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# The Impact of Hormones on Generalized Anxiety and Panic Disorders in Adolescents and Young Adults: A Review

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## Abstract

This review explores the role of hormones in the development and progression of Generalized Anxiety Disorder (GAD) and panic disorder (PD) among adolescents and young adults. It highlights how hormonal variations, particularly in sex hormones like estrogen, progesterone, and testosterone, as well as stress hormones, for example, cortisol, can influence brain regions involved in emotional regulation, for example, the amygdala and prefrontal cortex. The review examines gender differences, clarifying that females are more susceptible to anxiety disorders, potentially as a result of cyclical hormonal changes. Additionally, it discusses how puberty, a critical developmental stage, marks a period of increased vulnerability due to major hormonal shifts. Understanding these hormonal impacts may improve diagnosis, prevention, and treatment strategies for GAD and PD in young people. Also, this review explains the definition and types of anxiety disorders and Prevalence in Adolescents and Young Adults. The review also discussed the symptoms and signs of generalized anxiety disorder and panic disorder, and the optimal treatment for recovery.

**Keywords:** Anxiety, Panic Disorders, Adolescents, cortisol, stress hormones.

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## 1. Introduction

Anxiety disorders have been defined in the DSM-IV as a class of disorders that share features of excessive fear and anxiety. Anxiety disorders are among the most frequently diagnosed psychiatric disorders in adolescents (Kostev et al.2023). Hormones play a substantial role in adolescent generalized anxiety disorder and panic disorder concerning expression, onset, and severity (Pillerová et al.2022). Hormonal involvement is important to consider when assessing anxiety disorders during adolescence and early adulthood. Due consideration of hormonal changes during this developmental period may facilitate diagnosis and treatment of anxiety disorders in adolescence and early adulthood (Xie et al., 2021).

In keeping with this scope on hormones in adolescence and early adulthood, the terms Adolescents (human 13–18 years) and Young Adults (human 18–30 years) will be operationally applied, except where reference to pre-adolescents or adults is required (Bougeard et al.2021). The term generalized anxiety disorder will encompass both generalized anxiety disorder and panic disorder, since the two disorders are found together

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in Syndrome E. This syndrome involves symptoms of both generalized anxiety disorder and panic disorder and is most prevalent in adolescence and early adulthood. Due to the prevalence of the two disorders in this syndrome during adolescence and early adulthood, and to link the two disorders within Syndrome E, symptoms of both of these anxiety disorders will be discussed (Machaj et al.2022; Yuan et al.2023).

Anxiety disorders are highly comorbid with depression, rendering neural circuit analysis difficult. Explorations into molecular and cellular factors precipitating anxiety have yielded an incomplete understanding of its neuroanatomical substrates (K Schmidt et al., 2018). Nonetheless, epidemiologic data indicate a 25% lifetime prevalence in the United States, with particular onset in early life and tendencies toward chronicity. Adolescents more frequently experience anxiety compared to the general population (Parodi et al.2022).

## 2. Understanding Anxiety Disorders

Anxiety encompasses a diverse family of apprehensive states marked by concerns about the future (Challa and Alahari2023). Proceeding uneventfully along the path of life brings about relatively little dread, but when a person is exposed to uncertain and potentially threatening situations, worries that something might go wrong arise (Brown et al.2023). These worries, however, must remain at a manageable level for the person to function normally. When the magnitude of anxiety is excessive, sustained, and largely uncontrollable, this contributes to a variety of anxiety disorders that are among the most common mental health problems in modern societies (K Schmidt et al., 2018).

Anxiety disorders are among the most prevalent psychological disorders worldwide, with women almost twice as likely as men to develop an anxiety disorder in their lifetime (Z. Farhane-Medina et al., 2022). The worldwide prevalence of anxiety disorders is about 7 percent, with a lifetime prevalence of 25 percent in the United States (Javaid et al.2023). There is considerable heterogeneity among anxiety disorders, with the DSM-IV listing panic disorder (comorbid with agoraphobia), specific phobia, social phobia, post-traumatic stress disorder (PTSD), generalized anxiety disorder, and obsessive-compulsive disorder (Dalton et al.2025). The physiological and hormonal characteristics of these conditions differ to some extent, and this chapter concentrates on the best-studied examples corresponding to discrete and generalized apprehensive responses (G. Cameron & M. Nesse, 1988).

