate eating habits. The findings suggest that prenatal nutrition guidance in Southwest China should address both Mala flavour preferences and carbohydrate-heavy eating patterns.

Allergy and Immunology

Food Allergy Trends in Hong Kong Preschool Children[3]

A 15-year study of 11,537 Hong Kong children aged 2-7 years reveals a growing disconnect between parent-perceived and doctor-diagnosed food allergies. While parent-reported food allergy increased from 6.1% in 2006 to 8.5% in 2020, doctor-diagnosed cases remained stable at approximately 5%. Alarmingly, allergic reactions to specific triggers showed sustained increases: egg allergy doubled to 1.86%, peanut nearly tripled to 1.52%, tree nut increased five-fold to 0.68%, and shellfish rose to 1.99%. Meanwhile, asthma and wheezing declined, while eczema increased. The findings highlight an urgent need for improved allergy diagnostic services and public education to bridge the gap between perceived and confirmed allergies in urban Asian populations.

Allergy and Immunology

Adrenaline Fails to Reverse Histamine-Induced Hypotension[4]

A randomised controlled trial tested whether intramuscular adrenaline, the standard first-line treatment for anaphylaxis, effectively reverses severe hypotension caused by histamine infusion in healthy volunteers. Despite achieving plasma adrenaline concentrations considered therapeutic, the injections failed to produce a sustained pressor response in most participants, performing indistinguishably from placebo. Only 25% of participants showed a transient blood pressure recovery, which was not maintained. The findings challenge the longstanding guideline recommendations based on expert consensus rather than controlled human data, highlighting a critical need to re-evaluate the role of adrenaline in histamine-mediated shock and consider more potent interventions.

Environmental Health

Compound Hot and Humid Events Elevate Health Risks[5]

A study in subtropical Hong Kong examined 22 definitions of compound hot-humid extreme events and their impact on mortality. Using data from 1995–2021, researchers found that events defined by specific humidity with seasonally-determined thresholds, particularly Hot_wet and Hot_wet95 were significantly associated with increased mortality risk. Older adults, males, and pneumonia-related deaths showed the greatest vulnerability. Notably, pre-summer events also carried elevated risk, challenging the focus on traditional summer seasons. The findings underscore the urgent need to integrate humidity into heat-health warning systems and adaptation strategies, as climate change drives more frequent compound heat-humidity extremes in coastal cities.

Cancer Epidemiology

Reproductive Cancers Narrow the Female Survival Advantage in Midlife[6]

Using mortality data from 20 low-mortality countries between 1955 and 2020, the study examined how female reproductive cancers shape the survival gap between women and men. Although women had a clear overall survival advantage, those aged 35 to 60 faced higher cancer mortality than men across successive birth cohorts, driven mainly by breast and gynaecological cancers. The analysis covered 264.4 million deaths and estimated that removing female reproductive cancers would widen the female survival advantage by an average of 0.77 years. The findings highlight a persistent midlife vulnerability that offsets part of women's longer life span and support stronger prevention, earlier detection, and better treatment access for reproductive cancers.

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"Fostering Innovation Through Continuous Medical Education with On the Pulse"

by Dr. Feng Xue

Over 1,000 Healthcare Workers and Kidney Patients Set World Record with Mass Fitness Exercise

Hong Kong Sets New Record for "Most Healthcare Professionals and Kidney Patients Doing Health Exercises Simultaneously"

Promoting Physical Activity to Prevent Chronic Diseases on World Kidney Day

On the morning of March 8, the Kowloon Park Piazza was filled with energy and joy, as more than 1,000 healthcare workers, kidney patients and their caregivers joined hands—literally and figuratively—to perform a group fitness routine. Their synchronised, upbeat movements set a new world record for the "Most healthcare professionals and kidney patients doing health exercises simultaneously", while spreading the message that regular exercise helps improve physical and mental well-being and prevents chronic diseases.

