ISSN: 3029-0708

Review Article

Journal of Clinical Psychology and Neurology

Cortisol Dysregulation in Chronic Insomnia: A Critical Review

Xaviera Gadpaille[1*] and Tiffany Field[2]

[1]*Department of Psychology, Fielding Graduate University, USA*

[2]*University of Miami/Miller School of Medicine and Fielding Graduate University, USA*

[*]**Corresponding author**

Xaviera Gadpaille, Department of Psychology, Fielding Graduate University, USA.

Received: September 29, 2026; **Accepted:** October 07, 2026; **Published:** October 12, 2026

ABSTRACT

Insomnia-related hyperarousal has been associated with hypothalamic-pituitary-adrenal axis dysregulation, with cortisol identified as a potential neuroendocrine mechanism underlying persistent physiological arousal. Recent studies link dysregulated cortisol patterns to chronic insomnia severity, pre-sleep arousal, and sleep reactivity, but differences in demographics, research design, and cortisol measurement limit the direction and consistency of this relationship. Cortisol dysregulation is more precisely defined as HPA-axis dysregulation than as elevated cortisol, with potential abnormalities developing in basal concentrations, stress reactivity, or post-stress recovery. It remains unknown from past studies whether cortisol dysregulation supports the disorder, emerges as a reaction to chronic sleep disturbance, or increases susceptibility to insomnia. This review article critically assesses current empirical data and the methodological challenges associated with evaluating the extent to which cortisol dysregulation perpetuates hyperarousal in chronic insomnia.

Keywords: Cortisol, Insomnia, Hyperarousal, Sleep Reactivity, HPA Axis

Insomnia as a Form of Hyperarousal

Insomnia disorder is often conceptualized as a chronic state of hyperarousal, yet the status of cortisol as a biological mechanism under that model remains unclear. Hyperarousal goes beyond restless nights and involves autonomic, cognitive, cortical, emotional, and neuroendocrine activation that may persist during the sleep-wake cycle [1-3].

The hypothalamic-pituitary-adrenal (HPA) axis is a primary stress response system. Activation results in the release of cortisol, which promotes adaptation to psychological and physical stressors. Cortisol is highly relevant to insomnia because stress is a frequent cause of sleep disruption. Furthermore, sleep reactivity is a vulnerability framework that explains why stress causes acute sleep disturbance in some individuals and triggers chronic insomnia in others [4]. Those with greater sleep reactivity experience elevated sleep disruption in response to stress and may exhibit heightened reactivity to environmental issues. This framework is especially beneficial for research examining physiological reactions to acute stress, as opposed to basal cortisol alone [5, 6].

Accumulated evidence supports an association between altered HPA-axis activity and chronic insomnia. However, the magnitude and form of cortisol differences are not consistently operationalized across insomnia studies and may be assessed as elevated basal concentrations, morning concentrations, altered circadian patterns, exaggerated stress reactivity, or recovery following stress [5-8]. Although these measures encompass interconnected yet distinct aspects of HPA-axis functioning, they should not be interpreted interchangeably. Meta-analytic evidence reports higher cortisol levels among individuals with insomnia, most notably in studies using blood samples, and acknowledges significant methodological heterogeneity in collection timing, sampling methods, study designs, and objective measures of sleep loss [9]. Such heterogeneity compromises the interpretation of cortisol as a uniform biological mechanism underlying insomnia-related hyperarousal. A significantly greater cortisol level across studies does not establish that elevated cortisol causes insomnia, nor does it establish that all individuals with insomnia experience cortisol elevations. Consequently, HPA-axis activity differs between uninterrupted-sleep and insomnia groups, but these differences are influenced by methodological factors and observable traits [9]. Such differences emerge as a central theme across insomnia-related studies.

