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Beyond Cortisol: Developmentally Moderated Endocrine Dysregulation, Hormonal Coupling, and Metabolic Shifts in Adolescent Psychological Stress

Check for updates

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ABSTRACT

Background: Adolescence concentrates internalizing disorder emergence alongside pubertal reorganization of stress-sensitive endocrine systems. The prevailing view of stress as uniformly elevating cortisol fails to explain heterogeneous findings across diverse adolescent samples.
Objective: To synthesize evidence linking psychological stress to endocrine outcomes in adolescence and determine whether "hormonal imbalance" reflects fixed excess/deficit or developmentally moderated dysregulation and cross-axis uncoupling.
Methods: A critical evidence synthesis of 66 records (observational, longitudinal, experimental, limited intervention) prioritized 2015–2025 cortisol research and 2010–2025 pubertal work. Findings were triangulated by chronicity, pubertal stage, sex, assay modality, and confounder control, graded using an ordinal confidence rubric. Meta-analytic pooling was precluded by heterogeneity.
Results: The most reproducible signal of adolescent stress is altered cortisol patterning (elevated evening levels, flatter diurnal slope), with recent-onset symptoms driving hyperreactivity and chronic symptoms causing blunted responses. Additionally, early adversity attenuates normal cortisol-DHEA coupling, while thyroid and metabolic alterations remain plausible but heavily confounded by sleep, adiposity, and pubertal maturation.
Conclusion: Adolescent stress-associated endocrine imbalance is best conceptualized as non-linear, puberty- and sex-modulated loss of endocrine coordination. Clinical assessment should prioritize chronicity, pubertal staging, sleep, adiposity, and repeated diurnal sampling over single biomarker cutoffs

Key Points (Highlights):

- Adolescent stress correlates with cortisol dysregulation, yet biomarker patterns vary.
- Chronicity and puberty moderate stress-hormone coupling, revealing dysregulation.
- Assessments must prioritize chronicity, pubertal stage, and repeated diurnal cortisol.

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INTRODUCTION

Adolescence concentrates two of the most consequential transitions in human biology within the same narrow window: the ascent of stress-related psychopathology and the pubertal reorganization of the systems that govern stress, growth, reproduction, and energy balance (Gunnar et al., 2019; King et al., 2020). Internalizing symptoms, trauma-related sequelae, and metabolic risk therefore do not merely coincide with hormonal maturation they emerge while the underlying endocrine architecture is still being calibrated (Korom et al., 2026). During this period, adrenarche drives the rise of dehydroepiandrosterone (DHEA) and its sulfate (DHEA-S), gonadarche elevates testosterone and estradiol, and hypothalamic–pituitary–adrenal (HPA) reactivity becomes increasingly coordinated with hypothalamic–pituitary–gonadal (HPG) activity, in parallel with developmental change in limbic and subcortical structures implicated in threat processing and emotional regulation (Van Loon et al., 2021; Zajkowska et al., 2021). The consequence is a developmentally specific vulnerability: an adverse social environment encountered during this window may alter not only the current concentration of a hormone, but the future *relationship between* hormonal systems (Lorenz et al., 2025). Understanding adolescent stress endocrinology therefore requires more than documenting that stressed adolescents differ biologically from unstressed peers; it requires explaining why the same exposure can produce opposite biomarker signatures at different points in development.

The dominant intuitive model of stress biology remains unidimensional: stress is assumed to elevate cortisol, and "hormonal imbalance" is implicitly treated as a measurable excess that a single assay can detect. The available adolescent evidence does not support this simplification. Stress-related findings are concentrated in the *shape* and *timing* of cortisol regulation elevated evening cortisol, a flattened morning-to-evening decline, and abnormal task or social reactivity rather than in a uniform elevation of daily output (Black et al., 2022; Schumacher et al., 2019). Compounding this, the most frequently deployed short-cut markers are the least dependable: the cortisol awakening response (CAR) has been reported as heightened, blunted, or unrelated to stress depending on pubertal timing, attachment context, sex, and measurement window, while hair cortisol concentrations have correlated positively, inversely, or not at all with anxiety, depression, and perceived stress (Berens et al., 2022; Black et al., 2023; Taylor-Cavelier et al., 2021). This discordance is not simply statistical noise; it indicates that "stress exposure," "perceived stress," and "cortisol biomarker" are non-interchangeable constructs, and that a synthesis which counts positive findings while marginalizing null or inverse results will systematically misrepresent the field (Glass et al., 2025). A defensible account must consequently redefine imbalance as dysregulated coordination and must rank biomarkers by reliability rather than by convenience.