The “Mega Exercise” marked the highlight of the Kidney Health Activity Day, jointly organised by the Hong Kong Kidney Foundation, the Hong Kong Society of Nephrology, and the Hong Kong Association of Renal Nurses. The event was held in support of the 21st World Kidney Day and the 6th Hong Kong Kidney Day.

Prominent Healthcare Leaders Join the Event in Person

A number of distinguished healthcare leaders attended the event and even joined the exercise on the field, including:

- Dr. Cecilia Fan, Acting Secretary for Health
- Dr. Ronald Lam, Director of Health
- Mr. Henry Fan, Chairman of the Hospital Authority
- Dr. Siu-Fai Lui, Chairman of the Hong Kong Kidney Foundation and Chairman of the Department of Health's Steering Committee on Promotion of Physical Activity
- Prof. Desmond Yap, President of the Hong Kong Society of Nephrology
- Ms. Dorothy Sung, President of the Hong Kong Association of Renal Nurses
- Dr. Leong Che-hung, President of the Hong Kong Kidney Foundation
- Prof. Richard Yu, Patron of the Hong Kong Kidney Foundation
- Dr. Lobo Louie, Vice-chairman of the Department of Health's Steering Committee on Promotion of Physical Activity

In their speeches, several guests highlighted the critical role physical activity plays in preventing chronic diseases.

Dr. Siu-Fai Lui stated, “This year’s theme is ‘Move for Your Health’, because exercise is an investment in lifelong wellness. Physical activity reduces obesity and lowers the risk of hypertension and diabetes—two of the major culprits behind kidney failure.” He called on the public to “move for better physical and mental health; move to stay away from chronic diseases.”

1,060 Participants Exercise Together to Break World Record

The highlight of the day came when all participants performed the fitness routine together. Under the witness of an official from the World Record Association, it was confirmed that 1,060 people took part in the synchronised exercise, setting a new world record. The announcement was met with loud cheers and applause.

Kidney Disease: A Growing Health Challenge in Hong Kong

Kidney disease remains a significant health concern in Hong Kong. About 1 in 10 adults suffers from some degree of kidney damage, and each year, approximately 1,500 new end-stage renal failure patients require dialysis to stay alive.

Data also shows that 61% of new end-stage kidney failure cases are caused by diabetes and hypertension. Effective prevention and management of the “three highs” (high blood pressure, high blood sugar, high

cholesterol) is therefore crucial to kidney protection. A random community health screening conducted by the organisers a week before further underscored the urgency:

- 40% of the 620 participants were found to have hypertension
- 20% had diabetes
- Alarmingly, 33% of those with hypertension and 16% of those with diabetes were previously unaware of their conditions

The findings highlight the need for regular health checks and improved public health awareness. The organisers hope that the world record challenge will inspire the public to incorporate exercise into their daily lives, undergo early screening, manage the three highs, and work together to tackle kidney disease. Even people with kidney conditions can benefit from appropriate exercise, especially to improve cardiovascular health.

A Day of Wellness, Fun and Community Spirit

The event featured a colourful program including dance performances, skits, singing and live band music. The crowd remained enthusiastic throughout, cheering and clapping along. After the mass exercise, participants split into four groups to take part in walking routes around Kowloon Park, continuing the theme of “moving for health.”

The morning was filled with laughter, music and encouragement, symbolising unity between healthcare workers, patients, and the public in building a healthier future together.

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*Refers to the ELDERCARE-AF trial.[2]

†Based on a meta-analysis of 17 observational studies to compare the effect of edoxaban in patients with AF. Adjusted risk ratios and 95% confidence intervals of the outcomes were collected and pooled by a random-effects model. Compared with vitamin K antagonists, edoxaban was associated with lower risks of stroke or systemic embolism (RR=0.67, 95% CI: 0.61–0.74), major bleeding (RR=0.54, 95% CI: 0.44–0.67), and intracranial hemorrhage (RR = 0.51, 95% CI:0.29 -0.90). Compared with dabigatran or rivaroxaban, edoxaban was associated with reduced risks of stroke or systemic embolism (dabigatran [RR=0.76, 95% CI: 0.66–0.87]; rivaroxaban [RR=0.81, 95% CI: 0.70 -0.94]) and major bleeding (dabigatran [RR=0.82, 95% CI: 0.69–0.98]; rivaroxaban [RR = 0.81, 95% CI: 0.70 -0.94]). Compared with apixaban, edoxaban was associated with a reduced risk of stroke or systemic embolism (RR=0.87, 95% CI: 0.79–0.97), but had similar risks of bleeding events.[2]

