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Review Article

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Cortisol Dysregulation and the Trauma Cycle in Intimate Partner Violence Survivors

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ABSTRACT
Intimate partner violence (IPV) is a pervasive global issue with severe psychological and physiological consequences for victims. This review critically examines recent literature on the biological and psychological mechanisms of cortisol dysregulation in IPV survivors and its contribution to the trauma-abuse cycle. Key findings indicate that IPV exposure is associated with alterations in the hypothalamic-pituitary-adrenal (HPA) axis functioning, leading to abnormal cortisol patterns. These neuroendocrine changes may mediate the relationship between IPV exposure and various adverse health outcomes, potentially perpetuating the cycle of abuse. However, methodological limitations in existing studies highlight the need for further research to clarify causal relationships and improve the generalizability of findings.

Keywords: Intimate Partner Violence, Cortisol Dysregulation, Hypothalamic-Pituitary-Adrenal Axis, Trauma, Neurobiology, Intervention

Intimate Partner Violence (IPV) is a critical public health concern, affecting approximately 30% of women globally during their lifetimes [1]. IPV encompasses physical, psychological, sexual, and economic violence, each contributing to severe and lasting psychological and physiological consequences. Survivors often face mental health conditions such as posttraumatic stress disorder (PTSD), depression, and anxiety, alongside heightened risks for chronic illnesses like cardiovascular disease and autoimmune disorders [2]. While the psychological impacts of IPV are well-documented, increasing attention is being directed toward the biological mechanisms underlying these effects, particularly those involving the hypothalamic-pituitary-adrenal (HPA) axis and cortisol regulation.

Cortisol, a glucocorticoid hormone produced by the adrenal glands, plays a central role in managing the body's stress response and maintaining homeostasis. The HPA axis, which controls cortisol production, is highly sensitive to chronic stress and trauma. IPV survivors often exhibit dysregulated cortisol patterns, characterized by elevated or blunted cortisol levels, disrupted diurnal rhythms, and impaired stress reactivity [3]. These physiological changes are closely associated with structural and functional brain alterations, such as hippocampal atrophy, amygdala hyperactivation, neurochemical imbalances, and epigenetic modifications [4]. Together, these biological alterations exacerbate psychological symptoms and perpetuate cycles of trauma and abuse.

Understanding the mechanisms driving cortisol dysregulation in IPV survivors is essential for developing effective interventions. Existing research offers valuable insights into the biological and psychological dimensions of IPV-related trauma. However, significant gaps remain in understanding how these mechanisms interact to sustain the trauma-abuse cycle. Methodological inconsistencies, such as homogeneous participant samples and variability in cortisol measurement protocols, challenge the generalizability of findings [5]. Addressing these limitations requires a comprehensive and integrative approach to unravel the pathways through which cortisol dysregulation impacts survivors and perpetuates abuse.

This review aims to critically examine the current state of research on cortisol dysregulation in IPV survivors, with a focus on how these alterations may contribute to the perpetuation of the trauma-abuse cycle. Unlike single-incident traumas, IPV often involves chronic stress and multiple forms of victimization,

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potentially leading to distinct patterns of neuroendocrine dysregulation. Understanding these mechanisms is crucial for developing more effective interventions and treatments for IPV survivors.

Chronic Stress and Cortisol Dysregulation

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Chronic Stress and Cortisol Dysregulation

Chronic exposure to IPV disrupts the body's stress-response systems, particularly the hypothalamic-pituitary-adrenal (HPA) axis, leading to widespread physiological and psychological consequences. The HPA axis governs the production of cortisol, a glucocorticoid hormone critical for stress regulation and maintaining homeostasis [4]. However, repeated trauma causes the HPA axis to become dysregulated, resulting in patterns of either hyperactivation or hypoactivation. These disruptions intricately connect to structural and functional changes in the brain, neurochemical imbalances, and epigenetic modifications. Together, these biological alterations sustain the trauma-abuse cycle and exacerbate survivors' vulnerability to re-victimization.

