Review Article

# Neurodegeneration: A Convergence Hypothesis Linking Chronic Low-Dose Diagnostic Radiation to Accelerated Decline

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**Neurodegenerative diseases are not an inevitable consequence of aging, and understanding their etiology is essential for effective prevention and treatment. This hypothesis proposes that neurodegeneration arises from a convergence of stress across three systems that work together to maintain brain health: mitochondrial function, protein and waste management, and neuroimmune regulation. While several environmental and systemic stressors — such as vascular insufficiency and circadian disruption — have been identified, little attention has been directed toward how repeated low-dose diagnostic radiation contributes to this burden by incrementally taxing these pathways over the years. This systems view reframes neurodegenerative risk as measurable and modifiable, opening the door to individualized assessment and targeted mitigation.**

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## Introduction

Low-dose ionizing radiation is a plausible and significant contributor to neurodegenerative risk not because it causes dramatic injury, but because it can place ongoing stress on all of the systems the brain relies on to remain healthy over time. Unlike higher radiation doses, it does not typically cause cell death or genetic mutations. Instead, its potential risk lies in something more subtle: the creation of ongoing biological stress. [1] The brain depends on a delicate balance of energy production, waste removal, immune regulation, and the ability to repair these systems. Small disruptions can usually be compensated for, especially in the short term. However, repeated exposures over many years may

gradually weaken these protective mechanisms, setting the stage for accelerated decline once compensatory capacity is exceeded.

When the brain's ability to make repairs is slowly eroded, these critical systems can begin to fail together. [2][3] Oxidative stress, often cited as a hallmark of neurodegeneration, is treated here not as a separate initiating pathway but as a downstream consequence of dysfunction across multiple repair systems. When mitochondrial inefficiency, proteostatic failure, and chronic immune activation converge, they generate reactive oxygen species and redox imbalance that further degrade cellular resilience — helping to explain why harmful processes become increasingly difficult to reverse once underway.

This convergence model helps explain why neuropathology can be present without significant cognitive impairment and why not all individuals with similar exposures develop neurodegeneration. Isolated stressors may be absorbed, but the simultaneous erosion of multiple protective systems appears to be a critical factor in the transition from compensated dysfunction to progressive disease. It also explains why no single insult, pathway or exposure can reliably predict neurodegeneration. Regional variation in dementia may arise from how different cultures and environments differentially tax the three pathways that support brain resilience.

The increasing global prevalence of neurodegenerative diseases among the elderly – and the concerning trend of early-onset neurodegeneration – suggest that systems-level stressors associated with modern exposures and habits may be contributing factors. [4] This could include everything from disrupted sleep patterns or overtaxed immune systems to increased medical and dental X-ray exposure. Identifying and addressing one or more of these key stressors could open the door to true preventive strategies that reach beyond the existing recommendations of maintaining a healthy diet and lifestyle, which even when strictly practiced have failed too many in their later years.

While multiple environmental and physiological stressors have been identified as contributing to compromised brain functions, this paper will focus on a potential stressor that has received little study: lifelong head exposure to low-dose ionizing radiation. Emphasis is placed on the escalating radiation exposures emerging in dental X-ray technology, as well as the widespread use of head computed technology (CT) imaging – each of which disproportionately affect older adults.

This hypothesis does not propose a new disease mechanism, but reframes existing mechanisms within a cumulative, systems-level model that may help explain variability in neurodegenerative risk and resilience.

## Hypothesis evolution

The hypothesis that repeated head exposure to low-dose ionizing radiation may contribute to neurodegenerative disease risk was first proposed by the author in 2011 in the context of dental X-ray imaging and Alzheimer's disease [5] and later expanded in 2020 to encompass low-dose X-ray imaging more broadly and its relevance to other neurodegenerative conditions. [6] The present paper represents a third, hypothesis-driven examination of this proposed pathway. It synthesizes newer biological insights and contemporary exposure patterns to reassess the plausibility, scope, and potential public health relevance of cumulative head exposure to low-dose ionizing radiation as a contributor to neurodegenerative processes. The author's aim is not to dispute the clinical value of diagnostic imaging when medically necessary, but to encourage focused investigation of long-term neurological outcomes that may not yet be adequately captured in existing risk-benefit assessments.

