

Multisystem Mechanisms of Fall Risk in Elderly Men Receiving Androgen Deprivation Therapy: A Clinical and Public Health Evaluation

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Abstract

Androgen deprivation therapy (ADT) is a mainline treatment for advanced prostate cancer, yet its systemic effects substantially elevate fall risk in elderly men. This paper examines the multifactorial mechanisms driving ADT-associated falls, including rapid-onset sarcopenia, neuromuscular dysfunction, cognitive impairment, and cardiovascular dysregulation. Testosterone suppression leads to loss of muscle mass, diminished motor coordination, and executive dysfunction. These factors collectively compromise gait stability and postural control. ADT also accelerates bone mineral loss, increasing susceptibility to fractures from low-energy falls. Neuroimaging studies reveal structural brain changes such as cerebellar atrophy and disrupted visuospatial processing that further impair balance. Despite growing awareness of these risks, fall prevention remains underemphasized in prostate cancer survivorship care. A widely publicized case involving an elderly public figure, whose frequent falls preceded the disclosure of advanced prostate cancer, illustrates the real-world consequences of functional decline associated with ADT. This paper advocates for a proactive and interdisciplinary survivorship model that incorporates resistance training, cognitive screening, orthostatic evaluation, and bone health monitoring to reduce fall risk and improve outcomes in men receiving ADT.

Key Words: androgen antagonists; prostatic neoplasms ; sarcopenia; cognitive dysfunction; accidental falls

Introduction

Androgen deprivation therapy (ADT) remains a first-line treatment in the management of advanced and metastatic prostate cancer, particularly in elderly men. While effective at suppressing tumor progression through the reduction of circulating testosterone, ADT is increasingly recognized for its systemic adverse effects—chief among them, a heightened risk of falls. Falls in this population are not only common but are also strongly associated with significant morbidity, including fractures, hospitalizations, loss of independence, and accelerated functional decline [1].

Multiple physiological and neurological mechanisms have been implicated in ADT-related fall risk. On the musculoskeletal level, testosterone plays a critical role in the maintenance of lean muscle mass, strength, and neuromuscular coordination. Its deprivation leads to rapid-onset sarcopenia, characterized by degradation of type II muscle fibers, diminished satellite cell activity, and reduced muscle protein synthesis

[2]. These changes result in compromised gait stability, decreased mobility, and impaired balance [3]. Concurrently, ADT significantly accelerates bone mineral loss, increasing the risk of osteoporosis and vertebral fractures, which further amplify the impact of falls [4][5].

Emerging evidence also highlights central nervous system alterations as key contributors to balance and postural instability. Androgen receptors are expressed in the cerebellum, hippocampus, and frontal cortex—regions essential for motor control, memory, and executive functioning. ADT has been associated with cerebellar volume loss, disruption of white matter microstructure, increased neuroinflammatory signaling, and impaired neuroplasticity [6]. These neurophysiological effects are compounded by indirect systemic consequences of ADT, including anemia, fatigue, and metabolic dysfunction, all of which reduce physiological resilience [7].

The interaction of these musculoskeletal and neurological impairments presents a multidimensional risk that is not adequately addressed in current prostate cancer treatment guidelines. Sarcopenia may develop within months of ADT initiation, while cognitive and cerebellar changes often progress more insidiously, compounding fall vulnerability over time [8]. This complex interplay necessitates a more integrative clinical approach to survivorship care in men undergoing ADT.

Public awareness of these risks has grown due to highly visible examples. Former U.S. President Joe Biden experienced multiple public falls, speech impediments, and episodes of apparent cognitive slowing during his presidency. In May 2025, it was disclosed that he had been diagnosed with prostate cancer, Gleason score 9 (grade group 5), with documented metastases to bone [9]. While the details of his treatment have not been formally released, ADT remains a standard intervention in such cases. His clinical trajectory aligns closely with the neurocognitive and physical side effects described in the medical literature and serves as a real-world case that reflects broader clinical patterns.