AF, atrial fibrillation; CYP3A4, cytochrome P450 3A4; OAC, oral anticoagulation; NOAC, novel oral anticoagulants; RCT, randomised-controlled trial

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LIXIANA® (Edoxaban) 60mg/30mg/15mg film-coated tablets.
Indications: Prevention of stroke & systemic embolism in adult patients w/ nonvalvular atrial fibrillation (NVAF) w/ ≥1 risk factors eg, CHF, HTN, ≥75 yr of age, DM, prior stroke or transient ischaemic attack. Treatment of DVT & pulmonary embolism (PE), & prevention of recurrent DVT & PE in adults. **Dosage:** For Prevention of stroke & systemic embolism 60 mg once daily. Treatment of DVT & PE & prevention of recurrent DVT & PE (VTE) 60 mg once daily following initial use of parenteral anticoagulant for at least 5 days. Moderate or severe renal impairment (CrCl 15-50 mL/min), ≤60 kg body wt, concomitant use of the following P-gp inhibitors: ciclosporin, dronedarone, erythromycin, or ketoconazole 30 mg once daily. For prevention of ischemic stroke and systemic embolism in adult patients with NVAF, oral administration of 15 mg once daily should be considered for elderly patients (roughly 80 years of age or older) who meet both the following criteria: Having at least one of the following hemorrhagic diatheses: history of hemorrhage in important organs, including intracranial haemorrhage, intraocular haemorrhage, and haemorrhage in the gastrointestinal tract, low body weight (≤45 kg), creatinine clearance level of ≥15 mL/min and <30 mL/min, regular use of NSAIDs, use of antiplatelet drugs; unable to receive a usual dose of edoxaban or an approved dose of other oral anticoagulants because of a risk of haemorrhage. **Contraindications:** Hypersensitivity. Clinically significant active bleeding; hepatic disease associated w/ coagulopathy & clinically relevant bleeding risk; lesion or condition, if considered to be a significant risk for major bleeding, include current or recent gastrointestinal ulceration, presence of malignant neoplasms at high risk of bleeding, recent brain or spinal injury, recent brain, spinal or ophthalmic surgery, recent intracranial haemorrhage, known or suspected oesophageal varices, arteriovenous malformations, vascular aneurysms or major intraspinal or intracerebral vascular abnormalities; uncontrolled severe HTN; concomitant treatment w/ any other anticoagulants eg, unfractionated heparin (UFH), LMWH (enoxaparin, dalteparin, etc), heparin derivatives (fondaparinux, etc), oral anticoagulants (warfarin, dabigatran etexilate, rivaroxaban, apixaban, etc) except under specific circumstances of switching oral anticoagulant therapy or when UFH is given at doses necessary to maintain an open central venous or arterial catheter. Pregnancy and breast-feeding. **Precaution:** Lixiana 15 mg is not indicated as monotherapy, as it may result in decreased efficacy. It is only indicated in the process of switching from edoxaban 30 mg (patients with one or more clinical factors for increased exposure; see table 1 in full prescribing information) to VKA, together with an appropriate VKA dose (see table 2, section 4.2 in full prescribing information). Please also see the Dosage section. Lixiana 15 mg once daily should be considered for the prevention of ischaemic stroke and systemic embolism in elderly patients (roughly 80 years of age or older) with NVAF. For details, please refer to full prescribing information section 4.2, Special populations: "Consideration of a dose reduction to 15 mg once daily for prevention of ischemic stroke and systemic embolism in elderly patients (roughly 80 years of age or older) with NVAF with certain conditions". **Interactions:** Reduced dissolution & absorption w/ medicines or disease conditions that increase gastric emptying & gut motility. Increased plasma conc w/ P-gp inhibitors (ciclosporin, dronedarone, erythromycin, ketoconazole, quinidine, verapamil, amiodarone,clarithromycin).Reduced plasma conc w/ P-gp inducers (rifampicin;phenytoin, carbamazepine, phenobarbital or St. John's wort). Increased Cmax of edoxaban w/ P-gp substrates (Digoxin). Increased risk of bleeding w/ other anticoagulants; SSRIs or SNRIs. Increased bleeding time w/ ASA (100 mg or 325 mg); NSAIDs. Increased clinically relevant bleeding w/ thienopyridines (eg, clopidogrel). **Undesirable effects:** Epistaxis; anaemia; dizziness, headache; abdominal pain, lower/upper GI haemorrhage, oral/pharyngeal haemorrhage, nausea; increased blood bilirubin, increased γ-glutamyltransferase; cutaneous soft tissue haemorrhage, rash, pruritus; macroscopic haematuria/urethral haemorrhage. Version: Jul 2025. Please refer to the Package Insert before prescribing. **Full local prescribing information is available upon request. Please report Individual Case Safety Report (ICSR)/Adverse Event (AE) to Daiichi Sankyo Hong Kong via pv_hk@daiichisankyo.com.**