## Three core systems maintaining brain health

On a systems level, the three major pathways to healthy neurological function are:

1. **Mitochondrial capacity:** Synaptic signaling is energy intensive, which is why it is commonly understood that the brain, while representing roughly two percent of body weight, consumes 20 percent of the body's energy. When the energy supply is chronically constrained, neuronal resilience declines, increasing vulnerability to downstream dysfunction when additional stressors are present. Mitochondrial impairment is a consistent feature across virtually all neurodegenerative diseases. [7]
2. **Proteostasis:** This is the cell's system for maintaining healthy proteins and clearing damaged or misfolded ones. When these maintenance and clearance pathways are overwhelmed, abnormal proteins accumulate, contributing to neurodegenerative processes such as those seen in Alzheimer's and Parkinson's disease, Amyotrophic Lateral Sclerosis, and Frontotemporal Dementia, among others. [8]
3. **Neuroimmune regulation:** The brain's support cells — particularly microglia and astrocytes — coordinate immune surveillance and tissue repair. When chronically overtaxed, they can shift into a pro-inflammatory state, leading to immune dysregulation and disruption of the blood–brain barrier. Similar patterns are implicated in conditions such as multiple sclerosis and vascular dementia. [9]

Neurodegeneration arises when these three core systems experience sustained stress. The outcomes depend not on any single insult, but on the brain's repair and adaptive capacity over time.

## Why convergence – not isolated injury – matters

The failure to find a single cause for a neurodegenerative disease – with the exception of diseases that can be traced to a single gene mutation such as Huntington's – is not due to a lack of investment or effort. Many promising leads have been pursued and translated into therapeutic trials, only to deliver limited or no clinical benefit. When a potential cause has been identified, researchers are further baffled when not everyone equally exposed develops a neurodegenerative disease. Even more confounding is the fact that not everyone with brain pathology associated with a neurodegenerative disease -- such as individuals with pronounced amyloid brain plaque accumulation -- demonstrate corresponding cognitive impairment. These apparently conflicting observations can be reconciled by a model of neurodegeneration that depends on the convergence of multiple compromised pathways rather than a single insult.

With this model in mind, variations in outcomes may depend upon individual biological makeup, genetic influences, environmental conditions and health history, each of which shape baseline capacity to buffer stressors as they add up over time.

## Genetics do not equal destiny

Most neurodegenerative diseases are not inherited. Only a small percentage — generally estimated at 5 to 10 percent — can be traced to specific gene mutations. Even then, however, the genes involved do not introduce new disease mechanisms. Instead, they weaken one or more of the same biological systems that are critical for maintaining brain health. Some of the most commonly known genetic variants reveal the mechanisms at work: [10]

- **Alzheimer's disease**: The best-known genetic risk factor, the *APOE-ε4* variant, influences how the brain manages fats, clears waste proteins such as amyloid, and regulates inflammation, thus impacting two core pathways. [10]
- **Parkinson's disease**: Genetic changes in genes such as *LRRK2* and the *PARK* genes are linked to mitochondrial dysfunction and impaired cellular stress responses – also disrupting two pathways. [11]

- **Amyotrophic lateral sclerosis:** Mutations involving *SOD1* interfere with the brain's ability to manage oxidative damage. In each case, the gene does not act alone; it erodes systems that normally protect neurons over time, a reminder that some genetic changes can trigger downstream cascades that progressively erode compensatory capacity. [12]

Seen this way, genetics helps explain why people respond differently to similar exposures or age-related changes. These individuals begin life with less resilient protective systems, leaving them more vulnerable as stressors that add up across decades. Genetic risk, then, does not contradict a convergence model of neurodegeneration — rather it reinforces it, showing how multiple weakened pathways can combine to push the brain past a threshold from compensation to disease. [2]

## Environmental exposures associated with neurodegenerative risk

Humans have evolved a number of repair mechanisms that can address mitochondrial injury, protein dysregulation, or immune responses that threaten brain health. However, these defenses developed to address intermittent, naturally occurring stressors and may be less effective when challenged by chronic or novel exposures. Several such exposures have been linked to neurodegeneration, including:

- **Pesticides and herbicides**, such as organophosphates, paraquat, and rotenone, have been linked to higher Parkinson's disease incidence in agricultural workers and in individuals living near golf courses or farmland, where runoff chemicals can enter the water supply. Their effects are understood not as the result of a single exposure, but as chronic, low-level neurotoxic stressors.
- **Heavy metals**, including lead, mercury, manganese, and arsenic, can cause non-acute, cumulative damage to mitochondrial function, promote oxidative stress, and disrupt protein handling and immune regulation – a triple hit.
- **Air pollution:** There is increasing evidence in dementia and Parkinson's research that chronic exposure to pollutants such as fine particulate matter can induce oxidative stress, systemic inflammation, and compromise the blood–brain barrier. [3]

*Two other types of exposures that are not unique to modern life also deserve mention, as they are still external stressors that place long-term pressure on the brain's repair systems:*

- **Traumatic brain injury:** Repetitive or even mild traumatic brain injury has been associated with delayed neurodegenerative effects, sometimes emerging years after the initial injury.