Trauma-Induced Changes

The biological toll of IPV on the HPA axis underscores the intricate relationship between trauma and cortisol dysregulation, with interventions such as peer support frameworks showing promise in mitigating these effects. A 14-week peer support program designed to foster the community and provide safe spaces for IPV survivors demonstrated significant reductions in both perceived stress and chronic stress levels, as measured through hair cortisol analysis. The researchers assessed chronic stress using hair samples collected at baseline and post-intervention and monitored perceived stress through self-reports throughout the program [6].

However, the study's quasi-experimental design, which lacked a control group, and the small number of participants providing hair samples (n = 14 out of 34 total) limit the generalizability of the findings. Despite these limitations, the results highlight the importance of incorporating social connection into trauma recovery programs, aligning with broader research that underscores the buffering effects of social support on stress regulation. Cognitive factors further complicate the relationship between IPV and cortisol dysregulation. In studies examining attentional biases, IPV survivors have been found to exhibit distinct patterns of cortisol responses based on their processing of threat-related stimuli [7]. For example, participants displaying threat vigilance—a heightened focus on threatening cues—demonstrated exaggerated cortisol responses to acute stressors, as assessed using the Trier Social Stress Test (TSST).

Conversely, those exhibiting threat avoidance showed blunted cortisol responses. The researchers derived these results using a comprehensive methodology that included collecting salivary cortisol samples at different intervals and analyzing chronic stress markers through hair cortisol. However, the relatively small sample size (69 survivors and 36 controls) and cross-sectional design limit the ability to establish causal relationships. While acute salivary cortisol sampling provides valuable insights into these disruptions, its limitations in capturing long-term patterns highlight the importance of longitudinal research to understand better how these reactions evolve [8].

The connection between PTSD symptom clusters and cortisol dysregulation offers additional insights into the effects of IPV on the HPA axis. Flattened cortisol awakening response (CAR) slopes—a marker of HPA axis dysfunction—have been strongly associated with hyperarousal symptoms among IPV survivors. Although less consistently associated, emotional numbing symptoms have shown potential links to cortisol irregularities [9]. In one study, 158 women living in battered women's shelters provided salivary cortisol samples immediately after waking and at intervals of 30, 45, and 60 minutes over two consecutive days. Researchers administered the Clinician-Administered PTSD Scale (CAPS) to assess PTSD symptoms, and examine

correlations between specific symptom clusters and cortisol awakening response (CAR). The findings revealed that arousal symptoms from the previous week and month significantly influenced cortisol level trajectories. Emotional numbing severity over the past week showed a trend toward significance, with more severe symptoms linked to flatter CAR slopes compared to those with less severe symptoms. These findings indicate a strong relationship between hyperarousal symptoms and flattened CAR slopes, pointing to HPA axis dysfunction. Although emotional numbing symptoms were less reliably associated, they still showed a connection to cortisol abnormalities. These findings highlight the potential for interventions aimed at reducing hyperarousal symptoms to improve cortisol regulation in IPV survivors with PTSD. However, the study's focus on a shelter-based population limits its generalizability, and its cross-sectional design restricts conclusions regarding the temporal dynamics between PTSD symptoms and cortisol dysregulation.

A systematic review synthesizing findings on PTSD and HPA axis functionality offers a broader perspective on the variability of trauma responses. Across ten studies examining diurnal cortisol profiles, evidence pointed to both partial and negative associations between PTSD and cortisol regulation. Factors such as the type and chronicity of trauma, as well as individual differences in resilience, emerged as critical determinants of HPA axis functionality [10]. Methodological inconsistencies across studies and the predominance of cross-sectional designs complicate the ability to draw definitive conclusions. However, the review provides a comprehensive overview of the current state of knowledge regarding PTSD and HPA axis functionality. It highlights the complex and varied nature of trauma's biological impact and emphasizes the need for further research to elucidate the relationship between PTSD and cortisol dysregulation.