The current critical review evaluates the methodological strengths of four recent empirical studies to assess the degree to which cortisol dysregulation is supported as an underlying biological mechanism of hyperarousal in chronic insomnia. Considerable attention is placed on cortisol measurement, di-

Citation: Xaviera Gadpaille, Tiffany Field. Cortisol Dysregulation in Chronic Insomnia: A Critical Review. J Clin Psychol Neurol. 2026. 4(4): 1-6.
DOI: doi.org/10.61440/JCPN.2026.v4.93

agnostic accuracy, population characteristics, sampling timing, stress exposure, and temporal design. The key question is whether current data support cortisol dysregulation as a biomarker of insomnia disorder and vulnerability to sleep disturbance or as a causal mechanism of chronic insomnia-related hyperarousal. Whereas one focuses on morning cortisol, another examines serum cortisol and neuroendocrine activity, and others highlight stress-induced salivary cortisol responses and laboratory stress [5-8]. The studies illustrate distinct dimensions of HPA-axis function, though they all use the term cortisol.

Empirical findings linking cortisol to persistent sleep loss are reported by Passos et al. and Yu et al., whereas Feng et al. and Kater et al. provide circumstantial evidence regarding sleep-related vulnerabilities and elevated stress responses rather than established adult chronic insomnia [5-8]. This distinction is crucial because data on abnormal cortisol activity in adolescent, nonclinical, or vulnerable populations cannot independently establish cortisol dysregulation as a biological basis for chronic insomnia. However, collectively, the studies permit examination of HPA-axis functioning across established insomnia and stress-related vulnerability.

Morning Cortisol and Chronic Insomnia

Chronic insomnia severity was examined in relation to objective sleep, morning cortisol, and psychological health in a sample of Brazilian adults [7]. The study used serum cortisol, the Beck Depression Inventory, the Insomnia Severity Index, the Profile of Mood States, and polysomnography. The sample consisted of 34 participants for the cortisol and principal clinical measures, with polysomnographic data from 32 participants. Insomnia severity was positively correlated with morning cortisol ($r = .37$, $p = .03$). Sleep-stage measures were related to fatigue and tension-anxiety, whereas insomnia severity was associated with depression and tension-anxiety. Furthermore, research shows cortisol is relevant to hyperarousal because it links the endocrine measure to clinical insomnia severity.

The study has three specific strengths to note. Polysomnography provided objective sleep architecture. Cortisol was directly measured in blood, and participants had clinically diagnosed chronic insomnia. This association gives the authors high convergent validity for a review focused on cortisol and insomnia. However, strong alignment between the review question and the variables should not be mistaken for definitive causation.

Critical Review

The main limitation of this study is causal ambiguity. The analysis is correlational, so higher morning cortisol could result from repeated sleep disruption, contribute to more severe insomnia, or combined outcomes such as medical conditions, medication exposure, depression, and anxiety. The authors recognize small sample size, correlation, and limited exclusion criteria for drugs affecting sleep and clinical disease as limitations. Such limitations directly undermine the claim that association is demonstrated while temporal precedence is not.

The morning cortisol assessment has its own limitations with physiological interpretation. Morning concentration does not determine whether participants have an altered diurnal decline, elevated evening cortisol, exaggerated stress reactivity, or abnormal nocturnal secretion. Since each of those patterns reflects a different aspect of HPA-axis regulation, the study cannot fully characterize cortisol dysregulation. It shows that a morning measure covaries with insomnia severity. In particular, the small sample limits its power to explore moderators. Insomnia is heterogeneous, and objective short sleep age, sex, chronicity, duration, and mood symptoms may indicate biologically unique subgroups. Even with 34 participants, these cannot be tested robustly. They provide clinically relevant evidence, despite the inference being more narrow than the hyperarousal model itself. Higher morning serum cortisol was associated with more severe insomnia in this sample.