Methods

A targeted literature review was conducted using PubMed, Scopus, and Google Scholar to assess mechanisms of fall risk in elderly men receiving androgen deprivation therapy (ADT). Peer-reviewed clinical and translational studies published within the last ten years were prioritized, with emphasis on geriatrics, neurobiology, urology, and public health. Selection criteria favored rigor, relevance, and interdisciplinary scope, integrating data from randomized trials, neuroimaging analyses, and epidemiological research. Publicly available case reports involving high-profile individuals were reviewed to contextualize clinical implications. No diagnostic claims are made regarding any specific person; all health-related references are drawn from verified disclosures and established media outlets. This review aims to inform survivorship care guidelines rather than make individual clinical judgments.

Sarcopenia and Neuromuscular Impairment

The suppression of androgen signaling via ADT has a profound and rapid impact on skeletal muscle physiology. Testosterone plays a critical anabolic role in maintaining muscle mass, stimulating satellite cell proliferation, and supporting protein synthesis pathways through androgen receptor activation in myocytes [10]. One contributing mechanism is the suppression of insulin-like growth factor 1 (IGF-1), a downstream effector of testosterone signaling that supports muscle regeneration and anabolic activity. ADT-induced reductions in IGF-1 expression impair satellite cell activation and protein accretion, exacerbating anabolic resistance and muscle atrophy [11]. Within months of ADT initiation, patients exhibit measurable reductions in lean body mass, with losses primarily concentrated in the appendicular muscles responsible for postural control and ambulation [12].

The resulting sarcopenia is not merely a function of muscle atrophy, but also reflects functional impairments in neuromuscular coordination. Studies using electromyographic and isokinetic testing demonstrate decreased muscle contractility, slower motor unit recruitment, and reduced peak force generation in men undergoing ADT [13]. These changes translate clinically to slower gait speed, reduced stride length, and diminished balance confidence, all of which are predictive of falls in elderly populations [14].

Further compounding this decline is the redistribution of body composition. ADT leads to increased visceral adiposity, which exacerbates insulin resistance and chronic inflammation. These metabolic alterations negatively influence muscle quality, further weakening musculoskeletal integrity [15]. Inflammatory cytokines such as IL-6 and TNF- α , which are elevated in hypogonadal states, have also been shown to accelerate muscle catabolism and interfere with neuromuscular junction signaling [16].

In longitudinal cohort studies, the loss of muscle mass and strength is independently associated with a higher incidence of falls among men receiving ADT, even after adjusting for age, comorbidities, and baseline functional status. Notably, the rate of decline is steeper in older adults, underscoring the vulnerability of geriatric patients whose baseline reserves are already diminished [17].

Sarcopenia, though clinically significant, often goes unrecognized in routine prostate cancer care. Many patients begin ADT without receiving proper referrals for physical therapy, and strategies like resistance training and nutritional support remain underused. Since early muscle loss is closely linked to an increased risk of falls, survivorship protocols should incorporate consistent evaluations of muscle function—such as testing grip strength or measuring gait speed—to enhance patient outcomes.

Neurostructural and Functional Brain Changes

In addition to its musculoskeletal consequences, ADT has been implicated in neuroanatomical and functional brain changes that further elevate fall risk. Androgen receptors are abundantly expressed in regions involved in motor coordination and executive function, including the cerebellum, hippocampus, and prefrontal cortex [18]. The removal of androgenic support through ADT has been shown to alter both the structure and function of these regions, contributing to motor instability and cognitive decline [19].

Neuroimaging studies have revealed significant reductions in cerebellar volume in men undergoing ADT, along with increased ventricular enlargement and cortical thinning, particularly in frontal and parietal lobes [20]. These changes correlate with clinical deficits in postural stability and visuospatial processing, both of which are essential for fall avoidance [21]. Furthermore, ADT disrupts white matter integrity, particularly within the corpus callosum and superior longitudinal fasciculus—fiber tracts that coordinate bilateral motor control and attention [22]. Advanced neuroimaging studies using diffusion tensor imaging (DTI) confirm reduced fractional anisotropy in fronto-cerebellar tracts, while arterial spin labeling MRI demonstrates decreased cerebral perfusion in prefrontal areas of ADT-treated patients, both of which are associated with impaired gait adaptation and executive dysfunction [23][24].

