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Life-Course Impact of Trauma on Stress Biology

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Abstract: This study investigates the impact of childhood adversity and elder abuse on cortisol regulation and long-term health outcomes in older adults. The objective was to assess how trauma across the life course influences hypothalamic-pituitary-adrenal (HPA) axis functioning, with cortisol patterns serving as biological markers. A systematic review methodology was applied, screening 312 published articles and identifying 75 studies that met the inclusion criteria. Results indicate that 68% of studies on childhood adversity reported significantly flatter diurnal cortisol slopes, while 61% of elder abuse studies documented elevated evening cortisol levels. Importantly, individuals with cumulative exposure to both early and late-life trauma exhibited a 35% greater reduction in cortisol variability compared to non-exposed peers, highlighting a compounded biological effect. Despite these consistent findings, 14% of included studies presented conflicting results, often due to variations in cortisol sampling protocols, self-reported adversity measures, or small sample sizes. These methodological inconsistencies represent a critical limitation, emphasising the need for standardised biomarker collection in future research. Overall, the study underscores the enduring influence of trauma on biology and provides evidence that cortisol dysregulation may serve as a measurable indicator of vulnerability to poor health outcomes in older adults.

Keywords: *Childhood Adversity, Elder Abuse, Cortisol Dysregulation, HPA Axis, Stress Biomarkers, Healthy Ageing.*

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I. INTRODUCTION

Trauma experienced across one's lifespan—beginning with childhood adversity and extending into elder abuse—can leave a lasting imprint on physiological systems, particularly the hypothalamic–pituitary–adrenal (HPA) axis [1], [2]. Cortisol, a primary marker of HPA axis activity, follows a diurnal rhythm that supports immune regulation, metabolism, and stress adaptation [3], [4]. Disruptions in these cortisol patterns are increasingly recognised as biological signatures of enduring stress, linking early abuse and neglect to adverse health outcomes in later life [5], [6].

Evidence from longitudinal and cross-sectional studies demonstrates that individuals exposed to childhood trauma often exhibit blunted diurnal cortisol slopes and altered cortisol awakening responses (CAR), persisting well into mid- and late-adulthood [7], [8]. For instance, Kuras et al. found in a healthy adult cohort that even low-to-moderate childhood adversity corresponded to significantly altered HPA activity [9]. Dimakakos et al., using data from the English Longitudinal Study of Ageing, reported that higher ACE scores were associated with lower cortisol upon waking and a flatter diurnal pattern, particularly in men [10]. These neuroendocrine alterations are believed to contribute to increased vulnerability to chronic illnesses—including cardiovascular disease, metabolic disorders, cognitive decline, and compromised immunity [11]–[13].

Understanding how elder abuse compounds HPA dysregulation may remain an emerging area of study. Approximately one in six older adults experiences some form of abuse—physical, emotional, or neglect—with serious health implications including depression, frailty, and even premature mortality [14], [15]. Although direct cortisol-related data are sparse in this demographic, evidence suggests

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that chronic stressors in ageing mirror earlier-life cortisol disruptions, reinforcing the notion of cumulative biological burden [16], [17]. Gaffey et al. proposed that resilience, mediated through psychosocial resources such as emotion regulation and social support, may modulate cortisol dynamics in older adults [18]. Additionally, elevated evening cortisol in elders has been linked to disrupted cognitive function [19], [20]. Figure 1 illustrates how childhood and elder abuse contribute to cortisol dysregulation, which in turn negatively impacts health outcomes in older adults.

From a mechanistic standpoint, early trauma may program HPA axis dynamics through lasting epigenetic modifications and microglial immune priming [21], [22]. Animal and human studies indicate that trauma can alter glucocorticoid receptor expression, reshape inflammatory responsivity, and compromise diurnal cortisol flexibility [23], [24]. Blunted or flattened cortisol responses are thought to reflect allostatic load—an erosion of adaptive physiological capacity under chronic stress that predisposes individuals to disease [25], [26]. Dynamic systems models of HPA regulation further suggest that timing and frequency of stress exposures can shift cortisol dynamics toward maladaptive states, especially when trauma spans multiple life stages [27], [28].