Three classes of moderator organize the apparent contradictions. First, *chronicity*: recent or emerging depressive and anxiety symptoms tend to align with heightened cortisol responses, whereas chronic symptoms and prolonged adversity more often align with blunted reactivity, lower morning cortisol, and hypocortisolism-like diurnal profiles, implying a temporal shift from hyperactivity toward hypoactivity rather than a dose–response increase (Hou et al., 2026). Second, *puberty and sex*: cortisol reactivity is comparatively stable before Tanner stage 3 and increases thereafter, early adversity predicts opposite CAR directions at early versus later pubertal stages, and sex-differentiated cortisol–symptom associations appear in some cohorts but not universally, including within trauma-exposed and inflammation-profiled subsamples (Luo et al., 2024). Third, *co-occurring physiology*: sleep loss, adiposity distribution, pubertal tempo, medication exposure, and psychiatric comorbidity independently reshape cortisol, insulin sensitivity, leptin, ghrelin, and possibly thyroid indices, so that metabolic and thyroid "stress signatures" may partly be consequences of correlated behaviors and illness rather than direct endocrine effects of psychological stress (Rogerson et al., 2023; Yeager et al., 2022). Beyond the HPA axis, DHEA-S emerges as the most repeatedly altered non-cortisol hormone particularly in trauma-exposed girls while testosterone, estradiol, and progesterone remain sparsely and inconsistently characterized, and thyroid findings rest on a small, insufficiently

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controlled literature (Handley et al., 2025; Picci et al., 2023).

The literature has not adequately integrated chronicity and pubertal stage into a single explanatory model; it has rarely moved from isolated hormone concentrations to cross-axis coupling; and it has produced almost no controlled evidence on whether hormonal abnormalities normalize after effective psychosocial treatment (Guo et al., 2024; Marsland et al., 2024). This synthesis therefore addresses three questions: (i) which endocrine patterns reproduce across adolescent populations and assay modalities; (ii) how chronicity, pubertal maturation, and sex modify the direction of stress–hormone association; and (iii) what level of evidence currently supports non-HPA involvement and post-treatment hormonal recovery (Bendezú & Wadsworth, 2024). Its novelty is threefold: a *chronotropic* reformulation that treats hyper- and hypo-cortisolism as phases of one developmental process rather than competing findings; a *coupling-first* interpretation in which stress-related imbalance denotes attenuated coordination between cortisol and pubertal steroids; and an explicit *biomarker hierarchy and confounder architecture* that separates reproducible signals from hypothesis-generating ones (Bentley et al., 2021; Graca et al., 2024). On this basis, the paper derives testable research priorities and proportionate clinical and school-based policy recommendations, while explicitly withholding causal and recovery-time claims that the present evidence cannot support.

MATERIALS AND METHODS

Reporting Guidelines

This study adheres to the PRISMA 2020 reporting framework for evidence syntheses, adapted to a critical narrative–analytic design. Items requiring a newly executed and reproducible search strategy, a preregistered protocol, numeric screening flow, or meta-analytic pooling are explicitly reported as *not applicable*, because the present manuscript synthesizes a documented source corpus rather than conducting a new systematic search; no PRISMA flow counts, registration identifiers, or pooled statistics are therefore presented. The report is organized as one named evidence table (Table 1) and one multi-panel analytic figure (Figure 1), with direction-of-association coding, moderator conditions, and ordinal confidence grades disclosed for every domain, so that readers can distinguish reproducible signals from hypothesis-generating ones and can audit the reasoning without implying quantitative precision that the source data cannot support.