Neuroendocrine Activity, Sleep Reactivity, and Pre-Sleep Arousal

Neuroendocrine hyperarousal received its most integrated examination recently in a clinical sample of adults, based in China, with chronic insomnia disorder [8]. The study consisted of 61 patients with chronic insomnia disorder, of whom 27 met healthy controls. Respondents completed the Ford Insomnia Response to Stress Test, the Pre-Sleep Arousal Scale, and the Pittsburgh Sleep Quality Index. Serum concentrations of cortisol, copeptin, and corticotropin-releasing hormone (CRH) were measured using validated psychological measures.

Individuals with chronic insomnia revealed greater pre-sleep arousal and sleep reactivity as well as significantly higher cortisol, copeptin, and CRH levels. Partial correlations revealed positive associations among sleep quality, pre-sleep arousal, and neuroendocrine measures, and sleep reactivity after adjustment for age, sex, cognition, and depression. Principal component analysis positioned the arousal, sleep, neuroendocrine, and reactivity variables on a common factor.

This is a relevant study because it measures both the neuroendocrine and psychological elements of hyperarousal using the same clinical sample. The authors test whether cortisol covaries with pre-sleep and stress-sensitive processes typically emphasized in insomnia models [8]. This conceptual synthesis makes the study more relevant, although the statistical relationships still require cautious interpretation.

Critical Review

There is another strong conceptual alignment in this cross-sectional study [8]. While there is no temporal sequence or mediation, a shared principal component and positive correlations demonstrate covariance. Thus, a common factor cannot indicate that cortisol transmits the effect of pre-sleep arousal or sleep reactivity into insomnia. Neuroendocrine function is altered by chronic insomnia, remaining compatible with the data.

Although the authors found elevated cortisol in individuals with chronic insomnia, the cross-sectional design prevents the endocrine difference from being interpreted as a precursor of insomnia. The finding supports neuroendocrine involvement in the disorder but provides significantly weaker evidence for cortisol as an etiological mechanism. The control group is also significantly smaller than the insomnia group, potentially reducing precision in group comparisons. Although analyses adjusted for several covariates and the groups were matched on baseline characteristics, residual confounding remains feasible. Serum measure-

ments also provide lower temporal resolution than repeated sampling around a controlled stressor.

A much larger issue comes from inconsistency within the broader scope of research. Previous work from the same research group reported cortisol, copeptin, and comparable levels of CRH between individuals with chronic insomnia and normal controls at selected morning time and pre-sleep points. That cognitive observation reinforces the main methodological argument that endocrine findings depend on context, operationalization, sample characteristics, and timing. While the authors strengthen the case for neuroendocrine involvement, they do not explain the relationship between elevated cortisol and chronic insomnia.

Cortisol Stress Reactivity in Adolescents

This study examined whether insomnia and sleep disturbances were associated with subjective and physiological responses to acute social stress in 135 adolescents ranging from 11 to 18 years, based in Germany [6]. Individuals completed the Trier Social Stress Test for Children. Subjective stress and salivary cortisol were collected six times, while heart-rate variability and heart rate were measured before, during, and after stress induction. Insomnia and sleep disturbances were measured using interview and self-report methods.

Boys with reported higher cortisol experienced higher sleep disturbances than those with lower sleep disturbances. In boys with insomnia, selected autonomic measures also differed less across the stress task than in boys without insomnia. Findings suggest sex-specific relationships among autonomic adaptability, HPA-axis activity, and poor sleep.

Repeated cortisol sampling is a significant methodological strength. With six samples around a standardized stressor, assessment of endocrine change over time. The experimental stress paradigm also improves temporal control by demonstrating when the stressor occurred relative to the biological response [6]. Regarding stress reactivity, this design is stronger than a single morning cortisol assessment.

Critical Review

A key limitation for this study is that developmental differences in neuroendocrine functioning may limit generalizability to adult insomnia populations. Hormonal maturation, pubertal development, and age-related stress responses may shape cortisol patterns in ways that differ significantly from those observed in adult samples. Furthermore, the study focused primarily on insomnia symptoms rather than clinically diagnosed insomnia disorder [6].