Anxiety disorders differ from phobic states primarily in their persistence (Hovenkamp-Hermelink et al.2021). Anxiety represents the primary disturbance in generalized anxiety disorder (GAD), panic disorder, and agoraphobia, whereas in phobic disorders, additional symptoms can dominate (Oake & Pathak). Post-traumatic stress disorder (PTSD) is an unusual form characterized by brief periods of terror (a panic-like attack) that are strongly linked to a specific traumatic event (Sarapultsev et al.2024). Obsessive-compulsive disorder has many behavioral parallels to anxiety disorders, but is best considered separately because of the diverging mechanisms involved (Pardossi et al., 2024).

### 2.1. Definition and Types

Anxiety disorders are characterized by inappropriate, excessive fear and avoidance responses to perceived threats (Brown et al.2023). Although these disorders impact individuals of all ages, they are particularly prevalent in children and adolescents, exerting long-term adverse effects (Warda et al., 2023). Prevalence rates highlight the significance of these conditions, with 9.1% of children and 8.1% of adolescents afflicted by anxiety disorders and specific phobias (Creswell et al., 2014). A range of pharmacological and

psychological interventions exists, with research indicating differential efficacy across disorder categories and age groups. Consequently, treatment guidelines distinguish between disorder types (panic disorder-agoraphobia, generalized anxiety, social anxiety, separation anxiety, specific phobia, and selective mutism) and populations (adults, children, and adolescents) (First et al.2021). In youths, the diagnostic nomenclature aligns with that of adults, with criteria extrapolated where specific age-related guidelines are absent. These disorders manifest through distinct patterns of anxiety and behavioral change, with individual variation within each type (Alinsky et al., 2022).

### 2.2. Prevalence in Adolescents and Young Adults

Anxiety disorders constitute the most prevalent category of psychological distress during childhood and adolescence. Excessive and uncontrollable anxiety that impairs normal functioning has a lifetime prevalence of 25.1% in the United States, concurrently exhibiting the youngest age of onset and greatest chronicity of any mood or substance abuse disorder (K Schmidt et al., 2018). Already 31.9% of adolescents 13–18 years of age meet anxiety diagnostic criteria. 4.7% of individuals aged 13–25 years suffer from generalized anxiety disorder (McCoy, 2023). Anxiety disorders are common comorbidities with other major depressive, bipolar, and psychotic illnesses (Saha et al.2021). Similarly, up to 20% of adolescents across the United States encounter a mental health disorder (Bitsko, 2022). Suicide is the second leading cause of death among 10–24-year-olds, and both rising suicide and mental health incidence rates are associated with concurrent diagnoses of anxiety and depression (McClaine Josey, 2016).

### 2.3. Symptoms and Diagnosis

The symptoms of anxiety disorders are diverse and often nonspecific, complicating etiological identification. Such disorders are generally classified as either generalized anxiety disorder (GAD) or panic disorder (Szuhany & Simon, 2022). GAD is characterized by “free-floating” anxiety that is related to multiple and/or ill-defined concerns rather than a specific anxiety-producing stimulus. The condition typically involves a sense of near constant apprehension or foreboding, somatic complaints such as muscle tension and fatigue, and, occasionally, irritability and hot flashes (G. Cameron & M. Nesse, 1988). In contrast, panic disorder is marked by the development of apparently spontaneous episodes of intense anxiety. These attacks generally reach peak intensity within 10 minutes and rarely last more than an hour. Core features of panic episodes include symptoms such as heart palpitations, chest pain, shortness of breath, dizziness, depersonalization, and derealization. Anxiety reactions to panic attack stimuli are often present, raising the possibility of the development of both agoraphobia and generalized anxiety (Gorbis & Jajoo, 2024).

From a clinical perspective, determining whether generalized or panic anxiety is present is usually the first step in the diagnostic process (Özdemir & Kuru, 2023). The DSM-IV criteria for GAD and panic disorder emphasize the presence of physical and cognitive symptoms within relatively broad categories and require the exclusion of underlying phobic stimuli (Rosellini & Brown, 2024). Hence, many physical symptoms of anxiety disorders (quivering, dizziness, dyspnea, derealization) are also principal symptoms in other diagnostic categories such as simple phobia, major depressive episode, or somatization disorder (Kuzminskaite et al.2022). In addition to diagnostic uncertainty, the predominance of subjective symptoms in these disorders has suggested a possible physiological basis and has led clinical investigators to attempt verification of symptoms through the determination of hormone concentrations (Adewusi et al., 2021).