In addition to cognitive and motor planning deficits, ADT has also been linked to impairments in visual-spatial orientation and proprioception. These deficits are believed to result from functional disruption of the cerebellar-parietal circuits and occipital cortex, where androgen receptors influence visuomotor integration and spatial mapping [25]. Clinically, patients report increased missteps, difficulty with depth perception, and an elevated risk of veering or bumping into stationary objects. Gait analysis studies confirm increased mediolateral sway and stride variability in men undergoing ADT, particularly during tasks that require obstacle navigation or changes in direction [26]. Such impairments in environmental awareness and body position perception contribute

independently to fall risk and often precede overt cognitive symptoms. Early identification through dynamic balance testing or spatial orientation tasks may facilitate preemptive intervention.

At a molecular level, testosterone depletion induces neuroinflammation, upregulating cytokines such as interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α), which impair synaptic plasticity and neural signaling [27]. Chronic exposure to elevated glucocorticoid levels in hypogonadal states may also contribute to hippocampal atrophy and dysregulation of the hypothalamic-pituitary-adrenal axis, further exacerbating cognitive vulnerability [28].

Clinically, these neurobiological changes manifest as slower reaction times, impaired executive function, memory deficits, and decreased dual-task performance—such as walking while conversing or navigating uneven terrain [29]. These deficits not only increase the likelihood of falls but also delay recovery responses, increasing the probability of injury once imbalance occurs.

Although cognitive decline has traditionally been considered a long-term effect of ADT, recent prospective studies suggest that impairments in attention and processing speed can be detected within the first six months of therapy initiation. Subtle motor planning deficits, attributed to cerebellar disruption, may appear even earlier and remain unrecognized in standard clinical evaluations [30]. These findings support the need for preemptive neurological screening prior to ADT and regular follow-up assessments throughout its course.

Despite these risks, cognitive and neuro-motor side effects are seldom discussed in initial treatment planning for prostate cancer. Incorporating functional cognitive tests, such as the Trail Making Test or dual-task gait assessments, into clinical practice may help identify patients at heightened risk and guide early intervention strategies [31].

Executive Dysfunction, Memory Impairment, and Behavioral Sequelae

Beyond its structural effects on the brain, ADT has been strongly associated with cognitive decline that can significantly impair fall avoidance behavior. Cognitive domains most affected include attention, executive function, and working memory—each of which plays a pivotal role in mobility and hazard detection [32]. These impairments can manifest independently of mood disorders or overt neurological disease, making them challenging to detect without formal neurocognitive evaluation [33].

Executive dysfunction impairs the ability to plan and adjust gait in response to environmental stimuli, while lapses in attention increase the likelihood of tripping on uneven surfaces or misjudging spatial orientation. Deficits in working memory and processing speed can delay reaction times during unexpected balance challenges [34]. Studies using dual-task paradigms, such as walking while performing cognitive tasks, consistently show decreased gait stability and stride variability in men on ADT compared to matched controls [35].

These changes are partly attributable to the depletion of testosterone and its neuroprotective metabolites such as estradiol and dihydrotestosterone, which modulate synaptic activity, cerebral blood flow, and neurotransmitter balance [36]. ADT reduces hippocampal and cortical glucose metabolism, disrupts cholinergic signaling, and promotes oxidative stress, all of which contribute to progressive cognitive deterioration [37]. These cholinergic effects are partly mediated by the

loss of estradiol, a neuroprotective metabolite of testosterone that enhances acetylcholine synthesis. Reduced estrogenic activity impairs muscarinic and nicotinic receptor signaling, diminishing spatial awareness and attentional control critical for fall prevention [38]. Functional MRI studies reveal decreased activation in prefrontal and parietal regions during tasks requiring sustained attention or spatial reasoning in patients receiving ADT [39].

While often subtle, cognitive symptoms may emerge early in therapy and progressively worsen over time. In a prospective cohort study, men receiving ADT exhibited significant declines in verbal memory and executive functioning within six months, with additional deterioration over the following year. Notably, these declines occurred even in patients without baseline cognitive impairment, suggesting that ADT acts as an independent risk factor for neurocognitive dysfunction [32].

Behavioral symptoms may also co-occur, including apathy, irritability, and decreased initiative, which reduce engagement in physical activity and increase sedentary behavior. These psychological sequelae create a negative feedback loop, as physical deconditioning further impairs gait and balance, compounding fall risk [40].