- **Chronic infections:** Long-lasting infections such as herpesviruses or periodontal disease can act as environmental stressors. They keep the immune system in a low-grade activated state, increasing oxidative load and placing ongoing pressure on all three pathways, gradually eroding the repair capacities that keep these pathways stable. [9]

## Frequent, life-long head exposure to low-dose ionizing radiation

While the above factors have been the subject of extensive study, one chronic exposure experienced by a majority of people has received comparatively little attention: head exposure to low-dose ionizing radiation accumulated over a lifetime of dental imaging, which may be compounded by other exposures such as chiropractic radiography or head CTs. [5][6]

Low-dose ionizing radiation differs from many established neurotoxic exposures in an important way: it is not typically lethal to cells, nor does it reliably produce immediate or observable neurological injury. For this reason, its long-term neurological effects have been assumed to be negligible. However, the absence of acute harm does not imply biological neutrality. At low doses, ionizing radiation primarily acts as a source of recurrent cellular stress, activating repair, antioxidant, and immune-related processes that must be repeatedly engaged over time. What makes this exposure particularly relevant is that they are frequent throughout a lifetime starting at a young age and often occur in a series of exposures in rapid succession. This challenges each pathway at the same time, progressively straining the very repair mechanisms needed to sustain them. Here is how each system is compromised:

- **Mitochondrial stress:** Low-dose ionizing radiation produces reactive oxygen species that place a recurring burden on mitochondria. [7] While cells customarily neutralize this stress, repeated exposure increases the likelihood of cumulative mitochondrial inefficiency rather than acute failure. Over time, this can reduce the energy margin available to neurons, leaving them less able to respond to additional metabolic or physiological challenges. Such impairment need not be severe to have functional consequences; even small, chronic reductions in energy efficiency may cause dysfunction.
- **Disruption of protein handling and waste clearance:** As repair systems become strained, even modest increases in oxidative stress can further burden the machinery responsible for protein repair, refolding, and clearance. [8] When these systems are repeatedly engaged without full recovery, their efficiency may decline, allowing damaged or misfolded proteins to evade clearance. This feedback

loop is consistent with the slow accumulation of abnormal proteins observed long before clinical symptoms appear in many neurodegenerative diseases.

- **Neuroimmune activation:** Even in the absence of tissue injury, low-dose radiation can activate immune signaling pathways. Repeated activation may shift neuroimmune cells toward a chronically primed state, increasing baseline inflammation and diverting immune capacity away from neural maintenance and repair. [9] Over time, this may weaken blood–brain barrier integrity and amplify inflammatory signaling in response to otherwise minor insults, creating conditions that favor progressive neurological damage. Recent studies show that older adults who receive recommended vaccines — including influenza, pneumococcus, diphtheria, tetanus, herpes zoster and pertussis (Tdap) — have a significantly lower risk of developing dementia, likely due to reduced infection-related immune activation. [13]

What distinguishes low-dose ionizing radiation incurred from regular, life-long dental radiation from many other neurotoxic exposures is not the severity of damage caused at any one time, but the frequency with which repair systems must be mobilized. DNA repair pathways, antioxidant defenses, mitochondrial quality control, and immune regulation mechanisms are repeatedly engaged, often without sufficient recovery time.

These systems evolved to handle occasional injury, not persistent, low-level stress sustained across decades. As repair efficiency gradually declines, damage that was once fully reversible may become only partially resolved. This creates a cumulative burden that affects multiple systems simultaneously, increasing the likelihood that compensatory capacity will eventually be exceeded.

Individual differences in repair efficiency — shaped by genetics, early-life health, and cumulative stress history related to occupations, environments or stress — help explain why similar radiation exposures can result in widely different neurological outcomes.

Further, changes in dental technology and practices, particularly the introduction and proliferation of cone beam computed tomography (CBCT), which provides three-dimensional dental imaging, as well as medical imaging advances such as head CTs, put senior populations at higher risk because they are more likely to undergo these procedures.