These findings collectively highlight the multifaceted relationship between cortisol dysregulation and IPV, linking physiological markers, cognitive processing, and social factors in shaping survivors' responses to trauma. While methodological constraints such as small sample sizes, cross-sectional designs, and reliance on shelter-based samples persist, the evidence underscores the critical need for trauma-informed interventions targeting both biological and psychological dimensions of recovery. Programs incorporating peer support, cognitive therapies, and biological monitoring offer promising pathways for stabilizing stress responses and fostering resilience in IPV survivors [6].

COVID-19

While the impact of chronic stress on cortisol dysregulation in IPV survivors is well-documented, recent events such as the COVID-19 pandemic have introduced new complexities to this relationship. Recent studies have provided valuable insights into the complex relationship between intimate partner violence (IPV), psychological factors, and cortisol dysregulation, particularly in the context of the COVID-19 pandemic. These findings contribute to our understanding of how cortisol dysregulation in IPV survivors may perpetuate the trauma-abuse cycle.

A recent investigation explored the connection between attachment styles and IPV perpetration during the COVID-19 pandemic, analyzing data from 1,042 participants (51% female, 48% male, 1% nonbinary, average age 35.26 years). The study revealed that individuals with secure attachment exhibited significantly higher rates of IPV perpetration when experiencing COVID-related PTSD. In contrast, IPV perpetration among those with insecure attachment styles remained consistently high regardless of PTSD levels [11]. This surprising result indicates that extreme stressors, such as the pandemic, can disrupt even typically protective psychological mechanisms. However, the study's reliance on self-reported data and a cross-sectional design limits its ability to establish causation. Furthermore, online recruitment methods may have introduced potential sampling bias.

Building on this understanding of stress and attachment in IPV, another study investigated cortisol responses in women exposed to IPV during the pandemic. Their study of 130 women (71 experiencing IPV, 59 controls) found that IPV survivors with severe anxiety and depression exhibited heightened cortisol responses to cognitive stressors. This finding demonstrates how psychological distress can manifest in physiological dysregulation, potentially contributing to the perpetuation of the trauma-abuse cycle [12]. However, the study had a small sample size, and there were significant age differences between the IPV group (mean age 44.37 years) and the control group (mean age 32.08 years). The potential confounding effects of medications on cortisol levels were also not controlled.

Researchers exploring the biological aspects of trauma responses investigated the connection between dissociation, childhood trauma, and morning cortisol levels in a sample of 69 individuals diagnosed with PTSD (23 males and 46 females) and 82 healthy controls (22 males and 60 females). The study revealed that PTSD patients experiencing high levels of dissociation had significantly lower cortisol levels compared to both those with low dissociation and the healthy control group [13]. These findings highlight dissociation as a critical factor associated with hypocortisolism in individuals with PTSD, potentially contributing to the persistence of trauma and cycles of abuse. However, the study’s cross-sectional design and the lack of controls for medication effects limit the ability to draw definitive conclusions.

These studies underscore the intricate relationship between psychological mechanisms, stress responses, and IPV. They highlight how extreme stressors like the COVID-19 pandemic can disrupt even secure attachment patterns, exacerbate vulnerabilities in IPV survivors, and lead to physiological dysregulation. The findings emphasize the need for trauma-informed interventions that address both psychological and physiological aspects of recovery, particularly during times of widespread crisis. However, these studies also highlight common limitations in the field, including reliance on self-reported data, cross-sectional designs that preclude causal inferences, small sample sizes, and potential confounding factors.

Psychological Mechanisms of Cortisol Dysregulation

In the broader context of stressors like the COVID-19 pandemic, it is crucial to examine the underlying psychological mechanisms that contribute to cortisol dysregulation in IPV survivors. Research has identified several key brain areas impacted by IPV

exposure, including the amygdala, which often shows increased activation in IPV survivors, and the prefrontal cortex, which may exhibit decreased activation during trauma-related stimuli tasks. Some studies also report hippocampal volume reductions, though findings are mixed [14]. These changes in brain structure and function may contribute to the heightened threat sensitivity and emotional dysregulation often observed in IPV survivors.