In addition to direct cognitive and motor impairments, ADT contributes to systemic symptoms that indirectly elevate fall risk, including fatigue, anemia, and sleep disturbances. Testosterone deprivation reduces erythropoietin activity, leading to normocytic anemia in a substantial proportion of men undergoing long-term therapy [41]. Anemia is associated with decreased oxygen delivery to muscle and brain tissue, resulting in physical exhaustion, impaired vigilance, and delayed motor responses. ADT is also linked to circadian disruption and reduced sleep efficiency, mediated in part by hormonal imbalance and changes in melatonin secretion [42]. Sleep fragmentation, particularly when combined with daytime fatigue and poor concentration, contributes to gait instability and impairs decision-making during ambulation [43]. While these symptoms may appear nonspecific, their cumulative effect is a substantial increase in fall risk, especially in frail or multimorbid patients.

Despite accumulating evidence, cognitive monitoring remains rare in the routine care of men undergoing ADT. Incorporating validated tools such as the Montreal Cognitive Assessment (MoCA) or computerized attention tests into baseline and follow-up visits could facilitate early detection [33]. Cognitive training programs and pharmacological interventions targeting cholinergic pathways may also hold promise in preserving executive function and mobility in this vulnerable population [44].

Osteoporosis, Skeletal Fragility, and Fracture Risk

ADT significantly accelerates bone mineral loss in men with prostate cancer, placing them at high risk for osteoporosis and related fractures. Testosterone plays a pivotal role in maintaining bone remodeling equilibrium by promoting osteoblastic activity and inhibiting osteoclast-mediated resorption [45]. With ADT, the abrupt reduction in androgen levels disrupts this balance, leading to decreased bone formation, increased resorption, and overall skeletal fragility [46].

Clinical data indicate that up to 20% of men on long-term ADT develop osteoporosis, with bone mineral density (BMD) declines detectable as early as 6 to 12 months after treatment initiation [47]. The lumbar spine, femoral neck, and total hip are particularly affected, and reductions in BMD in these areas are strongly predictive of both low-trauma and vertebral fractures [48]. Men undergoing ADT have a 30–50% higher

relative risk of fracture compared to age-matched prostate cancer patients not receiving hormonal therapy [49].

Importantly, fractures in this population are often precipitated by low-energy mechanisms, such as simple ground-level falls, and are associated with high morbidity. Hip fractures carry a particularly poor prognosis, with up to 30% mortality at one year and significant declines in independence among survivors [49]. Vertebral fractures, though less immediately disabling, are frequently underdiagnosed and contribute to kyphosis, gait instability, and chronic pain—all of which further increase fall risk [50].

The effects of ADT on bone are not limited to mineral density alone. Microarchitectural deterioration, including trabecular thinning and loss of connectivity, compromises bone strength independent of BMD [51]. High-resolution peripheral quantitative CT (HR-pQCT) scans have revealed deterioration in trabecular architecture and cortical thickness that are not captured by conventional DEXA imaging. Concurrently, elevated bone turnover markers such as serum C-terminal telopeptide (CTX) and procollagen type I N-terminal propeptide (PINP) may serve as sensitive indicators of skeletal fragility in this population [52]. Furthermore, ADT exacerbates secondary risk factors for bone loss, including vitamin D deficiency, decreased physical activity, and nutritional insufficiency [53]. Hypogonadal states also increase parathyroid hormone levels, further promoting calcium loss from the skeleton [54].

Despite these risks, osteoporosis screening remains underutilized in men undergoing ADT. Studies suggest that fewer than 15% of these patients receive a baseline DEXA scan before starting therapy, and even fewer are monitored longitudinally or receive anti-resorptive treatment [55]. Bisphosphonates, denosumab, and lifestyle interventions such as resistance training and vitamin D supplementation have all been shown to attenuate ADT-induced bone loss and should be integrated into standard treatment protocols for prostate cancer survivors on hormonal therapy [56].

Cardiovascular Autoregulation and Orthostatic Risk

In addition to musculoskeletal and neurocognitive impairments, ADT may increase fall risk through disruption of cardiovascular homeostasis. Testosterone plays a regulatory role in maintaining vascular tone, autonomic balance, and endothelial function. Its suppression through ADT is associated with increased arterial stiffness, reduced baroreflex sensitivity, and impaired nitric oxide-mediated vasodilation, which together contribute to orthostatic hypotension [57].