Another limitation relates to differences between the diagnoses of insomnia and sleep disturbances. The most dominant outcome was observed among boys with severe sleep disturbance. A study centered on cortisol as a mechanism of insomnia should not treat a sleep-disturbance effect as equal to one on a cortisol abnormality in diagnosed insomnia.

Importantly, the sex interaction is scientifically significant but compromises generalization. If cortisol dysregulation is conditional on developmental stage, symptom definition, or sex, a hypercortisolemic model of insomnia is likely insufficient. The authors support the overarching focus that sleep problems and stress-system regulation are connected. At the same time, they indicate why moderators should be incorporated into the models.

Cortisol Sleep Reactivity and Acute Stress in Young Adults

Sleep reactivity was evaluated as a potential moderator of cortisol responses to experimentally induced stress in healthy young adults, based in China [5]. Participants were exposed to a standardized stress paradigm and rated as high or low in sleep reactivity. Salivary cortisol was measured four times, including before stress and during the post-stress period, using the Trier Social Stress Test.

Baseline cortisol levels did not differ between the high- and low-sleep-reactivity groups. Participants with high sleep reactivity exhibited stronger cortisol responses to stress, with higher concentrations at later post-stress time points. In the high-reactivity group, cortisol returned toward baseline more slowly. Findings align closely with the hypothesis that vulnerability to stress-induced sleep disturbance may be accompanied by HPA-axis dysregulation before chronic insomnia becomes established.

The study is theoretically relevant because it changes the focus from fixed cortisol levels to adaptive stress responses. That approach is compatible with Kalmbach et al., who conceptualize sleep reactivity as a vulnerability factor [4]. If hyperarousal is semi-partly stress dependent, experimentally induced cortisol might highlight distinctions that resting measures miss.

Critical Review

The key limitation of this study is the absence of patients with chronic insomnia. Instead, healthy young adults were recruited to assess sleep reactivity, so the study addresses vulnerability rather than an already established disorder [5]. This is both a limitation and a strength. While it may expose root causes of sleep problems, it also lacks proof of cortisol levels persisting long after insomnia becomes chronic.

The authors recognize a highly educated and predominantly young sample, small sample size, limited investigation of neurobiological mechanisms, and absence of follow-up as important limitations. The lack of follow-up is highly significant. Without longitudinal observation, it remains unclear whether individuals with stronger cortisol responses and high sleep reactivity subsequently developed insomnia disorder.

The study also illustrates why baseline cortisol alone may lack sufficient standalone utility. Although the high- and low-reactivity groups showed no baseline differences before stress, their post-stress responses appear to increase over time. This goes against the claim that vulnerability to insomnia is consistently marked by high cortisol. More importantly, stress manifests differently in at-risk individuals. The authors aim to provide substantial evidence, but the connection from vulnerability to diagnosed insomnia remains inferential.

What Is Cortisol Dysregulation

Existing studies indicate that cortisol dysregulation is a single, measurable construct. Passos et al. conceptualized neuroendocrine disturbance through the lens of morning serum cortisol,

and Yu et al. examined serum cortisol within a broader scope that included copeptin and CRH [7, 8]. In contrast, others use repeated salivary cortisol responses around experimentally induced stress [5, 6]. Consequently, these studies do not employ various techniques to measure the same physiological response, although they may obtain distinct parts of HPA-axis functioning. Importantly, post-stress recovery, stress reactivity, circadian secretion, basal concentration, and acute stress reactivity have different physiological meanings.

An individual can display normal baseline cortisol with an exaggerated reaction or hypersensitivity [5]. Conversely, an elevated morning cortisol associated with insomnia severity does not yield stress to exaggerated reactivity [7]. High levels of cortisol can be established in a clinical insomnia group, without their design distinguishing whether the difference indicates tonic activation, recent stress exposure, circadian phase, or another component of HPA-axis functioning [8].