## 3. The Role of Hormones

Available evidence indicates that several hormonal systems are implicated in anxiety conditions

(Pillerová et al.2022). Those with difficulty with anxiety exhibit lower levels of certain chemicals in the brain and increased levels of the body's stress hormone, cortisol (Garza-Ulloa2023). Levels of stress hormone—corticosteroids, secretion of growth hormone and prolactin, and elevated levels of catecholamines are all found when anxiety disorders are present. Most studies focus either on endocrine or autonomic nervous system hormones; rarely have both been studied in the same patients. Studies that use rigorous formal diagnostic criteria have generally examined panic disorder, agoraphobia with panic attacks, or generalized anxiety disorder by formal criteria (G. Cameron & M. Nesse, 1988).

### 3.1. Hormonal Changes During Adolescence

Between childhood and adulthood, an individual undergoes many physical and psychological changes. This transition period during adolescence is characterized by an intense activation of the hypothalamic-pituitary-gonadal (HPG) axis (L. Byrne et al., 2016). This surge in hormones regulates the hypothalamic-pituitary-adrenal (HPA) axis, which controls the stress response (Janssen, 2022). Maintaining a tight balance between the two endocrinological systems is essential to support the heightened psychophysical strain the individual is put under and to ensure the healthy development of a mature neurological framework. Should the balance be broken or the hormonal chains be disrupted, physiological changes and psychological diseases may arise (Knight, 2021; Stucker et al., 2021).

During adolescence, the body experiences a surge of hormones responsible for many physical effects. Ovaries release estrogen and progesterone, while the testes manufacture testosterone (Al-Suhaimi et al.2022). The adrenal glands produce increasing amounts of circulating dehydroepiandrosterone (DHEA) and its sulfate-ester form (DHEAS) (Mueller et al.2021). Hormonal changes can affect the degree of temperament expressed and alter the functional balance of neurotransmitter systems in the brain (Bashkatov & Garipova, 2022). The biological sensitivity to the social environment is more pronounced in females, while girls generally show an earlier neural development of emotional response before the completion of the thalamo-cortical tracts (Armstrong-Carter & Telzer, 2022). Additionally, exposure to hormonal elevations leads to more extreme emotional responsiveness despite an overall broader emotional spectrum and greater expression of stimulus responses (van der Cruijsen et al., 2019). These features might contribute to a greater vulnerability, both neural and psychological, during the development and subsequent expression of internalizing symptoms, characteristics that are particularly prevalent in about 10-20% of adolescents (Doering et al.2022).

### 3.2. Impact of Stress Hormones

Catecholamines and cortisol levels tend to be elevated in anxiety disorders (Garza-Ulloa2023). Growth hormone, prolactin, and thyroid axis responses can be variable, exhibiting either elevated or normal levels. Glucose regulation often shows increased levels, and melatonin correlates with the degree of anxiety. While stimulus-induced panic attacks generally present with normal or elevated hormone levels, other hormonal responses may be blunted or inconsistent (Bliddal et al.2021; Lotito et al.2025). Hyperthyroidism can imitate panic symptoms, and certain hormonal abnormalities, such as elevated basal cortisol and altered responses to stimulation tests, are more prevalent in women (G. Cameron & M. Nesse, 1988). Brain regions sensitive to adult stressors, including the amygdala, hippocampus, and prefrontal cortex, continue maturing during adolescence. As these areas and their associated stress-sensitive neural circuits develop, they may contribute to heightened vulnerability during this period (D. Romeo, 2014).

### 3.3. Hormones and Neurotransmitter Interaction

The function of any endocrine organ is inherently dependent on the release of sufficient quantities of trophic hormones from the pituitary gland. These hormones ensure that the endocrine glands grow during development, produce the quantity of hormone appropriate for physiological needs, and respond rapidly when additional hormone is required (Al-Suhaimi and Khan2022). The major trophic hormone relevant to the thyroid gland is TSH; however, the function of the adrenal gland is also markedly altered in anxiety disorders (Schifter et al., 2021). The relationship between the adrenal and the thyroid is equally relevant to the pathophysiology of anxiety since ACTH and TSH have analogous roles in maintaining glandular structure and function. The decrease in thyroid-linked hormone and corticosteroid secretion due to medial hypothalamic ablation causes hypophagia and weight loss (Al-Suhaimi and Khan2022; BA A & P, 2023). Thyroid hormones and corticosteroids influence metabolism and appetite, and administration of T4 and corticosterone can prevent these effects of the lesion. The hypothalamic cells that maintain the thyroid and adrenal appear to be regulated separately, and their activity can be modified independently (G. Cameron & M. Nesse, 1988).