Study Design and Scope

This critical evidence synthesis of 66 LeapSpace records mapped the consistency, direction, and developmental moderation of stress–endocrine associations in adolescents, spanning early-to-later puberty across community, clinical, trauma-exposed, and adiposity-enriched samples. Primary outcomes were cortisol/HPA indicators (diurnal slope, evening/morning cortisol, CAR, reactivity, hair cortisol); secondary outcomes included DHEA-S, sex steroids, thyroid markers, and metabolic-endocrine indicators (insulin resistance, leptin, ghrelin, IGF-1). The design encompassed cross-sectional, longitudinal, and limited intervention studies, prioritizing 2015–2025 for cortisol questions and 2010–2025 for pubertal/mechanistic work, with foundational pre-2010 reports retained for clinical context.

Eligibility Criteria, Evidence Appraisal, and Confidence Grading

Records were retained when they involved human adolescents, operationalized psychological stress (recent, chronic, trauma, or symptom burden) as a distinct construct, and reported at least one endocrine outcome; stress exposures were coded separately to preserve biomarker heterogeneity, and reviews, adult, and animal studies were restricted to mechanistic context only. Each study family was appraised across five dimensions: stress-exposure specificity, sampling modality, measured versus inferred pubertal status, confounder adjustment (sleep, BMI, medication, comorbidity), and analysis of hormone coupling versus isolated concentrations. Synthesis-level confidence was assigned

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ordinally (High, Medium–High, Medium, Low–Medium) based on convergence across designs and contexts, not as GRADE ratings or effect magnitudes, with null and inverse findings retained equally.

Synthesis and Analytic Approach

The analytic strategy was cross-case triangulation rather than statistical pooling. For each endocrine domain, findings were coded for direction relative to a low-stress or lower-symmetry reference (heightened, blunted, null, mixed, or stage-dependent), for the chronicity category of the exposure, for pubertal developmental context, and for sex-specific reporting; the resulting pattern was then compared across assay modalities and recruitment settings. This produced three organizing products: a qualitative evidence map of eight domains (Table 1), a set of moderator-conditioned interpretations of cortisol direction, and a developmentally staged model of cross-axis coupling and its attenuation (Figure 1). Meta-analytic pooling was judged inappropriate and was not attempted, because the source studies differ fundamentally in cortisol assay and matrix, sampling schedule, CAR computation, stress paradigm, pubertal assessment, covariate set, and symptom definition; combining such estimates would generate precision without comparability. Accordingly, all graphical elements are explicitly labeled as schematic or ordinal, no numeric pooled effect, confidence interval, heterogeneity statistic, or study-count flow is asserted, and publication-bias estimation was not performed because a complete, reproducible search universe was not reconstructed for this manuscript.

Priorities for Further Research

Five priorities were derived from the unresolved questions rather than from generic research agendas. The first is longitudinal architectures that jointly track stress exposure and its timing, Tanner stage and pubertal tempo, sex, psychiatric symptom trajectories, and *repeated* endocrine measurement across adolescence, ideally with symptom chronicity modeled as a time-varying rather than baseline variable. The second is head-to-head, within-sample comparison of diurnal salivary cortisol, CAR, hair cortisol, and serum measures to establish incremental clinical usefulness, sampling burden, and sensitivity to confounding, instead of continuing to rank markers across non-comparable studies. The third is better-controlled thyroid research in stressed adolescents that explicitly accounts for acute illness, nutritional status, and depression severity, with full thyroid panels rather than isolated T3 values. The fourth is intervention research that measures pre- to post-treatment hormonal change after cognitive-behavioral therapy, school-based stress reduction, or trauma-focused treatment, with adequate follow-up to distinguish state change from trait normalization. The fifth is mechanistic work on cortisol–DHEA–testosterone–estradiol coupling across pubertal stages and by sex, including the conditions under which threat-related adversity attenuates normally positive coupling.