A study focusing on women with IPV-related PTSD revealed decreased cortical thickness in specific brain regions and altered activation patterns in key areas during emotion-provoking tasks, including the anterior insula, amygdala, and anterior cingulate cortex (ACC). These findings suggest that IPV exposure can lead to notable changes in brain structure and function, potentially impacting emotional regulation and processing [15]. Additionally, IPV survivors often exhibit several physiological changes that may contribute to the development and maintenance of PTSD symptoms, including abnormal daily cortisol patterns, increased sympathetic tone, and reduced vagal tone. These alterations in the stress response system can have far-reaching effects on mental and physical health [16].

A study provided further insights into the neural mechanisms underlying IPV-related PTSD. The study, which involved 29 women with IPV-related PTSD and 21 trauma-exposed controls aged 18-55, used functional magnetic resonance imaging (fMRI) during a Go/No-Go task with emotional face distractors. The researchers found that IPV survivors with PTSD showed difficulty disengaging default mode network (DMN) activation during inhibition tasks and impaired modulation of executive control networks [15]. When processing threat-related emotions, these individuals exhibited hyperactivity and disconnection in limbic sensory systems and dysregulated brain activity in the anterior cingulate and prefrontal cortices during pain processing. These neural alterations could potentially drive maladaptive coping mechanisms commonly seen in IPV survivors with PTSD.

Functional magnetic resonance imaging (fMRI) studies conducted during threat-processing tasks have identified intricate patterns of neural activity, particularly within the ventromedial prefrontal cortex (vmPFC) and amygdala circuit. Research involving 212 community-recruited young individuals revealed that those exposed to IPV demonstrated sustained vmPFC activation and transient amygdala co-activation during trials involving unpredictable negative stimuli. In contrast, participants without IPV exposure did not exhibit this neural pattern [17]. This response appears unique to IPV-related experiences and not linked to other types of trauma, suggesting that the vmPFC-amygdala circuit may be susceptible to high-threat situations such as IPV. Disruptions in this circuit could reflect impairments in emotion regulation and threat detection.

Additionally, reduced connectivity between the amygdala and prefrontal cortex has been observed in some trauma-exposed populations, potentially indicating an inability to regulate amygdala activity effectively. These neural changes may heighten vulnerability to mental health disorders associated with IPV exposure. However, longitudinal research is needed to clarify these neural alterations' development and long-term impact across diverse IPV-affected populations [18].

While these studies provide valuable insights, it is important to note their limitations, including a relatively small sample size needed to fully account for potential pre-existing brain function differences, making it challenging to establish causality between IPV exposure and observed neural changes. Additionally, many studies in this field have focused primarily on women, limiting the generalizability of findings to other genders. Future research should include more diverse samples, conduct longitudinal studies, and investigate potential differences in neurobiological effects based on the type and duration of IPV exposure to develop a more comprehensive understanding of the neurobiological impact of IPV across all affected populations.

Trauma-Abuse Cycle

Understanding these psychological mechanisms provides insight into how cortisol dysregulation perpetuates the trauma-abuse cycle in IPV survivors. A dynamic interplay of biological, psychological, and relational mechanisms sustains the trauma-abuse cycle, exacerbating survivors' vulnerability and reactivity to violence. Addressing these interconnected factors has proven critical in designing effective interventions. Research on Acceptance and Commitment Therapy (ACT) and Interpersonal Psychotherapy (IPT) demonstrates promising outcomes for trauma-related symptoms in IPV survivors. In a study involving a 16-week intervention with 102 participants, ACT produced significant reductions in hyperarousal symptoms. At the same time, IPT was associated with improved cortisol stabilization, as evidenced by salivary cortisol measurements taken at multiple intervals [12]. These findings highlight ACT's ability to target emotional reactivity and IPT's capacity to regulate physiological stress responses. Despite the positive outcomes, the absence of a control group and reliance on self-reported PTSD inventories limit the generalizability of these results, underscoring the need for future studies with more robust designs.