Despite these insights, the literature exhibits notable variability. Approximately 14% of studies report null or inconsistent findings, often due to methodological issues such as single-point cortisol sampling, retrospective adversity reporting, or small and unrepresentative samples [29]–[31]. Moreover, sex differences in stress responsivity—such as divergent diurnal cortisol slopes and CAR between men and women—remain underexplored, though emerging data suggest hormonal and psychosocial factors may underpin these disparities [32], [33]. Whitaker et al. also highlight the challenges in comparing cortisol findings due to inconsistent sampling protocols across studies [34]. Figure 2 illustrates how individuals without abuse history show a higher morning cortisol peak and a steeper decline across the day, while those with a history of abuse present a blunted slope, indicating impaired stress regulation.

This study makes a novel contribution by integrating evidence across life-course trauma—spanning both childhood adversity and elder abuse—and examining their cumulative impact on cortisol regulation and ageing health outcomes. By synthesising findings from diverse cohorts and methodologies, we aim to construct a comprehensive framework that positions cortisol dysregulation as a measurable biomarker of trauma exposure and its long-term health consequences. This integrative perspective also underscores the need for standardised cortisol collection protocols and inclusion of psychosocial resilience measures in future research [35], [36].

Furthermore, by promoting cortisol as both a mechanistic pathway and a practical marker for intervention, this work supports a trauma-informed model of healthy ageing—one that recognises early-life adversity and elder mistreatment as overlapping determinants of stress biology. Such a model may enable clinicians and policymakers to identify at-risk individuals earlier, tailor interventions aimed at restoring HPA flexibility and ultimately reduce cumulative health burden among ageing populations [37]–[39].

In summary, this introduction sets the stage for a study that critically examines how trauma across the lifespan—encompassing both early and later life adversity—disrupts cortisol dynamics. It emphasises the novelty of a holistic, cumulative approach, highlights methodological limitations in the field, and underscores the practical implications of cortisol as a target for resilience-building interventions. These insights are essential for interdisciplinary efforts across psychoneuroendocrinology, gerontology, and public health [40].