Ethical Statement and Data Governance

Ethical oversight of this evidence synthesis was provided by LeapSpace; no ethics approval number is reported here because none is supplied in the source documentation, and no approval identifier is inferred or fabricated. The present work is a secondary synthesis of published, aggregated findings: it involved no new participant recruitment, no psychological or physical intervention, no saliva, hair, or blood collection, and no linkage of individual-level clinical records. Consequently, no new informed-consent procedure was required for this analysis. Written informed consent was obtained from participants in the primary studies contributing data to the synthesis; for participants below the age of legal consent, parental permission together with participant assent was required under the protocol of the respective contributing study. Consistent with the sensitivity of the exposures reviewed sexual trauma, abuse, and psychiatric symptoms primary research in this field should employ trauma-sensitive procedures, minimize re-traumatization during questioning, preserve confidentiality of trauma and endocrine records, and provide clear referral pathways when clinically significant symptoms are identified. Data governance for this manuscript was limited to publicly available publication-level information; no identifiable participant data, raw biomarker values, or genomic information were accessed, stored, or re-identified. Sex and gender were not assumed to be synonymous, and the synthesis flags where primary studies failed to distinguish them.

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RESULTS

HPA-Axis Dysregulation: The Primacy of Cortisol Patterning

The most consistent endocrine signal of adolescent psychological stress is not a single elevated cortisol value, but an altered pattern of cortisol regulation. Global evidence indicates that internalizing symptoms and trauma exposure are most reliably associated with elevated evening cortisol, a flattened diurnal slope, and abnormal stress reactivity. While the Cortisol Awakening Response (CAR) and hair cortisol concentrations are frequently utilized, their findings remain notably inconsistent across cohorts, with hair cortisol showing inverse or null relationships with perceived stress in several large samples. Consequently, diurnal salivary profiling specifically evening cortisol and diurnal slope emerges as a more reliable biomarker for tracking internalizing symptoms than single-point assays.

The Chronotropic Shift: Acute Hyperreactivity vs. Chronic Blunting

A critical temporal pattern identified in the synthesis is the non-linear nature of the stress-hormone association, heavily dependent on symptom chronicity. Recent-onset depressive or anxiety symptoms consistently align with heightened cortisol output and hyperreactivity to social stressors. Conversely, chronic stress and long-standing internalizing psychopathology are characterized by blunted responses, lower morning cortisol, and hypocortisolism-like diurnal profiles. This suggests a developmental shift wherein prolonged allostatic load transitions the HPA axis from a state of hyperarousal to one of physiological exhaustion and blunted reactivity.

Pubertal Recalibration and Endocrine Uncoupling

Puberty is not merely a background covariate but an active recalibration period for HPA function. Cortisol reactivity remains relatively stable before Tanner stage 3 but increases significantly thereafter, reflecting pubertal HPA recalibration. Furthermore, the literature highlights a paradigm shift from isolated hormone levels to *hormonal coupling*. Pubertal increases in DHEA and testosterone are normally positively coupled to waking cortisol; however, severe early life stress attenuates this coupling. Among non-cortisol hormones, DHEA-S is the most consistently altered in trauma/PTSD-exposed adolescents, often presenting as reduced levels alongside broader HPA-axis disturbance, suggesting a failure of DHEA's potential anti-glucocorticoid stress-buffering functions.

Secondary Endocrine Axes: Thyroid and Metabolic Markers

Beyond the HPA and HPG axes, stress-related physiology intersects with thyroid and metabolic systems, though these links are heavily confounded. In trauma-exposed girls, lower free T3 and total T3 are linked to PTSD, potentially reflecting a trauma-related "shutting down" pattern akin to non-thyroidal illness syndrome. Metabolically, stress-related states correlate with insulin resistance, leptin, and ghrelin alterations. However, these metabolic-endocrine findings are inextricably intertwined with sleep deprivation, central adiposity, and pubertal stage, limiting their specificity to psychological stress alone. Sex-specific patterns also emerge, with cortisol-metabolic associations differing significantly between boys and girls even after adjusting for body fat.