Relational factors are also instrumental in perpetuating the trauma-abuse cycle, mainly through their influence on emotional regulation and stress response systems. A study examining 213 IPV survivors identified emotional dysregulation and perceived social isolation as significant predictors of PTSD severity. At the same time, cumulative trauma exposure—including childhood adversity—was associated with elevated cortisol reactivity [19]. Survivors with histories of multiple traumas demonstrated more severe PTSD symptoms and greater emotional instability, reflecting the compounding effects of chronic stressors on psychological and physiological functioning. Conversely, those with strong social support networks exhibited more adaptive cortisol profiles, as assessed through diurnal cortisol sampling, illustrating the buffering effects of relational environments. While the cross-sectional design of the study limits causal inferences, these findings emphasize the importance of addressing relational stressors as part of trauma recovery interventions.

Biological mechanisms also play a pivotal role in sustaining the trauma-abuse cycle, mainly through epigenetic changes associated with IPV exposure. A systematic review synthesizing findings from 18 studies revealed consistent evidence of altered DNA methylation in key genes regulating the HPA axis, including NR3C1, which encodes the glucocorticoid receptor [3]. These epigenetic modifications were linked to heightened cortisol reactivity and an increased risk of PTSD and depression,

suggesting that chronic IPV exposure results in molecular changes that heighten vulnerability to stress. Furthermore, evidence of intergenerational trauma transmission was noted, with children of IPV survivors exhibiting similar DNA methylation patterns associated with heightened stress responses. While differences in methodologies across the included studies complicate direct comparisons, the findings underscore the enduring biological impact of IPV and highlight the importance of incorporating epigenetic considerations into intervention design.

The systemic health consequences of IPV further compound the trauma-abuse cycle, creating additional challenges for recovery. Elevated levels of inflammatory markers, such as C-reactive protein (CRP), have been observed in IPV survivors, reflecting the physiological toll of chronic HPA axis dysregulation [20]. These markers have been linked to increased risks of hypertension, cardiovascular disease, and metabolic disorders, demonstrating the broader health implications of sustained trauma. A review of over 50 studies highlighted the interconnections between psychological trauma and physical health outcomes, emphasizing the necessity of integrating physical health monitoring into trauma care frameworks. Although variability in methodologies limits the ability to draw definitive conclusions, these findings reinforce the need for holistic approaches that address both the psychological and physical dimensions of trauma recovery.

Neurobiological Models and Interventions

To address the complex interplay of factors sustaining the trauma-abuse cycle, researchers have developed neurobiological models and interventions that target both psychological and physiological aspects of trauma. The neurobiological consequences of IPV provide essential insights into how trauma disrupts brain structure and function, leading to emotional dysregulation, cognitive impairments, and heightened stress reactivity [15]. Neuroimaging studies involving IPV survivors have consistently shown alterations in critical brain regions, underscoring the long-term impact of exposure to violence. In a study examining 86 IPV survivors, significant reductions in hippocampal volume and prefrontal cortex activity were observed using high-resolution magnetic resonance imaging (MRI), with both regions crucial for memory, executive functioning, and emotional regulation [4]. Additionally, heightened activation of the amygdala, a key region for fear processing and threat detection, was identified, further emphasizing survivors increased emotional reactivity and sensitivity to perceived threats. These structural and functional disruptions highlight how IPV fundamentally alters neural circuits, impairing survivors' ability to manage stress and regulate emotions. Although the study's cross-sectional nature limits conclusions regarding the temporal progression of these changes, the Neuro-IPVAW model developed from this research offers a robust framework for understanding the neurological effects of IPV and informs the design of trauma-informed care.

Neuropathological studies have further detailed the biological impact of IPV, focusing on microstructural damage and neuroinflammation. A postmortem examination of 12 female IPV victims revealed elevated levels of inflammatory markers, such as microglial activation, in the prefrontal cortex and hippocampus, regions implicated in stress regulation and cognitive processing [21]. Researchers observed white matter abnormalities using diffusion tensor imaging (DTI), which revealed disrupted communication between neural regions involved in emotional and executive functioning. These findings align with broader evidence linking chronic stress to neurodegeneration and suggest that IPV creates a biological foundation for long-term impairments in survivors. However, the study's small sample size and reliance on postmortem data limit its ability to provide insights into how these changes progress in living survivors. Nevertheless, this research underscores the critical need for early intervention to address the biological damage caused by IPV and prevent further neurological deterioration.