## Changes in dental and medical imaging: implications for older adults

Over the past several decades, patterns of head exposure to ionizing radiation have changed in ways that may be particularly relevant to shifting neurodegenerative risk. In dentistry, the transition from film to digital radiography substantially reduced exposure, but the subsequent proliferation of CBCT, often for routine planning or evaluation, delivers much more radiation than typical intraoral imaging. Since the Food and Drug Administration (FDA) approved CBCT in 2004, its use has expanded beyond specialist settings into general dental practice. Although CBCT has been heralded for delivering lower radiation doses than conventional medical CT scans, it typically exposes a much larger volume of cranial tissue than traditional two-dimensional radiography, and, in some configurations, can approach orders of magnitude higher effective doses. Its use in orthodontics, diagnostic evaluations, complex restorative procedures, and implant installation may involve multiple exposures -- planning, implementation and follow-up – within a single treatment plan.

In addition to dose considerations, volumetric exposure itself may be biologically relevant. Irradiating a larger region of the head engages immune and support cells across a broader territory, increasing the cumulative burden on neuroimmune regulation. Rather than responding to a localized insult, repair and immune systems may be required to address diffuse, low-level stress across multiple regions simultaneously — a demand that may be difficult to meet in aging neural tissue. This may be especially relevant to today's senior population, many of whom were also exposed to higher dental radiation prior to the development of digital X-rays. Although the American Dental Association (ADA) has long recommended that imaging be ordered only after a clinical evaluation, the expanded availability of CBCT has led to its increasing use for routine screening and treatment planning. The 2026 ADA guidance reiterates that CBCT should be used only when clinically necessary, reflecting concern that current practice patterns may not align with long-standing principles of radiation minimization. [14]

Chiropractic care also contributes to cumulative exposure, as many treatment plans include imaging of the skull and cervical spine. Radiographs are obtained in nearly one-third of new chiropractic patients, with some clinicians using additional films to follow progress, although routine repeat imaging is not widespread. [15] Because chiropractic care is widely used in the United States and older adults represent a substantial proportion of patients — particularly those seeking care for chronic neck and back pain,

arthritis, and mobility limitations [16] — this group may be especially vulnerable to cumulative head and neck imaging over time.

Advances in medical imaging since the introduction of head CTs in the 1970s likewise have led to their more frequent use in emergency and inpatient settings. Older adults are disproportionately affected by conditions that prompt such imaging due to falls, head trauma, stroke evaluation, and altered mental status. As a result, cumulative head exposure to ionizing radiation tends to increase with age, often at a time when mitochondrial efficiency, immune regulation, and repair capacity are already declining.

This convergence is particularly relevant within a systems-level framework. Imaging-related radiation exposures that may be well tolerated earlier in life could place a greater burden on aging neural systems that have less reserve and reduced capacity for full repair. This process has the potential to accelerate the transition from subclinical dysfunction to overt neurodegenerative disease.

## Discussion

Vascular insufficiency is a well-established contributor to neurodegenerative risk, impairing metabolic supply, destabilizing proteostatic mechanisms, and activating inflammatory cascades. These vascular strains are intensified by modern conditions such as sedentary behavior, metabolic overload, chronic stress, and environmental pollutants. [17] Chronic circadian disruption — reflected in sleep fragmentation, irregular meal timing, and inconsistent daylight exposure — is increasingly recognized as a parallel stressor. It impairs mitochondrial efficiency, alters protein-clearance rhythms, and promotes sustained neuroinflammation. [18] Together, these exposures illustrate a broader pattern: diverse modern stressors can converge on the same core maintenance systems and produce similar downstream effects when chronically engaged.

Together, vascular dysfunction, circadian disruption, and cumulative diagnostic radiation represent three of the most widespread modern stressors capable of taxing all three pathways simultaneously, making them particularly relevant to population-level neurodegenerative risk.

Within this context, cumulative exposure to low-dose diagnostic radiation represents a less examined but biologically plausible contributor. Although sublethal, repeated head and neck imaging activates DNA repair processes, generates low-level oxidative stress, and may incrementally burden the same pathways implicated in other modern stressors. The question is not whether low-dose ionizing radiation acts

alone, but whether it participates in the same multi-system erosion that characterizes other well-studied exposures.