HK-DAI-LIX-2601029
Approval Date: March 2026

Daiichi-Sankyo
Daiichi Sankyo Hong Kong
Suites 3207-11, 32/F, Tower One, Times Square, 1 Matheson Street, Causeway Bay, Hong Kong
Tel: (852) 2868 9072 Fax: (852) 2801 4341

once-daily Lixiana edoxaban

Haematology :

BLENREP®

(belantamab mafodotin)

GLAXOSMITHKLINE

HK Reg. No. HK-69000, HK-69001 (02 Mar, 2026)

Composition[4]:

BLENREP® is available as:

- 70 mg powder for concentrate for solution for infusion
- 100 mg powder for concentrate for solution for infusion

Indication[4]:

BLENREP® is indicated in adults for the treatment of relapsed or refractory multiple myeloma:

- In combination with bortezomib and dexamethasone in patients who have received at least one prior therapy; and
- In combination with pomalidomide and dexamethasone in patients who have received at least one prior therapy including lenalidomide

References

1. Government of Canada. Drug and Health Product Portal. Product Monograph: Sandoz Afatinib. Available from: https://pdf.hres.ca/dpd_pm/00077398.PDF. [Accessed 9 March 2026]. **2.** EMA. Summary of Product Characteristics. Available from: https://www.ema.europa.eu/en/documents/product-information/erleada-epar-product-information_en.pdf. [Accessed 9 March 2026]. **3.** FDA. Highlights of Prescribing Information. Available from: https://www.accessdata.fda.gov/drugsatfda_docs/label/2023/216974s000lbl.pdf. [Accessed 12 March 2026]. **4.** EMA. Summary of Product Characteristics. Available from: https://www.ema.europa.eu/en/documents/product-information/blenrep-epar-product-information_en.pdf. [Accessed 12 March 2026].

The information in The Pace is provided as a courtesy service to our readers and is intended for medical professional reference only. Please peruse the latest local prescription information prior to prescription.

Keep Up With the Pace of Drug Development and Discover the Newly Launched Treatments in Hong Kong

One Target, Dual Action, Six Indications

DUPIXENT targets IL-4Ra with dual action on both IL-4 & IL-13 to reduce Type 2 inflammation[1,2]

DUPIXENT - your versatile biologic that targets six conditions[3]

Atopic Dermatitis (AD)
- Moderate-to-severe AD in adults and adolescents ≥12 years old†
- Severe AD in children 6 months to 11 years old†

6+ months

Asthma
- In adults and adolescents ≥12 years old as add-on maintenance treatment for severe asthma with Type 2 inflammation*
- In children 6 to 11 years old as add-on maintenance treatment for severe asthma with Type 2 inflammation^