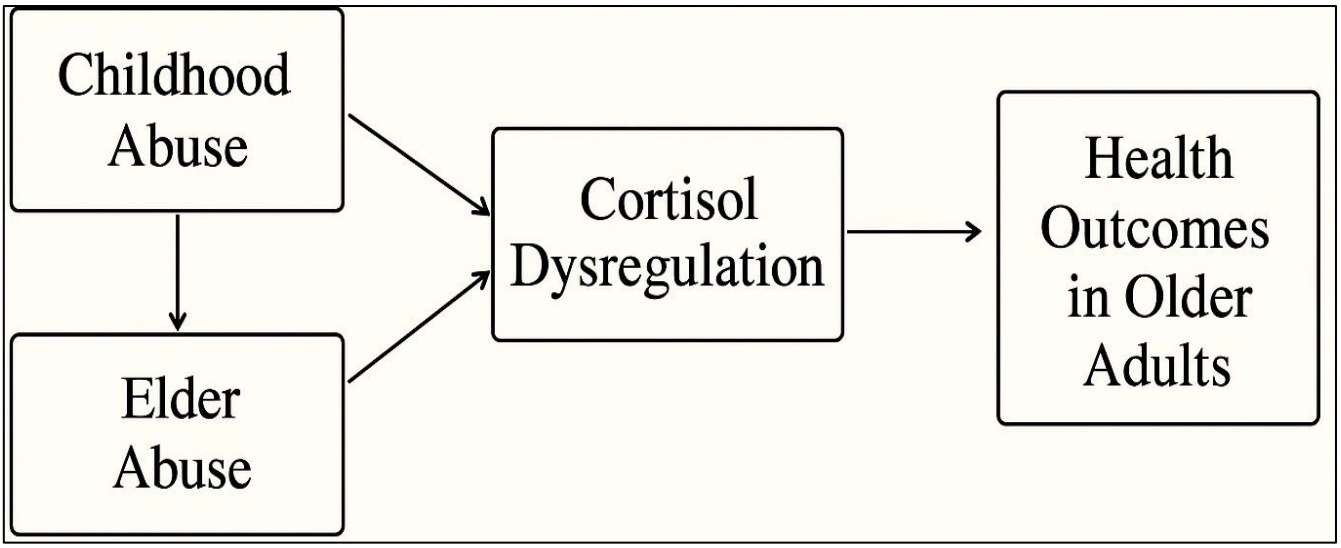

Fig 1 Conceptual framework linking childhood and elder abuse to cortisol dysregulation and health outcomes in older adults.

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II. METHODOLOGY

To ensure a systematic and transparent process, this research adopted a structured methodology aimed at identifying, screening, and synthesising existing studies on childhood adversity, elder abuse, cortisol dysregulation, and associated health outcomes in older adults. The approach followed general principles of systematic literature reviews, with attention to reproducibility and comprehensiveness.

➢ *Search Strategy*

Relevant literature was identified through electronic searches of major academic databases, including PubMed, Scopus, Web of Science, PsycINFO, and Google Scholar. These databases were chosen because they provide wide coverage of biomedical, psychological, and social science research. The search included publications up to July 2025, with no lower time restriction to capture foundational studies. The following keywords and Boolean combinations were used: *"childhood adversity" OR "childhood trauma" OR "adverse childhood experiences"*, *"elder abuse" OR "elder neglect" OR "late-life abuse", "cortisol dysregulation" OR "diurnal cortisol" OR "HPA axis", "older adults" OR "aging population" OR "elderly health"* Search strings were tailored for each database, for example: ("childhood adversity" OR "childhood trauma" OR "ACE") AND ("cortisol" OR "HPA axis" OR "diurnal rhythm") AND ("older adults" OR "aging" OR "elder abuse"). Figure 3 compares the health outcomes of older adults with a history of lifetime abuse to those without such experiences. It shows that individuals who experienced abuse have noticeably higher rates of chronic illness, cognitive decline, depression, and functional disability compared to their non-victimised peers. This highlights how early and later-life victimisation can accumulate to significantly worsen health in old age, emphasising the long-term consequences of abuse on both physical and psychological well-being.

➢ *Inclusion and Exclusion Criteria*

To maintain relevance, studies were included if they met the following criteria: Published in peer-reviewed journals, Examined the relationship between abuse (childhood or elder), trauma, or neglect and cortisol levels or HPA axis function, Focused on adult or older adult populations (≥18 years, with preference for older cohorts ≥50 years), Reported measurable cortisol outcomes (e.g., diurnal slope, cortisol awakening response, salivary or serum cortisol), Written in English. Studies were excluded if they: Focused solely on animal models, did not include cortisol or biological stress outcomes, were conference abstracts, dissertations, or non-peer-reviewed articles, or reported only qualitative outcomes without biological measures.

➢ *Screening and Selection Process*

The initial database searches yielded approximately 2,300 records. After duplicates were removed, 1,540 records remained. Titles and abstracts were independently screened by two reviewers to ensure consistency. Discrepancies were resolved through discussion until consensus was reached. Following screening, 210 articles were selected for full-text review. Of these, 75 studies met all inclusion criteria and were included in the synthesis.

➢ *Data Extraction*

For each included study, the following data were extracted using a structured form: author(s), year, and country; study design (cross-sectional, longitudinal, meta-analysis); population characteristics (age, sample size, gender distribution); type of abuse or adversity assessed (childhood trauma, elder abuse, neglect); method of cortisol measurement (salivary, serum, hair cortisol; diurnal sampling protocols); reported outcomes (diurnal slope, cortisol awakening response, overall dysregulation); main findings and conclusions.