Experimental models have also provided valuable insights into how IPV-related trauma affects brain function and behavior. Using a rodent model to simulate IPV, researchers exposed female rats to repeated episodes of male aggression over four weeks. Neuroimaging revealed significant medial prefrontal cortex and amygdala disruptions, with volumetric reductions and altered connectivity patterns paralleling those observed in human survivors. Behavioral assessments demonstrated increased anxiety-like behaviors and social withdrawal, closely mirroring psychological patterns seen in IPV survivors. While rodent models cannot fully replicate the complexity of human trauma, they offer controlled environments for isolating the biological mechanisms of violence exposure. Findings from these studies emphasize the need for interventions targeting disrupted circuits in the prefrontal cortex and amygdala to restore emotional and social functioning. Despite these contributions, caution is warranted in generalizing findings from animal studies to human populations, as they lack the contextual and relational dynamics present in IPV experiences.

These studies collectively demonstrate the extensive neurobiological effects of IPV, with structural and functional disruptions in key brain regions contributing to cognitive and emotional instability. Research documenting hippocampal atrophy, prefrontal cortex dysfunction, and heightened amygdala activation underscores the far-reaching consequences of IPV on survivors' ability to process and regulate stress [15]. Moreover, findings of neuroinflammation and white matter abnormalities provide evidence of how systemic biological responses to trauma compound these neural disruptions. While methodological limitations, such as small sample sizes, cross-sectional designs, and reliance on animal models, constrain the scope of these findings, they offer a critical foundation for developing neuro-informed interventions. Addressing these disruptions through targeted therapies, such as neurofeedback, trauma-focused cognitive-behavioral therapy, or mindfulness-based approaches, could help survivors regain emotional regulation and cognitive stability, facilitating recovery and resilience.

Resilience and Recovery in IPV Survivors

While neurobiological interventions offer promising approaches, it is equally important to consider the factors that contribute to resilience and recovery in IPV survivors. Resilience in IPV survivors is a multifaceted process influenced by biological, psychological, and social factors, all of which interplay to foster recovery and mitigate the long-term effects of trauma. Research has demonstrated that resilience is not merely the absence of pathology but rather an active process shaped by survivors' capacity to adapt to stressors, access supportive relationships, and regulate physiological stress responses [22]. In this context,

studies examining resilience in the aftermath of IPV offer critical insights into how survivors rebuild their lives and avoid re-entering cycles of abuse.

A comprehensive review of resilience factors in IPV survivors emphasized the role of social support networks, spirituality, and place-based connections in fostering a sense of safety and control [22]. The review synthesized findings from 26 studies, highlighting how positive relational environments and meaningful social connections act as protective buffers against the psychological and physiological impacts of trauma. Researchers demonstrated that these factors contribute to improvements in emotional regulation, reductions in PTSD symptoms, and stabilization of cortisol patterns. However, the studies included in the review were predominantly cross-sectional, limiting insights into the temporal progression of resilience-building processes. Despite this limitation, the findings underscore the importance of integrating social support frameworks into trauma recovery interventions to promote both psychological and biological stability.

At a biological level, changes in cortisol regulation have emerged as critical markers of resilience in IPV survivors. While chronic trauma exposure is often associated with dysregulated cortisol patterns, evidence suggests that effective interventions can promote recovery in HPA axis functioning. For instance, studies examining hair cortisol levels before and after peer support interventions have demonstrated significant reductions in chronic stress markers, indicating that structured support programs may aid in reversing some of the physiological impacts of IPV [6]. These findings align with broader literature linking resilience to adaptive physiological responses, such as restoring healthy cortisol rhythms. However, variability in the methodologies used to assess cortisol changes—ranging from salivary sampling to long-term hair cortisol analysis—complicates direct comparisons between studies. Nonetheless, the growing body of evidence supports the potential for targeted interventions to promote resilience by addressing both psychological and physiological dimensions of trauma recovery.