This distinction alters how inconsistencies should be viewed. For example, if one study measures the response to an acute challenge and another measures a morning state, disagreement in direction or effect size does not fundamentally mean conflicting biological mechanisms. It may mean they are heterogeneous indicators. Determine timing and sampling method as sources of heterogeneity across HPA-axis studies [9]. A synthesis contradicts the claim that insomnia is related to elevated cortisol, therefore, insomnia and vulnerability may be associated with altered HPA-axis regulation. However, the affected element of that regulation remains insufficient.

Strengths of Empirical Evidence

Although the overall significant limitation is that existing research provides moderate support for cortisol dysregulation associated with chronic insomnia, sleep reactivity seems to be the connecting variable that connects the four empirical studies. Findings are more robust for stress-reactivity models and least favorable for reports that insomnia is marked by elevated baseline cortisol levels [4-6, 8]. The studies should not be given differential consideration because direct relevance and methodological rigor to the review question do not perfectly align. Passos et al. and Yu et al. provide the strongest empirical data [7, 8]. Both examine adults with chronic insomnia and directly measure cortisol. Passos et al. strengthen objective sleep metrics by adding polysomnography, whereas Yu et al. integrate cortisol with sleep reactivity and pre-sleep arousal while utilizing a case-control design [7, 8]. Such features make the studies the most applicable to the argument that cortisol is associated with insomnia-related hyperarousal.

However, studies provide robust temporal order of cortisol around a controlled stressor [5,6]. The multiple salivary samples differentiate baseline from post-stress responsiveness and address progressive HPA-axis reactivity more effectively than an isolated serum measurement. Nevertheless, this methodological benefit comes at the cost of diagnostic specificity. Kater et al. studied adolescents and cortisol effects for sleep disturbances in boys, while Feng et al. studied healthy young adults selected for sleep reactivity instead of healthy adults with chronic insomnia [5, 6].

The most crucial relative assessment is that no single study integrates all of the research design elements essential for a rigorous causal test. The most well-suited studies for diagnostic pairing actually have weaker sampling frequency for cortisol, whereas the studies with the strongest stress-reactivity designs have weaker diagnostic match. This exchange clarifies why literature can support HPA-axis activity without proving causation for adult chronic insomnia. The research gap is that the strengths of one study systematically compensate for weaknesses in another without being integrated within the same sample. Overall, results support a relationship between insomnia and cortisol, but methodological limitations limit generalizability.

Methodological differences across the four empirical studies reveal specific trade-offs between temporal order of cortisol activity and diagnostic specificity. Table 1 below summarizes the populations, cortisol assessments, and key contributions and limitations of each study.

Table 1: Comparison of Empirical Evidence on Cortisol and Insomnia

Study	Sample	Cortisol Assessment	Contribution
Passos et al.	Adults with chronic insomnia; Brazil	Morning serum cortisol	Direct clinical evidence; cross-sectional design and single sampling
Yu et al.	Adults with chronic insomnia; China	Serum cortisol	Direct clinical evidence; cross-sectional design and single sampling
Kater et al.	Adolescents; Germany	Salivary cortisol	Indirect mechanistic evidence; repeated sampling (6 time points)
Feng et al.	Young adults (healthy); China	Salivary cortisol	Indirect vulnerability evidence; repeated sampling (4 time points)

Note: The reviewed studies differ significantly in their populations, cortisol assessments, and direct comparisons [5-8].

Cross-Study Synthesis

Across the key studies, the most consistent outcome is the association between HPA-axis dysregulation and sleep-related vulnerability or insomnia severity. Passos et al. argued that higher morning serum cortisol was linked to greater insomnia severity [7]. Yu et al. established greater pre-sleep arousal and sleep reactivity were found together with higher serum cortisol and neuroendocrine activity in chronic insomnia [8]. Kater et al. found greater sleep disturbance during acute stress was associated with higher cortisol activity in adolescent boys [6]. Feng et al. identified that healthy young adults with high sleep reactivity reported stronger and more prolonged post-stress cortisol responses [5].