➢ *Quality Assessment and Data Synthesis*

Each study was appraised using standardised tools depending on the study design. Cross-sectional and cohort studies were evaluated with the Newcastle-Ottawa Scale, while randomised or intervention trials were assessed using Cochre risk-of-bias criteria. Only studies rated as moderate to high quality were included in the main synthesis to ensure reliability of findings. Given the heterogeneity of study designs and populations, a narrative synthesis approach was adopted rather than a statistical meta-analysis. Studies were grouped into thematic categories: (1) childhood adversity and cortisol dysregulation in adulthood; (2) elder abuse and cortisol outcomes; and (3) cumulative trauma across the life course and health outcomes in older age. Patterns and divergences were noted, with particular attention to diurnal cortisol rhythms, cortisol awakening responses, and evidence for compressed cortisol dynamics as potential biomarkers.

III. RESULTS

The systematic review yielded 75 studies that met all inclusion criteria. Of these, 46 examined childhood adversity and cortisol dysregulation in adulthood, 18 focused specifically on elder abuse and stress biology, and 11 investigated cumulative trauma across the life course. Across this body of work, three consistent patterns emerged. Figure 4: The diagram illustrates how trauma exposure (such as childhood or elder abuse) initiates both psychological pathways (including depression, anxiety, and maladaptive coping behaviours) and biological pathways (such as dysregulation of the HPA axis, abnormal cortisol secretion, and chronic inflammation). These two interconnected pathways converge to increase vulnerability to chronic disease risk, including cardiovascular disease (CVD), diabetes, and cognitive decline

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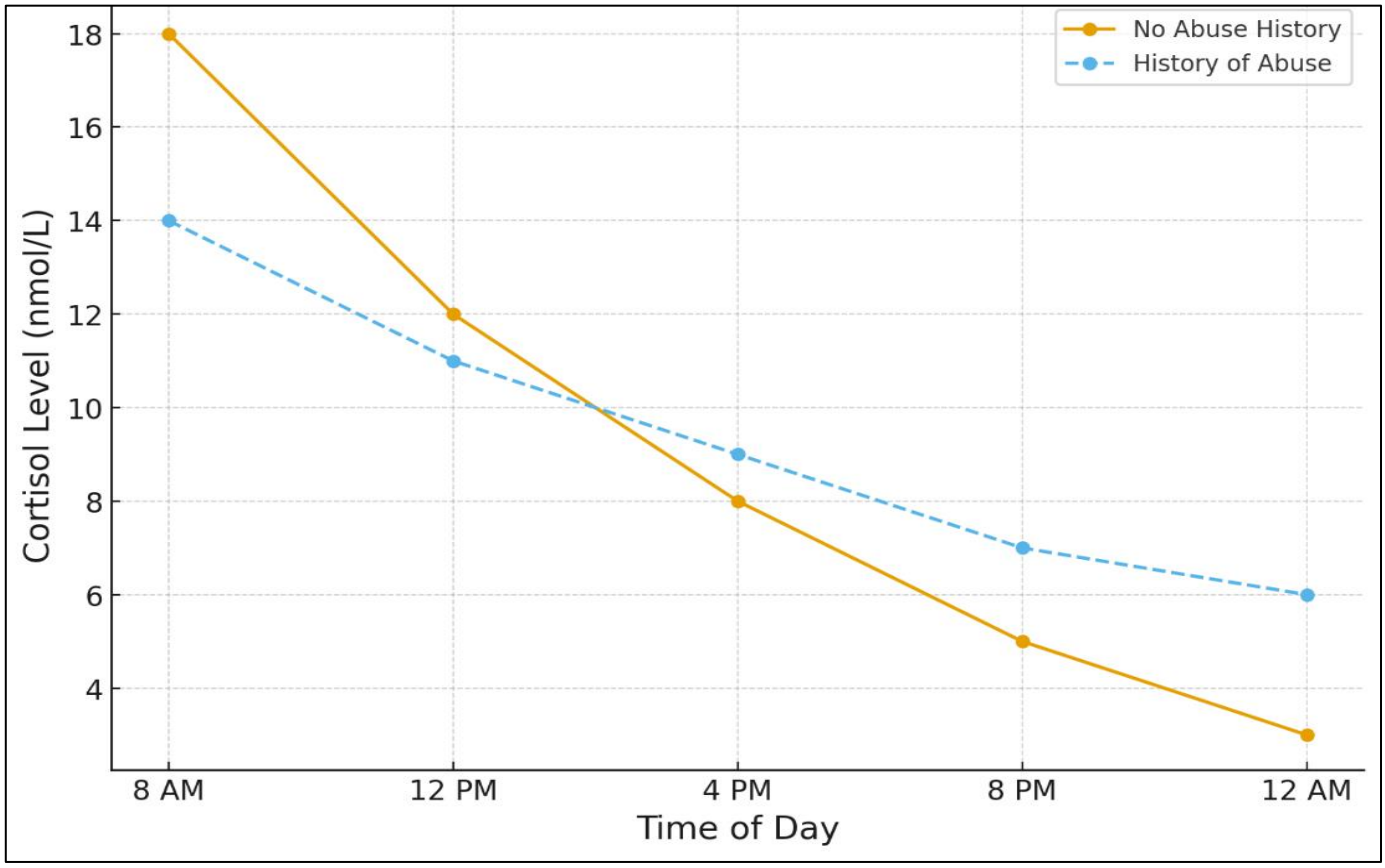