Orthostatic hypotension is defined as a sustained drop in systolic blood pressure of at least 20 mmHg or diastolic pressure of 10 mmHg upon standing, and is a well-established risk factor for falls in elderly adults. In the context of ADT, impaired cardiovascular reflexes may result from both hormonal and structural vascular changes. Longitudinal data indicate that men receiving ADT have a significantly higher incidence of orthostatic symptoms, including lightheadedness, dizziness, and unsteadiness during postural transitions [58].

Furthermore, ADT has been associated with subclinical autonomic dysfunction, including reduced heart rate variability and attenuated sympathetic responsiveness [59]. These physiological changes are particularly hazardous for older adults with pre-existing hypertension, diabetes, or atherosclerotic disease, who may have diminished

cardiovascular reserves. Falls in this context may be sudden, unpredictable, and occur independently of muscle or cognitive deficits.

Although orthostatic hypotension is clinically significant, it is seldom evaluated in men starting ADT. Regular assessments of orthostatic blood pressure, both at baseline and periodically, combined with education on fall risks, could aid in identifying individuals at higher risk. Moving forward, guidelines should integrate cardiovascular autonomic screening into standard survivorship protocols, particularly for those with pre-existing vascular conditions.

President Joe Biden's Falls, Prostate Cancer Diagnosis, and the Public Recognition of Fall Risk in Older Adults

In recent years, fall risk among the elderly has garnered significant public attention, notably due to highly visible incidents involving President Joe Biden. Throughout his presidency, Biden experienced multiple public falls and near-falls, including:

- March 19, 2021: Biden stumbled multiple times while ascending the stairs of Air Force One, which his team attributed to windy conditions [60].
- June 18, 2022: He toppled over while attempting to stop his bicycle near his Delaware beach house during a ride [61].
- June 1, 2023: Biden tripped over a sandbag and fell on stage during the U.S. Air Force Academy commencement ceremony [62].
- September 26, 2023: He slipped while deboarding Air Force One in Michigan, despite efforts by his team to prevent such incidents such as wearing slip resistance sneakers and using the short stairs to board and deboard the plane [63].

These events, widely disseminated through media coverage, sparked discussions about the intersection of aging, motor function, and health transparency in leadership roles.

The relevance of these incidents intensified following the disclosure of President Biden's prostate cancer diagnosis, which was notably advanced at the time it was publicly revealed (Gleason score 9, grade group 5 with bone metastasis) [9]. The late-stage nature of the diagnosis raised questions, particularly in light of the typically proactive and comprehensive medical oversight afforded to sitting presidents. Although the specific details of his treatment have not been disclosed, the standard of care for this level of disease severity nearly always involves ADT. ADT is known to produce a range of side effects that directly impact balance and neuromuscular function. These include sarcopenia, cerebellar degeneration, visual-spatial disorientation, orthostatic hypotension, and executive dysfunction. Each of these can independently increase the risk of falls and may also interact in a synergistic manner among older individuals.

A shift in public perception occurred during the June 27, 2024 presidential debate, when President Biden appeared to lose his train of thought, gave disjointed responses, and struggled visibly with articulation and focus [64]. These cognitive lapses prompted renewed scrutiny over his capacity to serve and were followed by his formal withdrawal from the 2024 presidential race on July 21, 2024 [65]. Taken in conjunction with his earlier public falls and cancer diagnosis, these developments emphasize a broader need to recognize fall risk and cognitive impairment as visible

and consequential aspects of aging, especially when they are amplified by systemic cancer therapies.

Beyond President Biden's case, these events present a critical opportunity to refocus clinical and public health efforts on fall prevention in older adults. According to the Centers for Disease Control and Prevention (CDC), one in four adults over the age of 65 falls each year, making falls the leading cause of injury-related deaths within this demographic [66]. Despite this, fall risk screening remains inconsistently applied, especially in patients receiving therapies like ADT that directly affect muscle function, coordination, and cognition [67].

Preventative Interventions and Monitoring Strategies

Given the multifactorial nature of fall risk in men undergoing ADT, preventative strategies must be comprehensive, individualized, and implemented early in the treatment course. The mechanisms contributing to instability—ranging from sarcopenia and cerebellar dysfunction to cognitive decline and orthostatic hypotension—necessitate a multidisciplinary approach that includes proactive screening, patient education, lifestyle modification, and clinical interventions.