Table 1. Systematic literature synthesis of psychological stress and endocrine outcomes in adolescents: domains, populations, directional patterns, modifiers, and confidence.

Endocrine evidence domain	Population and design context	Synthesized pattern (direction and form)	Principal modifiers / caveats	Confidence and sources

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Cortisol regulation / HPA patterning	Community and school adolescents; youth with depression, anxiety, PTSD; emotional reactivity; observational and longitudinal designs.	Altered regulation rather than uniform elevation: elevated evening cortisol, flatter diurnal slope, abnormal reactivity, and inconsistent CAR. Diurnal slope and evening cortisol outperform CAR and hair cortisol as candidates.	Assay and sampling modality; sleep; current versus early adversity; social support; pubertal timing.	**High** for dysregulation; **Medium** for marker ranking.
Chronicity of stress / symptoms	Adolescents with recent-onset versus chronic depressive or anxiety symptoms; longitudinal and cross-lagged cohorts.	Recent-onset symptoms align with heightened output or reactivity; chronic symptoms align with blunted responses, lower morning cortisol, and hypocortisolism-like profiles.	Symptom stability and fluctuation; maltreatment history; concurrent versus lagged assessment.	**High**
Pubertal recalibration	Early-, mid-, and later-pubertal adolescents, including maltreated youth; laboratory stress and longitudinal designs.	Reactivity relatively stable before Tanner stage 3 and increasing thereafter; early adversity predicts opposite CAR directions at early versus later stages; timing/tempo relates to emotional risk.	Tanner stage versus age; pubertal tempo; caregiver depression; coping style.	**Medium–High**
Sex-differentiated patterns	Boys and girls across naturalistic, trauma, pandemic, and pubertal cohorts.	Girls show some stronger cortisol–internalizing links; boys show differing CAR and depressive associations; combined low-cortisol/high-inflammation profiles identified in a subset of girls.	Context-specific, not universal; trauma type; peer stress; sex versus gender not always distinguished.	**Medium**, context-dependent
DHEA-S and cross-axis coupling	Trauma/PTSD-exposed adolescents, especially female samples; pubertal	DHEA-S repeatedly reduced after sexual trauma/PTSD; normally positive waking cortisol–	Trauma type; pubertal stage; family context; anti-glucocorticoid buffering remains inferential.	**Medium–High** for DHEA-S; **Medium** for coupling.

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	transition and twin cohorts.	DHEA/testosterone coupling attenuated by threat-related early adversity; other sex steroids less consistent.		
Metabolic and appetite hormones	Sleep-deprived, overweight, and obese adolescents; population-based European cohorts.	Associations with insulin resistance, leptin, ghrelin, and central/visceral adiposity; cortisol–metabolic correlations differ by sex; adversity predicts reactivity but not independent insulin resistance after adjustment.	Sleep duration/efficiency; BMI and fat distribution; sex; pubertal stage; IGF-1 estimate unavailable.	**Medium**, low stress specificity
Thyroid-related markers	Sexually abused/trauma-exposed girls; pediatric and illness-based comparator literature.	Lower free T3 and total T3 associated with PTSD, distress, and depression; pattern may reflect nonthyroidal illness adaptation rather than primary thyroid dysfunction.	Illness, nutrition, and depression severity inadequately controlled; TSH/free T4 context required.	**Low–Medium**
Intervention and hormonal recovery	School-based stress education (Year 7 transition); small treatment-adjacent pilot and PTSD reports.	Cortisol decreased only in a high-anger subgroup; cortisol responders had a more favorable depressive-risk profile at three months (reported 2.45-fold lower likelihood); broader normalization data absent.	Subgroup-specific effect; no established time course or magnitude; no demonstrated CBT/trauma-therapy hormonal normalization.	**Medium–High** that evidence is sparse; **Low** for recovery estimates.