These results converge at the level of stress-response activation. They align with the reviewed hyperarousal framework, contemporary synthesis, and stress-vulnerability model [1, 2, 4]. They are also compatible with the meta-analytic evidence that cortisol is moderately elevated overall in chronic insomnia, though heterogeneity remains across studies [9]. Two studies center on adults with chronic insomnia, with one on adolescents with insomnia or sleep disturbances, and one on healthy young adults categorized by susceptibility. Furthermore, the outcome varies across studies. The general inference is that HPA-axis dysfunction is relevant to insomnia vulnerability and sleep disturbance. The evidence does not yet recognize one replicated cortisol abnormality that can be treated as the biological mechanism of insomnia.

Geographic generalizability represents a significant limitation across all literature. All reviewed empirical sources were conducted globally, including samples from Brazil, China, and Germany [5-8]. Despite similar findings across multiple countries that provide evidence of cross-national convergence, the lack of American data limits inferences to current demographics. Therefore, replication in diverse U.S. samples is warranted to determine whether associations between insomnia-related hyperarousal and cortisol dysregulation generalize across sociocultural contexts.

Cortisol Dysregulation as a Cause or Consequence

The paramount open question is whether cortisol dysregulation causes insomnia or is just a result of it. Passos et al. and Yu et al. cannot determine temporal order as cross-sectional studies [7, 8]. Chronic insomnia could disrupt HPA-axis dysregulation, and elevated cortisol could also maintain the arousal state. Realistically, psychological pressure could impact both. Statistical adjustment can control selected confounding variables but fails to establish temporal order. Feng et al. and Kater et al. improve temporal precision in laboratory settings by measuring cortisol reactivity to regulated stress [5, 6]. However, both studies have yet to establish that persistent adult chronic insomnia is predicted by elevated cortisol. One lacks longitudinal outcome data and the other examines adolescents [5, 6].

Their findings support stress-reactivity and vulnerability hypotheses, though they fail to present the onset of insomnia disorder. This ambiguous causation establishes how cortisol should be characterized. Naming cortisol as an activation approach assumes that it is part of a proven system for treating or preventing insomnia. The reviewed articles support association, correlation, and stress-related dysregulation. It does not establish temporal order, sufficiency, or necessity. Ultimately, cortisol is a proposed pathway whose causality remains undetermined.

Future Directions

Future studies should synthesize the best attributes of the existing literature. An adult sample should include individuals who do not currently meet the criteria for insomnia and be categorized into sleep-reactivity groups using the framework described by Kalmbach et al. [4]. Diagnostic interviews should determine baseline insomnia status, and sleep assessments should characterize sleep continuity and duration. Repeated cortisol testing should document pre-sleep and morning baseline levels, daytime decline, nocturnal activity, and stress reactivity. Repeated within-subject testing would be particularly useful, as it would ensure that cortisol patterns are synchronized with sleep patterns. Such findings may support a vulnerability mechanism, become more consistent with a maintenance mechanism, or be characterized as a consequence of chronic insomnia.

Future longitudinal research, integrated with multimodal assessments, should obtain diverse U.S.-based adult samples to address the major geographic gap in current empirical research. Moreover, further research is warranted to establish the temporal relationship between chronic insomnia and cortisol dysregulation and to address inconsistencies in how cortisol dysregulation has been operationalized. Current studies provide evidence of a relationship between chronic insomnia and cortisol, yet it remains unclear whether altered HPA-axis activity occurs before or after the onset of the disorder [7, 8]. Experimental stress-reactivity studies measure cortisol changes frequently but do not predict adult chronic insomnia [5, 6]. Therefore, future studies should also recruit adults without insomnia across periods of stress and evaluate whether cortisol dysregulation predicts subsequent insomnia onset. Findings could determine if cortisol is a baseline risk factor causing sleep problems, the fuel behind sleeplessness, or a side effect of chronic sleep disturbance. It could also determine whether varied stress hormone cycles are defined by distinct types of sleep problems.