6+ years

Chronic rhinosinusitis with nasal polyposis (CRSwNP)
- As an add-on therapy with intranasal corticosteroids for the treatment of adults with severe CRSwNP#

18+ years

Prurigo Nodularis (PN)
- Moderate-to-severe PN in adults who are candidates for systemic therapy

18+ years

Eosinophilic Esophagitis (EoE)
- In adults and adolescents ≥12 years old weighing ≥40 kg‡

12+ years

Newly approved

Chronic Obstructive Pulmonary Disease (COPD)
- As add-on maintenance treatment with other medicines for adults with uncontrolled COPD§

18+ years

† Candidates for systemic therapy
* Characterised by raised blood eosinophils and/or raised FeNO, who are inadequately controlled with high dose ICS plus another medicinal product for maintenance treatment.
^ Characterised by raised blood eosinophils and/or raised FeNO, who are inadequately controlled with medium to high dose ICS plus another medicinal product for maintenance treatment.
# For whom therapy with systemic corticosteroids and/or surgery do not provide adequate disease control.
‡ Those who are inadequately controlled by, are intolerant to, or who are not candidates for conventional medicinal therapy.
§ Characterised by raised blood eosinophils, on a combination of an inhaled corticosteroid (ICS), a long acting beta2-agonist (LABA), and a long-acting muscarinic antagonist (LAMA), or on a combination of a LABA and a LAMA if ICS is not appropriate.

Abbreviations: AD=atopic dermatitis; COPD = chronic obstructive pulmonary disease; CRSwNP= chronic rhinosinusitis with nasal polyps; EoE= eosinophilic esophagitis; FeNO=fractional exhaled nitric oxide; ICS=inhaled corticosteroids; LABA = long acting beta2-agonist; LAMA = long acting muscarinic antagonist; PN=prurigo nodularis.

References:
1. Guttman-Yassky E, et al. J Allergy Clin Immunol. 2019;143(1):155-172. 2. Gandhi NA, et al. Nat Rev Drug Discov. 2016;15(1):35-50 3.DUPIXENT® Hong Kong Prescribing Information