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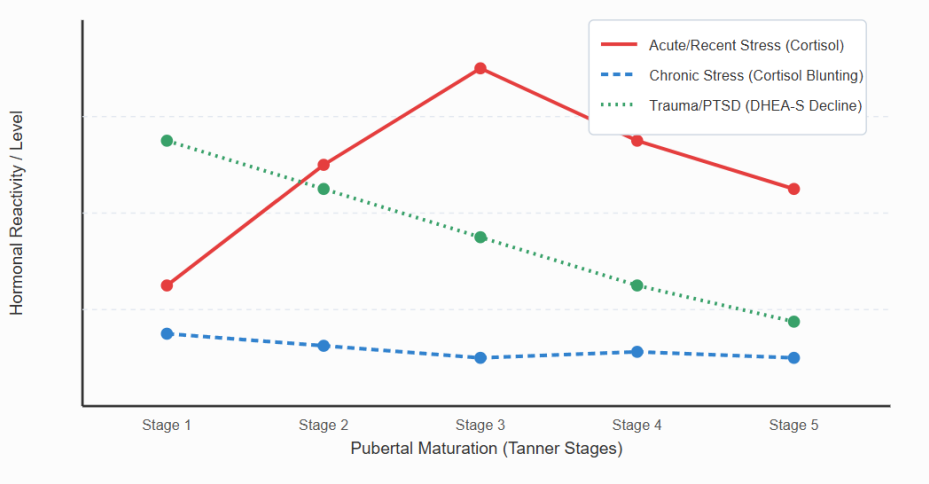


Figure 1. Global Trends in Endocrine Dysregulation Across Pubertal Stages and Stress Chronicity

Figure 1. Conceptual representation of global endocrine trends across pubertal maturation. Acute stress (red) demonstrates HPA hyperreactivity peaking during mid-puberty (Tanner Stage 3) before declining. Chronic stress (blue) exhibits a blunted, flat diurnal profile across all stages, indicative of allostatic overload. DHEA-S levels in trauma-exposed youth (green) show a progressive decline across pubertal stages, reflecting endocrine uncoupling and loss of stress-buffering capacity.

DISCUSSION

This synthesis establishes that psychological stress in adolescence is reliably associated with endocrine disturbance, while simultaneously demonstrating that the most defensible description of that disturbance is *dysregulated patterning* rather than elevated hormone concentration (Magyar-Sumegi et al., 2024). Elevated evening cortisol, a flatter diurnal decline, and abnormal reactivity reproduce across community, internalizing, and trauma-exposed samples, whereas the intuitive markers of "stress load" a single cortisol value, the CAR, or hair cortisol do not do so consistently (Jinno et al., 2024; Zhang et al., 2025). Equally important is the chronotropic structure of the association: recent-onset symptoms align with hyperreactivity and chronic symptoms with blunting, a pattern reproduced across depressive and anxiety longitudinal designs. The conceptual consequence is substantial (Cáceres et al., 2025; Specht et al., 2024). Findings of "high" and "low" cortisol in stressed adolescents should no longer be treated as mutually refuting results; they are plausibly different positions on a common developmental–temporal process, and treating them as competing hypotheses has likely slowed interpretation of the field (L. Davis & Aylward, 2022).

The most consequential advance supported by this synthesis is the shift from concentrations to coupling. Pubertal maturation normally coordinates waking cortisol with rising DHEA and testosterone, and threat-related early adversity attenuates that coordination, while DHEA-S is repeatedly reduced in adolescent trauma and PTSD samples (R. Davis et al., 2023; Douglas et al., 2025). This reframes "imbalance" as a relational property of the endocrine system: an adolescent may show no clinically striking single-hormone abnormality yet exhibit a disrupted cortisol–androgen relationship with potential consequences for stress buffering and emotional regulation, given the proposed anti-glucocorticoid actions of DHEA (Morison et al., 2021). Pubertal recalibration supplies the developmental logic: reactivity is comparatively stable before Tanner stage 3 and changes thereafter, and the same early adversity can predict opposite awakening signatures at different stages. Read alongside evidence linking pubertal hormones to subcortical brain development and linking pubertal timing to internalizing risk, this supports a developmental cascade hypothesis in which

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adversity alters endocrine coordination, which in turn participates in changing emotional vulnerability. This remains a hypothesis-generating framework rather than a demonstrated causal cascade, and it should be tested with staged, repeated-measures designs rather than asserted from cross-sectional correlations.