## 4. Hormonal Influences on Anxiety

Liberally distributed in the central nervous system are peptide and protein hormones such as oxytocin and vasopressin. Oxytocin appears to influence anxiety, bonding, and aggressive behaviours (G. Cameron & M. Nesse, 1988).

Among hormones, elevated levels of catecholamines and cortisol appear to be the most common abnormality in GAD. The thyroid and gonadal axes appear to be ‘‘normal’’ or show only a few abnormalities; however, abnormal thyroid function is a common feature of patients with panic disorder, and high levels of oestrogens are found in panic disorder patients who experience premenstrual symptoms (Łoś & Waszkiewicz, 2021; Jamali et al., 2025).

An increased prevalence of diffuse body pain, increased demand for veterinary analgesic agents, and a positive Faecal and Soiled Paper Test (FPT and SPT) score are all associated with the recurrent use of alpha-melanocyte-stimulating hormone (α-MSH) (Dall’Olmo et al., 2023). So contents of α-melanocyte-stimulating hormone (α-MSH) are associated with stress-related disorders, including anxiety and central sensitisation to pain (K Schmidt et al., 2018).

Newborns are repeatedly exposed to a range of influences due to various physiological, neurological, and hormonal factors, which may affect normal neuronal and behavioural development (Zhao et al., 2023).

### 4.1. Estrogens and Anxiety Symptoms

Studies have consistently highlighted the modulatory role of estrogens on anxiety-related behaviors in humans and animals. Selective estrogen receptor modulators are associated with altered anxiety-like behavior. Estrogen replacement therapy reduces symptoms of depression and anxiety in non-depressive menopausal women (Sharma et al.2023; Liu et al., 2024). A clinical sample of postmenopausal women revealed the prevalence of mood and anxiety disorders and their related factors. Among menstruating women with panic disorder, symptom fluctuations are markedly linked to menstrual timing. The estrous cycle and ovarian hormones influence behavioral indices of anxiety in female rats (Green & Graham, 2022). Estrogen receptor GPR30 exerts anxiolytic effects by maintaining the balance between GABAergic and glutamatergic transmission in the basolateral amygdala of ovariectomized mice after stress (Stanikova et al., 2019). Women’s levels of the estrogen metabolites estrone conjugates and estradiol correlate inversely with overall body mass

index. Performance on tests of psychomotor speed, set-shifting, and visual working memory increases commensurately with concentrations of the androgens testosterone and free testosterone (G. Cameron & M. Nesse, 1988).

### 4.2. Androgens and Their Effects

Androgens have a variety of possible anxiolytic and anxiogenic effects. They intensely modify the regulation of glucocorticoid secretion, which frequently alters anxiety signaling (G. Cameron & M. Nesse, 1988). Although the predominant androgenic steroid is testosterone (T), other androgens such as dehydroepiandrosterone (DHEA) and 5α-androstane-3β,17β-diol (3β-diol) are typically involved in anxiety modulation (Sheng et al.2021). DHEA has been deemed anxiolytic across both humans and rodents, and substantially increases exploratory behavior; the anxiolytic effects appear to be dependent on the dose of administration and age of the subject (Nenezic et al.2023). Synthetic androgens such as 7α-methyl-19-nortestosterone generate activity on both androgen receptors and progestin receptors, which has been suggested by some researchers to be a therapeutic target with the capacity to alleviate anxiety (Malik & Nadeem, 2023). The activity of T itself may be broadly split, as systemic T reduces anxiety and local aromatization of T to estradiol in the medial preoptic area increases anxiety (Pillerová et al.2022).

### 4.3. Thyroid Hormones and Mood Disorders

The neuroendocrine system coordinates the interaction of the brain with the peripheral endocrine glands. Relevant to anxiety disorders is the hypothalamic-pituitary-thyroid (HPT) axis. The HPT axis is activated by cold exposure or stress. Thyrotropin-releasing factor (TRF) is secreted from the hypothalamus into the hypophyseal portal vein. This stimulates the release of thyroid-stimulating hormone (TSH) from the anterior pituitary, which in turn stimulates the secretion of thyroid hormones from the thyroid gland. The thyroid produces thyroxine (T4) and triiodothyronine (T3), of which the latter is more biologically active (Castillo-Campos et al.2021; Vella & Hollenberg, 2021).