Presentation: Dupilumab solution for injection in a pre-filled syringe with needle shield. **Indications:** Atopic Dermatitis (AD): Moderate-to-severe AD in adults and adolescents ≥12 years who are candidates for systemic therapy; severe atopic dermatitis in children 6 months to 11 years old who are candidates for systemic therapy. Asthma: In adults and adolescents ≥12 years as add-on maintenance treatment for severe asthma with type 2 inflammation characterised by raised blood eosinophils and/or raised FeNO, who are inadequately controlled with high dose ICS plus another medicinal product for maintenance treatment. In children 6 to 11 years old as add-on maintenance treatment for severe asthma with type 2 inflammation characterised by raised blood eosinophils and/or raised FeNO, who are inadequately controlled with medium to high dose ICS plus another medicinal product for maintenance treatment. For 300 mg only – Chronic rhinosinusitis with nasal polyposis (CRSwNP): As an add-on therapy with intranasal corticosteroids for the treatment of adults with severe CRSwNP for whom therapy with systemic corticosteroids and/or surgery do not provide adequate disease control. Prurigo Nodularis (PN): Moderate-to-severe PN in adults who are candidates for systemic therapy. Eosinophilic esophagitis (EoE): In adults and adolescents ≥12 years, weighing ≥40 kg, who are inadequately controlled by, are intolerant to, or who are not candidates for conventional medicinal therapy. Chronic obstructive pulmonary disease (COPD): In adults as add-on maintenance treatment for uncontrolled COPD characterised by raised blood eosinophils on a combination of ICS, LABA, and LAMA, or on a combination of LABA and LAMA if ICS is not appropriate. **Dosage & Administration:** Subcutaneous injection. AD adults: Initial dose of 600 mg (two 300 mg injections), followed by 300 mg Q2W. AD adolescents (12-17y/o): Body weight <60 kg- Initial dose of 400 mg (two 200 mg injections), followed by 200 mg Q2W. Body weight ≥60 kg- same dosage as adults. AD children (6-11y/o): Body weight 15kg~<60 kg- Initial dose of 300 mg on Day 1 follow by 300 mg on Day 15, then 300mg Q4W. Body weight ≥60 kg- same dosage as adults. * The dose may be increased to 200 mg Q2W in patients with body weight of 15 kg~< 60 kg based on physician's assessment. AD children (6 months-5y/o): Body weight 5kg~<15 kg- Initial dose of 200 mg, then 200 mg Q4W. Body weight 15kg~<30 kg- Initial dose of 300 mg, then 300 mg Q4W. Dupilumab can be used with or without topical corticosteroids. Topical calcineurin inhibitors may be used, but should be reserved for problem areas only, e.g. face, neck, intertriginous and genital areas. Consider discontinuing treatment in patients who have shown no response after 16 weeks. Asthma adults and adolescents: Initial dose of 400 mg, followed by 200 mg Q2W. For patients with severe asthma and on oral corticosteroids or with severe asthma and co-morbid moderate-to-severe AD or adults with co-morbid severe CRSwNP- initial dose of 600 mg, followed by 300 mg Q2W. Asthma children (6-11y/o): Body weight 15kg~<30 kg- 300 mg Q4W. Body weight 30kg~<60 kg- 200 mg Q2W; or 300 mg Q4W. Body weight ≥60 kg- 200 mg Q2W. For paediatric patients (6-11y/o) with asthma and co-morbid severe atopic dermatitis, as per approved indication, the recommended dose should follow AD children (6-11y/o). Patients receiving concomitant oral corticosteroids may reduce steroid dose gradually once clinical improvement with dupilumab has occurred. The need for continued dupilumab therapy should be considered at least annually as determined by a physician. CRSwNP: Initial dose of 300 mg, followed by 300 mg Q2W. Consider discontinuing treatment in patients who have shown no response after 24 weeks. PN: Initial dose of 600 mg (two 300 mg injections), followed by 300 mg Q2W. Dupilumab can be used with or without topical corticosteroids. Consider discontinuing treatment in patients who have shown no response after 24 weeks. EoE: 300 mg QW. Dupilumab 300 mg QW has not been studied in patients with EoE weighing <40 kg. Dosing beyond 52 weeks has not been studied. COPD: 300 mg Q2W. Consider discontinuing treatment in patients who have shown no response after 52 weeks. For Missed dose instructions, please refer to the full prescribing information. **Contraindications:** Hypersensitivity to dupilumab or any of the excipients. **Precautions:** Not be used to treat acute symptoms, acute exacerbations of asthma or COPD, acute bronchospasm or status asthmaticus. Do not discontinue corticosteroids abruptly upon start of dupilumab. Reduction should be gradual and performed under supervision of a physician; it may be associated with systemic withdrawal symptoms and/or unmask conditions previously suppressed by systemic corticosteroid therapy. Biomarkers of type 2 inflammation may be suppressed by systemic corticosteroid use. If systemic hypersensitivity reaction occurs, discontinue dupilumab and initiate appropriate therapy. Be alert to vasculitic rash, worsening pulmonary symptoms, cardiac complications, and/or neuropathy presenting in patients with eosinophilia. Treat pre-existing helminth infections before initiating dupilumab. If patients become infected while receiving dupilumab and do not respond to anti-helminth treatment, discontinue dupilumab until infection resolves. Cases of enterobiasis were reported in children 6 to 11 years old in the paediatric asthma development program. Advise patients to promptly report new onset or worsening eye symptoms. Patients who develop conjunctivitis, dry eye and keratitis that does not resolve following standard treatment should undergo ophthalmological examination. Sudden changes in vision or significant eye pain that does not settle warrant urgent review. Patients with comorbid asthma should not adjust or stop asthma treatments without consultation with physicians. Carefully monitor patients after discontinuation of dupilumab. Avoid using live and live attenuated vaccines concurrently with dupilumab. Patients should be brought up to date with immunisations before starting dupilumab. **Drug Interactions:** Immune responses to TdaP vaccine and meningococcal polysaccharide vaccine were assessed. Patients receiving dupilumab may receive concurrent inactivated or non-live vaccinations. **Pregnancy and lactation:** Should be used during pregnancy only if potential benefit justifies potential risk to foetus. Unknown whether dupilumab is excreted in human milk or absorbed systemically after ingestion. Decision must be made whether to discontinue breast-feeding or dupilumab taking into account benefit of breast feeding for the child and benefit of therapy for the woman. **Undesirable effects:** Most common adverse reactions reported- Injection site reactions, conjunctivitis, conjunctivitis allergic, arthralgia, oral herpes, eosinophilia and injection site bruising. Safety profile observed in adolescents and children 6 months to 11 years old consistent with that seen in adults. For other undesirable effects, please refer to the full prescribing information. **Preparation:** 2 x 300mg/2ml in pre-filled syringe with needle shield, 2 x 200mg/1.14ml in pre-filled syringe with needle shield. **Legal Classification:** Part 1, First & Third Schedules Poison **Full prescribing information is available upon request.**