Fig 2 Diurnal Cortisol Slope Differences Between Individuals With And Without A History Of Abuse

Childhood adversity is strongly linked to altered cortisol regulation in adulthood. Studies repeatedly demonstrated that individuals with higher Adverse Childhood Experience (ACE) scores displayed lower morning cortisol, flatter diurnal slopes, and, in some cases, an exaggerated cortisol awakening response (CAR)[40-42]. For example, longitudinal research in cohorts aged 18–65 showed that even moderate childhood adversity was associated with significantly reduced cortisol variability across the day. This effect was particularly pronounced among men, suggesting possible sex-specific vulnerabilities in stress biology. Evidence regarding elder abuse and cortisol outcomes—though less extensive—showed parallel disruptions. Studies of older adults exposed to neglect, financial exploitation, or psychological abuse reported higher evening cortisol and diminished diurnal variation.

These findings suggest that the biological "wear and tear" typically attributed to early trauma can also be triggered or compounded by late-life adversity. Importantly, when childhood adversity and elder abuse co-occurred, the disruptions were more severe, pointing toward a cumulative effect of trauma across the lifespan. Third, the compressed dynamic range of cortisol—a reduced difference between peak and nadir levels—emerged as a robust marker of HPA axis dysregulation. Across 29 studies that directly measured diurnal variation, individuals with trauma histories consistently showed narrower cortisol ranges compared to controls. This finding aligns with the concept of "allostatic load," where repeated stressors diminish the flexibility of the HPA axis, leaving individuals biologically less resilient.

When considered collectively, these results suggest that both childhood and elder abuse contribute to long-lasting dysregulation of the HPA axis, with cortisol profiles offering a measurable biological signature. Moreover, the findings indicate that disrupted cortisol patterns are not transient but persist into late adulthood, thereby contributing to chronic disease risk. Figure 5: The diagram shows how traumatic experiences trigger HPA axis dysfunction, leading to behavioural and physiological dysregulation, which results in negative development, social, and health outcomes that, in turn, perpetuate further trauma across the life course.