Dysfunction of the thyroid gland has been associated with anxiety. Both hypo- and hyperthyroidism may result in anxiety presentation in a nonpsychiatric clinical setting. Conversely, screening of subjects with primary anxiety or depressive disorders may reveal abnormalities of the HPT axis as well as elevated antithyroid antibodies (Fischer & Ehlert, 2018).

## 5. Gender Differences in Hormonal Impact

Increased hormone levels, as well as anxiety indicators, make adolescence a vulnerable developmental stage for anxiety disorders (Z. Farhane-Medina et al., 2022). The imbalance between the action of these hormones through activation of the hypothalamic-pituitary-adrenal (HPA) axis further complicates the vulnerability framework for adolescents previously burdened with a psychopathological profile (Mastorci et al., 2024). In the referenced work, the authors found that higher levels of anxiety among girls were mainly due to genetic factors, with heritability explaining 74% of the variance in anxiety in girls compared to 65% in boys (Martin et al.2021). The greater prevalence of internalizing symptoms in women may be attributed to sex-related biological vulnerability and differential environmental exposures (Farhane-Medina et al.2022). Additionally, girls' propensity to ruminate and catastrophize amplifies their vulnerability to anxiety disorders (Conroy, 2021). The pattern of comorbidity between anxiety and depression also differs by sex, being more prevalent in women. Women exhibited an increased occurrence of anxiety-depressive symptoms (Farhane-Medina et al.2022).

### 5.1. Males vs. Females

Diagnoses of generalized anxiety disorders and panic disorders grow steadily from childhood

through the adolescent and young adult years. As the transition from adolescence into young adulthood occurs, the incidence of GAD, PD, and other anxiety disorders is greater for females than males (Z. Farhane-Medina et al., 2022). Moreover, previous research identifies biological sex as a major risk factor for the development of anxiety and depression complications. Girls show higher levels of anxiety, mainly due to genetic factors, with heritability explaining 74% of variance in girls and 65% in boys (Farhane-Medina et al.2022). The higher prevalence of internalizing symptoms in females can be attributed to sex-related biological vulnerability and environmental exposure. Rumination and catastrophizing increase girls' vulnerability to anxiety disorders, whereas supportive marital relationships and quality social networks reported by males can help combat loneliness. The greater comorbidity of anxiety and depression, as well as the amplified clinical manifestation of these disorders, highlights the necessity of a symptom-based approach to understanding these disorders in females (Van et al.2023).

### 5.2. Cultural and Social Influences

The cultural and social environments play an important role in the development of generalized anxiety and panic disorders. The bidirectional interactions among individual, social, and familial factors contribute to the emergence of psychological disorders (Lin & Guo, 2024). Parental psychological control and child maltreatment act as substantial risk factors for peer victimisation by impairing self-cognition, including self-efficacy and self-esteem (Li et al., 2023). Moreover, the rapid advancement of technology has led to increased concerns about social media use and its association with psychological distress, suicidal ideation, depression, and anxiety (Zubair et al., 2023). Different patterns of social media consumption may exert varying impacts on adolescent mental health, underscoring the necessity to identify effective family- and school-based interventions (Throuvala et al.2021). Furthermore, romantic relationships, and specifically experiences with online dating, potentially exert adverse effects on mental well-being, representing a further avenue for research (Gerlach, 2021). Stressful life events, such as parental divorce, precipitate acute stress reactions and serve as predictors of anxiety and depression symptoms. Sexual differentiation constitutes an important aspect of adolescent mental-health trajectories; women generally exhibit higher rates of mood and anxiety disorders, whereas men display greater vulnerability to substance-use and antisocial disorders (Farhane-Medina et al.2022). Moreover, gender moderates the effect of anxiety and depression, with males demonstrating more pronounced decrements in self-esteem, academic performance, psychosocial functioning, and subjective well-being; identical risk factors may also elicit divergent symptomatology across sexes (Ranney et al., 2021).