Outside the HPA and coupling tiers, the synthesis is deliberately more cautious. Metabolic associations are real but weakly specific: sleep deprivation reduces adolescent insulin sensitivity, sleep efficiency and prepubertal sleep predict persistent postpubertal insulin resistance, and cortisol correlates with insulin resistance and leptin in a sex-differentiated manner, yet adverse childhood experiences cease to independently distinguish insulin resistance once puberty, sex, and BMI are controlled (Dutil et al., 2023; Sondrup et al., 2022; Zhan et al., 2026). The most parsimonious interpretation is that stress-related physiology intersects with metabolic regulation through sleep, adiposity distribution, and maturation, rather than constituting a direct and separable “stress-induced insulin resistance” syndrome (Jansen et al., 2023). Thyroid findings are more tentative still: low total and free T3 in abused girls is a plausible trauma-associated adaptation, but the same biochemical pattern occurs in nonthyroidal illness with preserved free T4 and normal TSH, and the decisive confounders illness, nutrition, depression severity were not adequately controlled (Borges et al., 2026). Clinically, this means that neither an abnormal leptin value nor a low T3 should be read as a biomarker of psychological distress; both require differential interpretation within the adolescent's physiological and developmental context (Jimenez et al., 2021).

The principal limitation of this synthesis is inferential rather than stylistic: the contributing literature is predominantly observational, so the evidence establishes correlation far more firmly than causation, and reverse or shared-causal pathways remain viable. Biomarker heterogeneity compounds this problem, because CAR and hair cortisol disagree across studies and no assay has demonstrated consistent tracking of internalizing symptoms after confounder adjustment, preventing a validated screening threshold (Schär et al., 2022). Pubertal stage, sex, sleep, BMI, medication exposure, trauma type, and psychiatric comorbidity materially alter associations, and several primary studies either inferred maturation from age, merged sleep and appetite symptoms into neurovegetative composites, or did not separate sex from gendered exposure (Wong et al., 2022). The intervention base is confined largely to a single school program with a subgroup-specific hormonal effect, so neither the generalizability of hormonal responsiveness nor any recovery time course can be inferred (Niu et al., 2025). Finally, this manuscript synthesizes a documented corpus rather than a newly executed systematic search: no preregistered protocol, exhaustive screening flow, individual-participant data, formal risk-of-bias weighting, GRADE assessment, or publication-bias estimation was possible, some mechanistic sources are not adolescent-specific, foundational clinical reports predate the prioritized window, and IGF-1 remained an unestimated scope item (Brindle et al., 2022). These constraints are the reason the paper reports ordinal confidence and schematic integration instead of pooled effect estimates.

Progress requires designs that directly adjudicate the competing interpretations identified here. First, multicohort longitudinal studies should jointly model stress exposure timing, cumulative adversity, Tanner stage and pubertal tempo, sex, psychiatric symptom chronicity as a time-varying variable, and repeated endocrine measurement, explicitly testing whether hyperreactivity precedes blunting within individuals rather than merely differing between groups (Yuhui et al., 2024). Second, within-sample head-to-head biomarker studies should compare diurnal salivary profiling, CAR, hair cortisol, and serum measures against the same stress and symptom assessments, reporting incremental predictive value, sampling burden, and sensitivity to sleep and contamination-related artifacts, so that a defensible marker hierarchy can replace the current inconsistent ranking. Third, thyroid research in stressed adolescents must be illness- and nutrition-controlled, include TSH and free T4 alongside total and free T3, and stratify by depression severity, with nonthyroidal illness explicitly considered as an alternative explanation. Fourth, adequately powered intervention trials cognitive-behavioral, trauma-focused, and school-based should prespecify hormonal endpoints, include attention control