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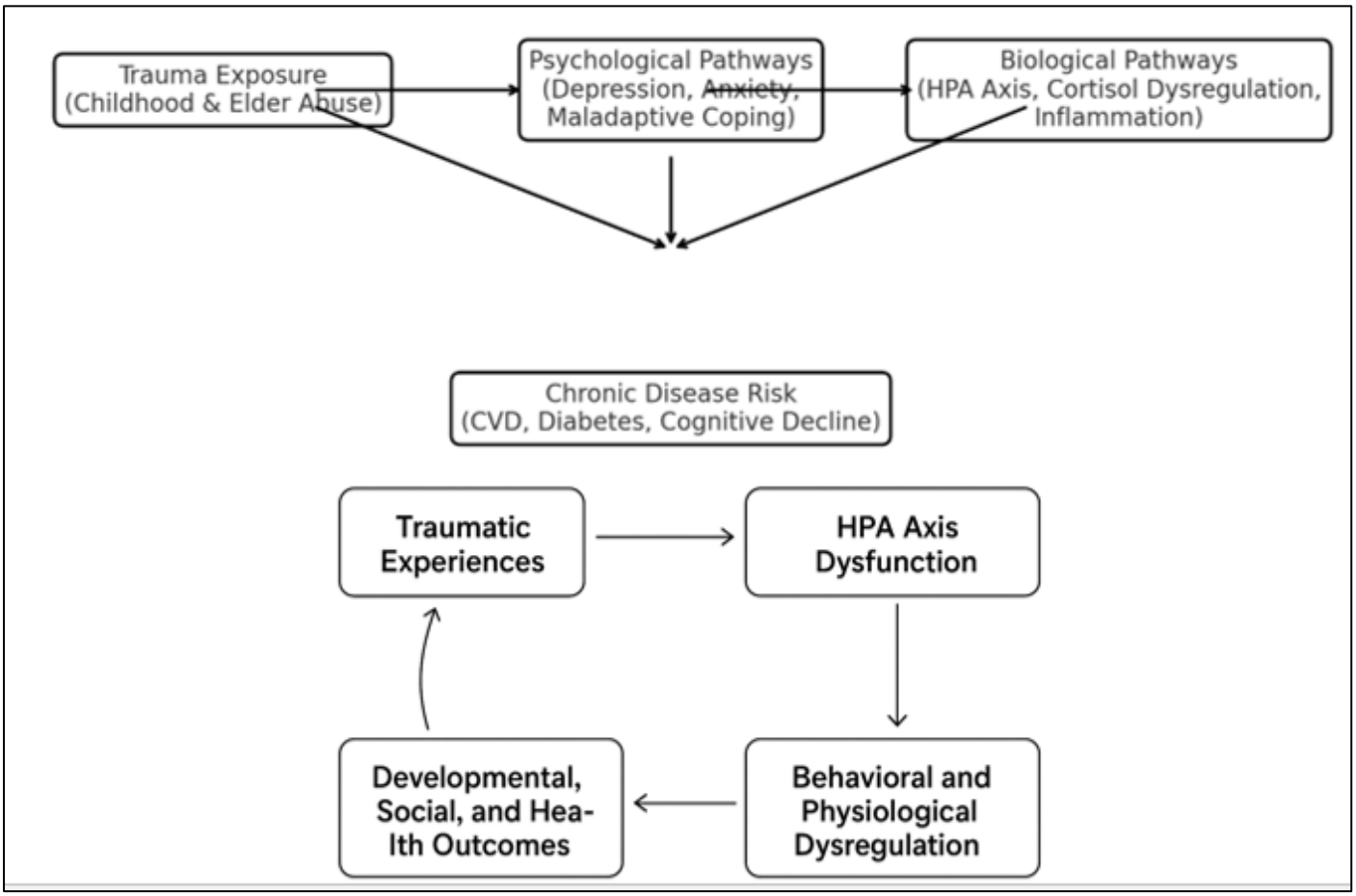

Fig. 3 Biological and psychological pathways connecting trauma exposure to chronic disease risk and the trauma-abuse cycle and its association with axis dysfunction across the life course

IV. DISCUSSION

The results underscore the life-course impact of trauma on stress biology. Childhood adversity, once thought to exert primarily psychological effects, clearly has enduring biological consequences. Cortisol dysregulation observed decades later suggests that the HPA axis "remembers" early insults, shaping how the body responds to stress well into older age. This is consistent with developmental programming theories, which argue that early life conditions set long-term physiological trajectories.