This pattern is consistent with long-term observational evidence showing that individuals can remain cognitively intact even when one pathway is compromised, provided that other sources of chronic stress are minimized. Stable routines, sustained cognitive engagement, low inflammatory burden, and consistent metabolic patterns appear to preserve overall system integrity despite localized vulnerabilities. These findings reinforce the central premise of the convergence model: neurodegeneration emerges not from a single failing pathway but from the cumulative erosion of multiple maintenance systems over time.

A decades-long study of nuns offers a compelling illustration of how strong compensatory systems can protect cognitive function even in the presence of substantial neuropathology. This study combined early-life autobiographical essays, detailed health records, cognitive assessments, and postmortem brain analyses. Many participants who remained cognitively healthy into advanced age showed extensive amyloid plaques and neurofibrillary tangles at autopsy — levels that, in the general population, are often associated with dementia. Their resilience has been attributed to lifelong habits such as sustained cognitive engagement, stable metabolic patterns, and low levels of chronic inflammation — but their well-regulated lives supported these maintenance systems without major disruptions. In this light, the fact that neuropathology alone does not determine cognitive outcomes is not confounding, but rather predictable, and demonstrates that lifestyle patterns that maintain metabolic stability, waste clearance, and immune balance can play a decisive role.[19]

At the opposite end of the spectrum, Down syndrome illustrates how simultaneous stress across multiple protective systems can accelerate neurodegenerative vulnerability. Triplication of chromosome 21 increases production of amyloid precursor protein, placing a lifelong burden on protein management and waste-clearance pathways. Overexpression of genes involved in oxidative stress contributes to chronic mitochondrial strain and reduced energetic resilience. Neuroimmune regulation is also altered, with heightened immune activity evident from early life. Each of these alterations alone might be manageable, but together they narrow compensatory capacity over decades, leading to the near-universal early-onset development of Alzheimer-type pathology. Clinical symptoms then appear years earlier than the age at which Alzheimer's typically emerges in most adults. Down syndrome thus reinforces the central premise of this hypothesis: neurodegeneration emerges not from a single cause, but from the convergence of three key pathways.[20]

Between these two extremes lies the general population, where individuals differ widely in their baseline resilience and in the cumulative stress experienced over a lifetime. Genetic variants, early-life health, metabolic status, sleep quality, environmental exposures, infections, and lifestyle patterns all influence how much compensatory capacity a person begins with and how quickly it erodes. Although dementia is not a natural consequence of aging, advancing age increases the likelihood that all three protective pathways will be under strain at the same time. When these systems and their repair mechanisms are compromised together, age emerges as a risk factor not because it causes neurodegeneration directly, but because it marks the point at which compensatory capacity becomes harder to maintain.

Taken together, this systems-level framework suggests that everyday clinical decisions may influence neurodegenerative vulnerability by altering the cumulative load on these pathways. For example, routine annual checkups in older adults could include brief questions about sleep quality, regularity, and duration, with sleep studies pursued when indicated to identify and treat conditions such as sleep apnea that can impair nightly waste-clearance. Similarly, ensuring that older adults remain up to date on recommended vaccinations may help reduce their immunological burden by preventing infections that activate neuroimmune pathways. And when selecting dental restorations, particularly in seniors, clinicians and patients might weigh not only cost, appearance and durability but also the cumulative radiation burden of imaging modalities such as CBCT, potentially favoring options that minimize repeated cranial exposure.

## Conclusion

This hypothesis proposes that neurodegenerative diseases arise when core systems that sustain brain health can no longer compensate for cumulative stress. It further proposes that repeated exposure to low-dose ionizing radiation from dental imaging, chiropractic evaluations, and head CTs may contribute to this loss of resilience by persistently engaging all three pathways. Although each exposure is small, their cumulative biological impact over decades may narrow the brain's physiological reserve and accelerate decline. Whereas vascular and circadian disruptions are increasingly recognized as contributors to this systems-level strain, the potential impact of cumulative low-dose ionizing radiation has remained largely overlooked. Such a framework raises the possibility that neurodegenerative risk could one day be estimated — and mitigated — by assessing the status of these pathways on an individual basis and developing interventions that restore resilience where it is most compromised.

## Statements and Declarations

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Caroline C. Rodgers is an independent science theorist whose peer-reviewed work spans autism, neurodegeneration, and maternal and neonatal health. She explores the potential biological roots of public health issues that are incompletely explained by prevailing theories.

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## Declarations

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