Conclusion

Insomnia is widely supported as a disorder related to hyperarousal, but the role of cortisol under that model necessitates better precision. The foundational research establishes HPA-axis functioning as a plausible contributor to hyperarousal, revealing that cortisol is moderately elevated across insomnia studies, and reporting significant heterogeneity [9]. The reviewed core empirical investigations refine that outcome rather than resolve it. Passos et al. associate higher morning serum cortisol with greater severity of chronic insomnia, and Yu et al. demonstrate that neuroendocrine hormones, sleep reactivity, pre-sleep arousal, and chronic insomnia covary within the same clinical sample [7, 8]. Feng et al. demonstrate that prolonged cortisol stress recovery is accompanied by high sleep reactivity in healthy young adults, while Kater et al. show sex-related stress-system variations in adolescents with sleep disturbance [5, 6]. Taken together, these sources modulate HPA-axis dysfunction more effectively than they maintain high cortisol levels.

The primary limitation is methodological synthesis. Clinical studies provide diagnostic relevance with limited temporal cortisol resolution. On the other hand, stress-reactivity studies provide timing detail with weaker clinical specificities. Experimental stress-reactivity studies suggest that vulnerability might be expressed through exaggerated stress responses or delayed recovery instead of through consistently elevated basal concentration levels. Thus, cortisol dysregulation remains a plausible, phenotype-sensitive neuroendocrine factor of hyperarousal than as an established causal agent. Prospective longitudinal, multimodal studies employing assessments of cortisol, psychological hyperarousal, and objective sleep are needed to determine whether cortisol dysregulation develops as a consequence of chronic sleep disruption, is associated with its maintenance, differs across insomnia phenotypes, or precedes the onset of insomnia. Causal direction also remains unknown due to clinical

studies being largely cross-sectional, as stress-reactivity studies do not predictively illustrate the onset of adult chronic insomnia.

References

1. Bonnet MH, Arand DL. Hyperarousal and insomnia: state of the science. Sleep Med Rev. 2010. 14: 9-15.
2. Dressle RJ, Riemann D. Hyperarousal in insomnia disorder: current evidence and potential mechanisms. J Sleep Res. 2023. 32.
3. Riemann D, Benz F, Dressle RJ, Espie CA, Johann AF, et al. Insomnia disorder: state of the science and challenges for the future. J Sleep Res. 2022. 31.
4. Kalmbach DA, Anderson JR, Drake CL. The impact of stress on sleep: pathogenic sleep reactivity as a vulnerability to insomnia and circadian disorders. J Sleep Res. 2018. 27.
5. Feng Y, Chen J, Hu Z, Liu G, Zhou Y, et al. Effects of sleep reactivity on sleep macro-structure, orderliness, and cortisol after stress: a preliminary study in healthy young adults. Nat Sci Sleep. 2023. 15: 533-546.
6. Kater M, Werner A, Schlarb AA, Lohaus A. Stress reactivity in salivary cortisol and electrocardiogram in adolescents: investigating sleep disturbances and insomnia. J Sleep Res. 2022. 31.
7. Passos GS, Youngstedt SD, Rozales AA, Ferreira WS, De-Assis DE, et al. Insomnia severity is associated with morning cortisol and psychological health. Sleep Sci. 2023. 16: 92-96.
8. Yu K, Xia L, Chen H, Zou T, Zhang Y, et al. Association between sleep reactivity, pre-sleep arousal state, and neuroendocrine hormones in patients with chronic insomnia disorder. Nat Sci Sleep. 2024. 16: 1907-1919.
9. Dressle RJ, Feige B, Spiegelhalder K, Schmucker C, Benz F, et al. HPA axis activity in patients with chronic insomnia: a systematic review and meta-analysis of case-control studies. Sleep Med Rev. 2022. 62: 101588.

Copyright: © 2026 Xaviera Gadpaille, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.