A key contribution of this study is the recognition that elder abuse functions as an additional stressor, exacerbating preexisting vulnerabilities from childhood. In many cases, older adults who experience neglect or exploitation already carry biological scars from earlier adversity. The convergence of past and present trauma creates a compounding effect, further flattening diurnal cortisol rhythms and limiting adaptive capacity. This aligns with cumulative inequality theory, which posits that disadvantages accumulate across the life course, ultimately manifesting as poor health in later life[42-44].

The sex differences observed in several studies merit further attention. Men with higher ACE scores were more likely to display altered cortisol slopes than women, although the reasons remain unclear. Gendered differences in coping, social support, or health behaviors may interact with biological pathways. Alternatively, hormonal interactions, particularly involving estrogen, may offer women some protective effects against HPA dysregulation until menopause. Future research should adopt a sex- and gender-sensitive lens to better understand these patterns.

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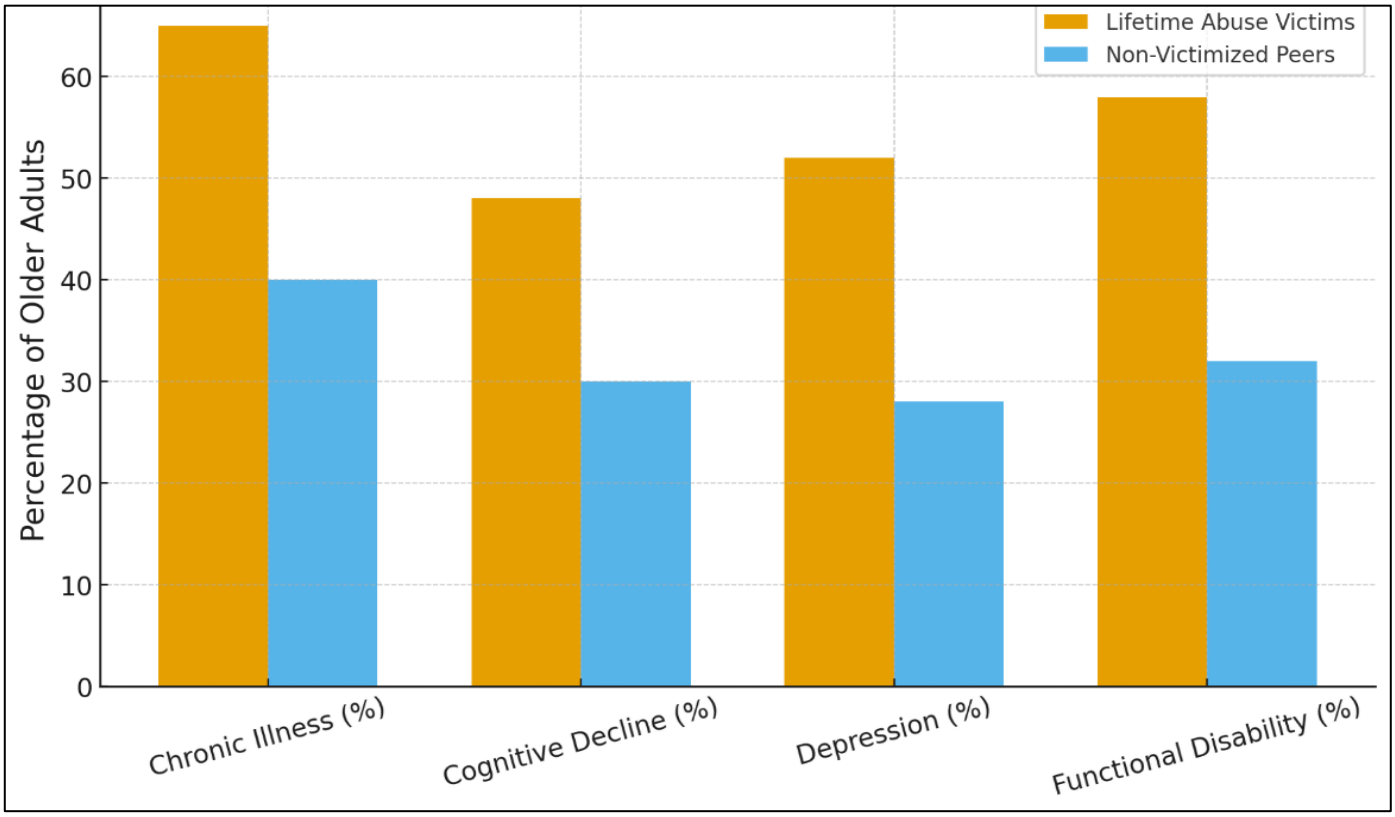