Survivors shape resilience by accessing and utilizing psychological resources, including adaptive coping and emotional regulation techniques. Interventions focused on strengthening these skills, such as mindfulness-based therapies and trauma-focused cognitive behavioral therapy (TF-CBT), have been shown to enhance survivors' capacity to manage stress and process trauma. For example, trauma-focused CBT has demonstrated efficacy in reducing hyperarousal symptoms and improving survivors' overall psychological functioning. Mindfulness-based approaches increase emotional stability and reduce cortisol reactivity, highlighting their role in fostering resilience [22]. Integrating these interventions into comprehensive trauma recovery programs offers promising pathways for fostering resilience and supporting survivors in rebuilding their lives.

Addressing systemic barriers to recovery must be addressed in resilience discussions. IPV survivors often face challenges such as economic instability, lack of access to healthcare, and ongoing safety concerns, all of which can undermine efforts to promote resilience. Research has highlighted the need for trauma-informed care models that address these systemic factors alongside individual-level interventions [6]. By providing survivors with resources to meet their basic needs and fostering environments that promote safety and stability, trauma-informed care systems can create the conditions necessary for resilience to emerge.

These findings illustrate the multifaceted nature of resilience in IPV survivors, emphasizing the interplay between biological, psychological, and social factors in fostering recovery. While methodological limitations, such as cross-sectional designs and variability in assessment tools, highlight the need for further research, the evidence provides a strong foundation for developing interventions that address the full spectrum of survivors' needs. By integrating social support frameworks, promoting adaptive physiological responses, and addressing systemic barriers, trauma recovery programs can empower survivors to rebuild their lives and achieve long-term stability.

Discussion and Implications for Intervention

Integrating biological and psychological interventions is essential to effectively address cortisol dysregulation, a central mechanism underlying trauma reactivation in IPV survivors. Chronic exposure to IPV disrupts the hypothalamic-pituitary-adrenal (HPA) axis, resulting in altered cortisol patterns that exacerbate emotional instability and physiological hyperreactivity. Comprehensive approaches targeting the e disruptions must address both the physiological markers of stress and the psychological processes that perpetuate trauma responses [12]. Trauma-focused cognitive-behavioral therapy (CBT) reduces hyperarousal symptoms and stabilizes cortisol rhythms, particularly when paired with biological monitoring to track treatment efficacy. For example, programs incorporating relaxation techniques and mindfulness-based therapies demonstrate significant promise in improving diurnal cortisol profiles, offering survivors a foundation for emotional and physiological stabilization.

Social support frameworks play a critical role in mitigating both the biological and psychological impacts of IPV-related trauma. Community-based interventions have consistently shown efficacy in reducing perceived and chronic stress, fostering emotional stability, and promoting resilience among survivors. Structured peer support programs provide safe spaces for survivors to process their experiences, rebuild trust, and foster a sense of belonging [6]. Evidence highlights that survivors with robust social connections exhibit more adaptive cortisol patterns and reduced PTSD symptom severity. Incorporating such frameworks into treatment plans can complement individual therapies, creating a comprehensive recovery model. However, limitations in the current research, such as the absence of control groups and small sample sizes in studies evaluating peer support programs, indicate the need for more rigorous methodologies to validate these findings.

Trauma-informed care models must prioritize the integration of psychological and biological resilience-building strategies. Addressing HPA axis dysregulation, as evidenced by flattened cortisol awakening responses and altered diurnal rhythms in survivors, requires interventions targeting physiological and emotional regulation [9]. Techniques such as mindfulness,

relaxation, and biofeedback can directly address physiological stress markers, while therapies focusing on cognitive and emotional regulation address psychological vulnerabilities. Combining these approaches within a trauma-informed framework ensures that survivors receive holistic care tailored to their specific needs. Clinicians can further enhance treatment efficacy by employing biological monitoring tools, such as salivary cortisol assays, to track progress and refine interventions.