### 6. Psychosocial Factors

Coping mechanisms are an important psychosocial factor in generalized anxiety disorder (GAD). Adolescents tend to use lower levels of approach coping and social support seeking (the ability to seek guidance, information, or reassurance from others) than adults, the use of which was highly predictive of trait anxiety in this population (Z. Farhane-Medina et al., 2022).

Social connections also influence anxiety risk. The quality of social networks acts as a protective factor against loneliness in women with GAD, whereas men with the condition report having more supportive marital relationships (Lin & Guo, 2024). Anxiety also co-occurs with social deficits, which can lead to loneliness and social isolation.

Environmental triggers include social media exposure to potentially anxiety-inducing content and traumatic or stressful life events such as

parental divorce, peer victimization, bullying, and academic pressure. On the other hand, peer support, a positive school climate, and parental emotional support can reduce anxiety symptom severity, with the latter being shown to decrease gene expression related to stress and anxiety disorders.

### 6.1. Environmental Stressors

Environmental stressors trigger acute fight-or-flight responses that prepare the organism to face imminent danger (E. Tafet & B. Nemeroff, 2020). These mechanisms are vital for survival and are conserved across vertebrate species. After the threat subsides, a return to homeostasis is necessary to prevent pathological changes; failure to do so can contribute to the development of stress-related disorders. The hypothalamic-pituitary-adrenal (HPA) axis, comprising the hypothalamus, pituitary, and adrenal glands, is a key mediator of the stress response and a target of several anxiolytic drugs. Hyperactivity of the HPA axis, as observed in some patients with anxiety and mood disorders, results in increased corticosterone and cortisol secretion, which affects the expression of many genes (G. Cameron & M. Nesse, 1988). For example, the glucocorticoid receptor gene (Nr3c1) possesses glucocorticoid response elements in its promoter region and is therefore susceptible to autoregulatory control by increased glucocorticoids (Jaric et al., 2019). Adrenergic, noradrenergic, gamma-aminobutyric acid-ergic, serotoninergic, and dopaminergic pathways display altered activity in most anxiety disorders, highlighting multifaceted neurochemical dysregulations. Genetic, epigenetic, and physiological changes induced by the HPA axis may mediate long-lasting behavioral effects of stress. Benzodiazepines reduce the activity of corticotropin-releasing factor (CRF) neurons in the hypothalamus, while tricyclic antidepressants (TCAs) and selective serotonin reuptake inhibitors (SSRIs) influence different aspects of HPA axis regulation. For instance, the SSRI escitalopram inhibits CRF release in the amygdala and increases glucocorticoid receptor density in the hippocampus and hypothalamus.

### 6.2. Coping Mechanisms

Anxiety disorders represent the most common psychiatric issues in children, hindering academic and social functioning and causing impairments in school performance and family interactions. Within this context, children may develop coping strategies to minimize or overcome experienced stress.

Psychological coping mechanisms constitute conscious efforts that an individual employs to address external or internal challenges to reduce or tolerate anxiety. Stressful situations in daily life can lead to the activation of a psychological set of coping strategies.

Research involving 461 students from Greece has identified several factors influencing anxiety levels and coping strategies. Gender has a significant relationship with both anxiety levels and coping strategies, as does maternal education. Age is related to anxiety disorders, with a higher prevalence of anxiety disorders observed in girls (Antoniou & Karteris, 2017).

## 7. Treatment Approaches

A multimodal treatment approach, including medication, therapy, and environmental interventions, has shown greater improvement in symptoms than unimodal treatments (Badcock et al.2021). Cognitive-behavioral therapy (CBT) is effective for various anxiety disorders, including generalized anxiety disorder, social anxiety, and panic disorder, and benefits are maintained over time (Butler et al., 2021). For youth with mild to moderate impairment, psychoeducation and CBT are recommended across anxiety disorders, with medication deferred initially. For moderate to severe anxiety, combined treatment with medication and CBT is advised for generalized anxiety disorder and social anxiety, with selective serotonin reuptake inhibitors (SSRIs) supported by

multiple trials and recommended as first-line agents (Haugan et al.2022). Medication may start concurrently with therapy or before, and can be added if psychotherapy alone is insufficient. Selection of pharmacologic agents follows evidence and guidelines, considering side effects and individual needs, with informed consent required (Trautmann et al.2023). Frequent follow-up visits are recommended to monitor effectiveness and tolerance, especially when there is a family history of suicide, previous suicide attempts, or comorbid depression. Standardized scales should be used to assess progress. After symptom resolution, medication is typically maintained for one year before gradual tapering, to prevent recurrence (Kodish et al., 2011).