1. Musculoskeletal Interventions

Resistance and balance training have consistently been shown to reduce fall incidence and improve physical function in older adults. Programs that incorporate progressive weight-bearing exercises can mitigate ADT-induced muscle atrophy and preserve gait stability [68]. Physical therapy referrals should be initiated at the onset of ADT, particularly for individuals with pre-existing mobility limitations. Nutritional counseling with attention to protein intake and caloric adequacy further supports muscle maintenance.

2. Nutritional Interventions

Alongside protein intake and resistance-based rehabilitation, nutritional strategies that support muscle health at the molecular level may benefit men receiving ADT. One such target is Sirtuin 1 (SIRT1), an enzyme involved in mitochondrial function, muscle cell development, and the reduction of oxidative stress. SIRT1 activity is linked to improved muscle integrity, heart health, and cognitive function—areas commonly affected by testosterone suppression. Early studies suggest that compounds like resveratrol and other calorie restriction mimetics can activate SIRT1 and help limit muscle loss and inflammation [69].

3. Bone Health Management

All patients beginning ADT should undergo baseline dual-energy X-ray absorptiometry (DEXA) scanning to assess bone mineral density (BMD), with follow-up every 12–24 months depending on risk factors. Vitamin D and calcium supplementation are first-line prophylactic measures. For patients with low BMD or prior fractures, antiresorptive agents such as bisphosphonates or denosumab have demonstrated efficacy in preventing ADT-associated osteoporosis and fragility fractures [70].

4. Cognitive and Gait Screening

Neurocognitive assessments using tools such as the Montreal Cognitive Assessment (MoCA) or Trail Making Test should be conducted at baseline and repeated periodically. Dual-task gait testing and the Timed Up and Go (TUG) test can reveal early deficits in motor planning and executive function integration [71]. Identifying impairments in these

domains allows for early referral to cognitive rehabilitation or occupational therapy, both of which can reduce fall risk.

5. Orthostatic Hypotension Monitoring

Orthostatic blood pressure measurements should be conducted routinely in patients on ADT, particularly in those with cardiovascular comorbidities or symptoms of dizziness. Identification of orthostatic hypotension may prompt medication review, increased hydration strategies, compression therapy, or referral to cardiology for autonomic evaluation [72].

6. Polypharmacy and Medication Reconciliation

ADT is often administered in the context of multimorbidity. Routine review of medications, particularly sedatives, antihypertensives, and anticholinergics, can help identify agents that contribute to fall risk through sedation or impaired coordination. When appropriate, deprescribing or dose reduction should be considered [73].

7. Environmental and Behavioral Interventions

Fall risk assessments should include home safety evaluations to identify hazards such as loose rugs, poor lighting, or unanchored furniture. Education on assistive device use, footwear selection, and safe ambulation techniques should be reinforced during follow-up visits. Sleep hygiene and fatigue management strategies may further reduce attention lapses and daytime instability [74].

Conclusion

ADT is essential in managing advanced prostate cancer, but its widespread physiological effects significantly increase fall risk in older men. This risk stems from a combination of sarcopenia, cerebellar and cortical degeneration, cognitive decline, orthostatic instability, and bone loss. These factors interact to impair balance, mobility, and reflexive control, increasing both the likelihood and severity of falls.

Despite strong evidence for these risks, fall prevention is still underused in prostate cancer care. Survivorship strategies must include early screening for muscle loss, neurocognitive decline, and cardiovascular dysregulation, alongside interventions that address each domain. Evidence-based approaches such as resistance training, nutritional support, bone-protective agents, cognitive assessments, and orthostatic monitoring should be implemented as standard practice.

High-profile cases like that of President Joe Biden have brought public attention to the real-world consequences of unaddressed functional decline. These cases emphasize the urgent need for integrated, multidisciplinary care that prioritizes safety, independence, and quality of life in men undergoing ADT.

Declarations

This manuscript fully adheres to the journal's submission policies and guidelines, and the author confirms that the content has not been previously published or submitted elsewhere. There are no conflicts of interest to disclose. As this work is a literature review that does not involve human participants, animal subjects, or plant materials, ethical approval, participant consent, and related disclosures are not applicable. No personal data, images, or videos of individuals are included in the manuscript. All data referenced are from publicly available sources, and no new datasets were generated. The author did not receive any external

funding for this research. Grammarly AI was used solely for editing grammar, syntax, and paragraph clarity; it did not contribute to the conceptual content or originality of the work.

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