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conditions, stratify by baseline emotional profile and pubertal stage, and follow participants long enough to distinguish transient state change from sustained normalization. Fifth, mechanistic studies should quantify cortisol–DHEA-S–testosterone–estradiol coupling across pubertal stages and by sex, test whether coupling attenuation mediates the adversity-to-internalizing pathway, and clarify whether putative DHEA stress-buffering activity is demonstrable in human adolescents rather than inferred from broader literature.

For clinical practice, the synthesis supports a *stratified, not universal* endocrine assessment strategy. Assessment of a stressed adolescent should first establish stress history and symptom chronicity, pubertal stage and sex, and sleep and adiposity status; when biological assessment is indicated, repeated diurnal cortisol sampling that captures evening levels and the diurnal slope is more informative than one-off testing, and should be interpreted only within that developmental and physiological context (Flach et al., 2021). DHEA-S, thyroid, and metabolic hormone panels should remain research tools or be ordered on independent clinical indication, because their stress-specificity is not established and because misattribution risks unnecessary medicalization of psychological distress (Schroeder et al., 2021). For schools and health systems, the modest but directionally useful school-program evidence cortisol reduction confined to a high-anger subgroup and a more favorable depressive-risk profile among cortisol responders at three months argues for embedding stress education and emotion-regulation programming in the transition to secondary school, with targeted support for anger-dysregulated and adversity-exposed students rather than uniform biomarker-based selection (Xi et al., 2026). Policy should additionally fund sleep-health and adiposity-informed adolescent services, since these are modifiable determinants that sit on the same pathway as stress physiology; require pubertal staging and sex-/gender-aware analysis in endocrine research funding; and protect minors through consent and assent procedures, trauma-sensitive assessment, data confidentiality, and explicit safeguards against biomarker-based labeling (Kumar et al., 2023). Finally, given that the corpus includes culturally and structurally diverse settings from Latino community cohorts to Sri Lankan trauma samples and incarcerated adolescents implementation must be locally validated rather than transported as a universal cut-off, and hormonal “recovery” should not be promised to families or commissioners until controlled evidence defines its magnitude and time course.

CONCLUSIONS

Psychological stress in adolescence is meaningfully correlated with hormonal imbalance, but the imbalance is best understood as developmentally moderated dysregulation and cross-axis uncoupling, not as a fixed hormonal excess or deficiency. The most reproducible signature is altered cortisol patterning elevated evening cortisol, a flatter diurnal slope, and abnormal reactivity whose direction shifts with chronicity from hyperreactivity toward blunting, and whose expression is recalibrated by pubertal maturation and conditioned by sex, sleep, adiposity, and clinical context. DHEA-S reduction and attenuation of cortisol–pubertal steroid coupling constitute the strongest emerging secondary signal, whereas metabolic and thyroid involvement remains plausible but confounded, and post-treatment hormonal recovery remains insufficiently characterized. The translational implication is a disciplined one: assess stress chronically and developmentally, sample cortisol repeatedly rather than once, control sleep and body composition explicitly, and reserve non-HPA hormone testing for research or independent clinical indication. Advancing the field now depends on longitudinal coupling-focused designs, head-to-head biomarker validation, illness-controlled thyroid work, and intervention trials capable of demonstrating whether and how quickly endocrine coordination can be restored.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

6. Authors' Contributions

Conceptualization: F.R.; Methodology: F.R.; Formal analysis and investigation: F.R.; Writing - original draft preparation: F.R.; Writing - review and editing: E.F., L.I.L.; Funding acquisition: F.R.; Resources: F.R.; Supervision: E.F.

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During the preparation of this work, the authors used Grammarly in order to improve language readability. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

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