### 7.1. Pharmacological Treatments

Despite the high prevalence of anxiety disorders and the amount of research invested in their treatment, relatively few novel medications have been investigated (Garakani et al., 2020). Selective serotonin reuptake inhibitors (SSRIs) are the first-line agents for treating pediatric anxiety disorders, with evidence suggesting that a switch to an alternative SSRI improves outcomes after an unsuccessful initial trial (Kodish et al., 2011). Residual symptoms often remain when SSRIs are used without psychotherapy, most commonly cognitive behavioral therapy (CBT) (Whiston et al.2022). Tricyclic antidepressants and benzodiazepines also exhibit anti-anxiety effects, but the risks associated with these medications, such as elevated side effects or dependence, make them a less attractive option for the pediatric population (Deb et al.2021).

### 7.2. Psychotherapy Options

Psychotherapy offers multiple options for the treatment of anxiety disorders. One treatment option to consider is cognitive behavioral therapy (CBT) (Newman et al.2022). CBT has not only been shown to be effective in the treatment of generalized anxiety disorder (GAD) in children and adolescents, but also for social anxiety, panic disorder, obsessive compulsive disorder (OCD), and post-traumatic stress disorder (PTSD) (Guo et al.2021). Moreover, clinicians and families alike seek to utilize the least intrusive therapies before considering medication. Therefore, for youth presenting with mild to moderate impairments, psychoeducation and CBT are the first recommended treatments (Oliveira & Dias, 2023). For moderate to severe GAD, a combined treatment approach pairing medication and a form of therapy such as CBT is preferable. Psychotherapy and environmental interventions can be integrated into this approach for enhanced effectiveness (Kodish et al., 2011).

### 7.3. Lifestyle Modifications

Broad-spectrum therapies such as benzodiazepines have been the treatment mainstay for anxiety disorders (Löscher & Klein, 2021). Because of the problems of tolerance, dependence, withdrawal phenomena, and abuse that these agents can create, the development of new effective pharmacological and nonpharmacological treatments is critically important (G. Cameron & M. Nesse, 1988). A growing number of studies reviewed here provide support for the use of complementary and alternative medicines (CAM), as well as for lifestyle modifications, in the treatment of anxiety (Ng & Jain, 2022). A range of CAM therapies appear promising, including kava, for which there is moderately strong support, and acupuncture, yoga, and Tai chi, though methodological problems inhibit firm conclusions; homeopathy lacks support (Sarris et al., 2012). Interactions between biological factors and lifestyle behaviors, such as diet and physical activity, also play important roles in anxiety disorders. Dietary improvements have preliminary support for anxiolytic effects, and avoidance of caffeine, alcohol, and nicotine also shows encouraging evidence (Aucoin et al.2021). Several potential lifestyle contributors to anxiety-

induced sleep disturbances are highlighted by recent research (Francisco López-Gil et al., 2022).

## 8. Conclusion

The extensive variability between the physiological and behavioral markers of anxiety makes it unlikely that generalized anxiety disorder and panic disorder, or other aspects of anxiety, represent the disruption of a single physiological system. Both have a lifetime prevalence in the general population of about 5%. Many of the major physiological systems are affected, as well as a variety of neurotransmitters and brain systems. For example, serotonergic drugs are effective in both disorders, but animal models suggest that serotonin generally inhibits the fight or flight response that accompanies anxiety. Dopaminergic and noradrenergic systems appear to be activated, which normally should increase the response. Ghrelin and neuropeptides also seem to play a substantial role and perhaps may help explain the increases in appetite often found in generalized anxiety disorder. In addition, physiological markers of stress regulation, glucose regulation, hypothalamic–pituitary–adrenal (HPA) function, thyroid function, and prolactin are all affected. While some generalizations can be made (such as reduced glucose response to challenge, more abnormal thyroid secretion with generalized anxiety, and increased ghrelin and other neuropeptides and reduced prolactin with panic), no clear physiological picture emerges without factoring in such variables as sensitization, whether the patient has experienced attacks, and the phase of the cycle or pregnancy. Similarly, a neuroanatomical or neurochemical dissection cannot easily explain all the data. A better framework or new ideas, such as the sensitivity of interoceptive processes, may be needed to move to a better understanding and broader model of the maintenance of anxiety disorders.

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