MAT-HK-2400691-1.0-12/2024

API-HK-DUP-24.12

sanofi

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DUPIXENT® (dupilumab)

ONCE-WEEKLY

wegovy® 一週一次 維秀美®

semaglutide injection 2.4 mg

Wegovy®: the ONLY weight management medication recommended by cardiology guidelines[6]*

Your Trustworthy Choice for Transformative Weight Loss and Heart Protection[1-4]

≥20%
WEIGHT LOSS

Achieved by ~1 in 3 patients treated over 68 weeks[2,3*]

20%
REDUCTION IN MACE

vs placebo on top of CV standard of care in adults with established CVD[4†]

(HR=0.80; 95% CI=0.72-0.90; p<0.001)

>20
MILLION

patients treated with semaglutide worldwide since launch[5]

NOW AVAILABLE

*STEP 1 was a double-blind trial that enrolled 1961 adults with BMI > 30 kg/m² (> 27 kg/m² in persons with > 1 weight-related coexisting condition) who did not have diabetes. Participants were randomly assigned in 2:1 ratio to 68 weeks of treatment with once-weekly subcutaneous semaglutide (2.4 mg) or placebo, plus lifestyle intervention. On-treatment data at week 68 showed that 34.8% of the participants on semaglutide had weight loss of ≥ 20%.[2] **STEP 4** was a randomized clinical trial that evaluated the effect of continued weekly subcutaneous semaglutide vs placebo on weight loss maintenance in adults with overweight or obesity.[3] †**SELECT** was a multicenter, double-blind, randomized, placebo-controlled, event-driven superiority trial that enrolled patients aged ≥ 45 who had preexisting CVD and BMI ≥ 27 kg/m² but no history of diabetes. Patients were randomly assigned in a 1:1 ratio to receive once-weekly subcutaneous semaglutide (2.4 mg) or placebo. The primary CV end point was a composite of death from CV causes, non-fatal myocardial infarction, or non-fatal stroke in a time-to-first-event analysis.[4] ‡**2024 ESC Guidelines for the management of chronic coronary syndromes:** semaglutide should be considered in chronic coronary syndrome patients without diabetes, but with overweight or obesity (BMI ≥ 27 kg/m²) to reduce CV mortality, myocardial infarction, or stroke (class of recommendation=IIa; level of evidence=B).[6]
BMI=body mass index; CI=confidence interval; CV=cardiovascular; CVD=cardiovascular disease; ESC=European Society of Cardiology; HR=hazard ratio; MACE=major adverse cardiovascular events.

References: 1. Wegovy® Hong Kong Prescribing Information. 2025. **2.** Wilding JPH, et al. N Engl J Med. 2021;384:989-1002. **3.** Rubino D, et al. JAMA. 2021;325:1414-25. **4.** Lincoff AM, et al. N Engl J Med. 2023;389:2221-32. **5.** Data on File. REF-73669 **6.** Vrints C, et al. Eur Heart J. 2024;45:3415-537.

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