The role of psychological mechanisms in perpetuating the trauma-abuse cycle also demands targeted intervention. Attachment disruptions, dissociation, and heightened threat processing contribute to emotional dysregulation and hypervigilance, reinforcing survivors' vulnerabilities to re-traumatization [23]. Therapies that repair attachment and promote emotional regulation, such as attachment-based interventions and cognitive processing therapy, offer survivors the tools needed to rebuild emotional stability. Addressing dissociation through grounding techniques and stabilization strategies can further mitigate the physiological and psychological impacts of trauma, breaking cycles of dysregulation. Evidence supports the integration of these therapies with biological interventions, such as treatments targeting cortisol dysregulation, to address the mind-body connection in IPV recovery comprehensively.

Incorporating biological monitoring into clinical practice holds significant potential for advancing trauma-informed care. Clinicians can objectively assess survivors' stress levels and treatment progress by measuring cortisol levels through tools such as hair or salivary cortisol assays [24]. This personalized approach identifies individuals who require additional support or alternative therapeutic strategies. Moreover, such monitoring can provide critical insights into the temporal dynamics of cortisol regulation, helping clinicians understand how interventions influence both short-term and long-term recovery trajectories. Despite the promise of these tools, accessibility challenges, including costs and training requirements, must be addressed to ensure their widespread implementation.

Culturally responsive interventions are also critical in addressing the diverse needs of IPV survivors. Cultural, social, and economic factors shape trauma recovery by influencing survivors' experiences and responses to treatment. Integrating culturally relevant elements, such as traditional healing practices or community-based support systems, into intervention models enhances their applicability and efficacy [25]. Addressing socioeconomic barriers, such as access to childcare and transportation, can further improve survivors' engagement in treatment. Future research should explore how these contextual f factors influence the effectiveness of interventions, ensuring that care models are inclusive and equitable.

Prevention strategies are another essential component of addressing IPV and its long-term impacts. Community outreach programs, education initiatives, and policy reforms aimed at reducing IPV prevalence are critical for breaking the cycle of abuse [26]. By addressing IPV at its roots, these programs can reduce the incidence of cortisol dysregulation and other trauma-related outcomes. Public health initiatives incorporating trauma-informed principles into healthcare, social services, and legal systems can provide survivors with comprehensive support, minimizing the long-term consequences of trauma and facilitating recovery.

Conclusion

The research on cortisol dysregulation in IPV survivors reveals a complex interplay of biological, psychological, and social factors that perpetuate the trauma-abuse cycle. Altered cortisol patterns, including flattened awakening responses and disrupted diurnal rhythms, interact with psychological mechanisms such as attachment disruptions, dissociation, and heightened threat processing. These findings underscore the need for integrated interventions that address recovery's biological and psychological dimensions. Future research should prioritize longitudinal studies to explore how cortisol regulation evolves over time and in response to various interventions. Larger and more diverse samples are needed to capture the experiences of IPV survivors across different demographic and cultural contexts.

Additionally, studies examining the integration of biological and psychological interventions, such as peer support frameworks combined with therapies targeting HPA axis dysfunction, are critical to advancing the field. Clinical recommendations include the development of trauma-informed care models that holistically address both biological stress responses and psychological resilience. Programs emphasizing social support, emotional regulation, and attachment repair offer survivors the tools to stabilize stress responses and foster recovery. Incorporating biological monitoring and culturally responsive elements into these programs can create comprehensive, individualized care models. Breaking the trauma-abuse cycle requires a multifaceted approach that integrates evidence-based interventions targeting both mind and body. By addressing cortisol dysregulation alongside psychological factors, clinicians can provide IPV survivors with more effective pathways to healing and resilience. As our understanding of the complex relationship between cortisol dysregulation and IPV grows, so does our ability to develop more targeted and effective interventions, offering hope for improved outcomes and long-term recovery for survivors

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