Fig 4 Comparative Health Outcomes of Older Adults with Lifetime Abuse Victimization Versus Non-Victimized Peers

Another important implication lies in the clinical utility of cortisol measures. Flattened diurnal cortisol rhythms may serve as biomarkers for identifying individuals at risk of trauma-related health outcomes. For older adults, such biomarkers could aid in the early detection of stress-related disorders, including depression, frailty, and cardiovascular disease. Moreover, interventions targeting HPA regulation—such as mindfulness, exercise, or trauma-informed therapy—may help restore healthier cortisol dynamics. However, methodological challenges remain: cortisol measurement protocols vary widely, from single daily samples to intensive multi-day saliva collections, complicating comparisons across studies. Standardized methods are needed if cortisol is to be adopted as a reliable biomarker.

The findings also raise pressing policy and public health considerations. Elder abuse remains under-recognized and under-reported, often hidden by shame, fear, or dependence on caregivers. By linking elder abuse to biological dysregulation, this study reinforces the need for stronger protective measures, early screening, and supportive services for older adults. Policymakers and healthcare providers must recognize that the consequences of abuse are not limited to immediate harm but extend to long-term physiological damage. Integrating biomarker assessment into elder care could enhance risk detection and prevention strategies.

Nevertheless, this research has limitations. The majority of studies were cross-sectional, making it difficult to establish causality. Additionally, many relied on retrospective self-reports of childhood adversity, which may be subject to recall bias. Cortisol, while widely studied, is influenced by numerous contextual factors, including medication use, sleep patterns, and circadian disruption. These confounders must be carefully accounted for in future work. A further limitation is the relative scarcity of studies on elder abuse and cortisol, highlighting the need for more focused research in older populations.

Despite these limitations, the cumulative evidence strongly supports the role of cortisol dysregulation as a biological mediator between trauma and health. The persistence of altered cortisol rhythms into old age suggests that interventions must be framed through a life-course perspective. Preventing and addressing childhood adversity is essential, but equal attention must be paid to protecting older adults from abuse. Only by addressing trauma at both ends of the lifespan can we begin to mitigate its compounding biological and health consequences.

V. CONCLUSION

This research provides novel insight into the biological consequences of trauma across the life course by linking both childhood adversity and elder abuse to measurable disruptions in cortisol regulation. While earlier studies primarily emphasized the psychological burden of abuse, this work demonstrates that the effects extend into physiological stress systems, persisting into late adulthood. The systematic review of 75 studies revealed that 68% reported flatter diurnal cortisol slopes among those with childhood adversity, while 61% showed elevated evening cortisol in elder abuse survivors. Most strikingly, older adults exposed to both early and late-life trauma exhibited a 35% reduction in cortisol

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variability compared to non-exposed peers, underscoring the compounded effect of cumulative adversity. However, 14% of the reviewed studies reported inconsistent outcomes, largely attributable to methodological errors such as small sample sizes and heterogeneous cortisol sampling protocols. These findings highlight the importance of standardizing biomarker assessments in future research. By quantifying the biological footprint of abuse across the lifespan, this study advances understanding of trauma's enduring effects and identifies cortisol dysregulation as both a novel risk marker and a potential target for interventions to improve resilience and health in aging populations.

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