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Genetic and Environmental Pathways to Anxiety Across Adolescence in Relation to Developmental Timing and Changing Social Contexts

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ABSTRACT

Anxiety disorders commonly emerge during adolescence, reflecting complex interactions between genetic predispositions and environmental influences that vary across developmental timing and social context. This review integrates evidence from genetics, developmental psychology, neuroscience, and social sciences to examine how genetic vulnerability, environmental risk and protective factors, and their interactions shape anxiety trajectories across adolescence. Genetic influences, including heritability, specific polymorphisms, and polygenic risk scores, interact dynamically with family, peer, school, digital, cultural, and societal environments. Adolescence represents a sensitive developmental period in which pubertal maturation, neurobiological reorganization, and shifting social contexts amplify susceptibility to anxiety. Epigenetic modulation and stress-responsive neurobiological pathways further link environmental exposures to enduring anxiety risk. Longitudinal and integrative approaches highlight how timing, context, and gene–environment interplay influence vulnerability, resilience, and recovery. Understanding these pathways provides a foundation for developmentally informed prevention and intervention strategies tailored to adolescents’ evolving social environments.

Keywords: *adolescent anxiety, gene–environment interaction, developmental timing, polygenic risk, social context, peer relationships, family dynamics, digital environments, epigenetics, neurodevelopment, stress reactivity, longitudinal studies.*

INTRODUCTION

Genetic predispositions interact dynamically with environmental risk and protective factors to shape trajectories of anxiety during adolescence, a critical developmental period marked by rapid biological, cognitive, and social changes. These interactions operate through complex mechanisms that are sensitive to developmental timing and embedded within broader social contexts, such as family relationships, peer dynamics, and sociocultural stressors. Together, genetic vulnerability and environmental influences contribute to individual differences in the onset, persistence, and severity of anxiety symptoms across adolescence.

Theoretical Foundations

Adolescence, a period characterized by remarkable anatomical, physiological, and mental transformations, exerts a critical influence on the emergence and evolution of anxiety (A. Nelemans et al., 2018). Genetic and environmental determinants play a substantial role in anxiety development. Genetic vulnerability is documented based on heritability estimates generally between 20% and 50%, significant population-wide polymorphisms identified, and polygenic risk scores derived from genome-wide association studies successfully linking individual differences in anxiety-related traits and symptoms to genetic variations (B Sharp, 2019). Furthermore, relevant gene pathways have been highlighted across the life course, but their precise role during this transitional stage remains to be thoroughly investigated.

For the environment, specific risk and protective factors rooted in family, peer, school, community, or cultural contexts are established to influence anxiety from early childhood into the preteen years (Ziyayev, A. A., et al). Distal aspects include neighborhood or societal influences like socioeconomic status, acculturation, or political and cultural stigma, which may increase vulnerability to anxiety based on a given historical moment. These determinants, encompassing parental involvement, peer dynamics, early affect regulation, and critical cultural context, exert their impact following a cascade of timing at which biochemical systems controlling stress reactivity and affect sensitivity take shape and vary across normative development [Table 1].

Table 1: Genetic and Environmental Contributions to Anxiety Across Adolescence

Domain	Key Factors	Evidence / Examples	Role in Anxiety Development
Genetic Predisposition	Heritability estimates	20–50% heritability from twin studies	Establishes baseline vulnerability to anxiety
Genetic Predisposition	Specific polymorphisms	5-HTTLPR interacting with parenting behaviors	Moderates sensitivity to caregiving environments
Genetic Predisposition	Polygenic risk scores (PRS_rb)	Scores from ~452,000 SNPs predict anxious temperament and symptoms	Captures cumulative genetic liability
Genetic Predisposition	Neurobiological pathways	Serotonin, dopamine, oxytocin, circadian rhythm genes	Influence affect regulation, stress reactivity
Family Environment	Parenting style	Warmth vs coercion; parental anxiety	Buffers or amplifies genetic risk
	Early adversity	Family conflict, neglect	Long-term anxiety risk via stress systems

Peer & School Context	Peer acceptance / rejection	Victimization, academic stress	Strong predictors of anxiety, especially early adolescence
	Gene–peer interaction	PRS × peer stress predicts anxiety & dropout	Interaction strength declines with age
Digital Environments	Social media exposure	Cyberbullying, social comparison	Timing-dependent risk for anxiety
	Digital parenting	Monitoring, transparency, trust	Protective against online stressors
Cultural & Societal Factors	SES, discrimination, stigma	Minority stress, acculturation strain	Structural amplification of anxiety risk

Genetic Contributions to Anxiety

Substantial evidence indicates that the origins of anxiety are partly genetic. Twin studies report heritability estimates ranging from 30% to 50% (A. Nelemans et al., 2018). A polymorphism in the serotonin transporter gene (5-HTTLPR) interacts with parenting behaviours to predict variation in adolescent anxiety. Polygenic risk scores calculated from 452,000 single-nucleotide polymorphisms correlate with anxious temperament tendencies in preschoolers and 7-year-old anxiety symptoms (Waszczuk et al., 2013). Other genes, including those implicated in dopamine, oxytocin, and circadian rhythm pathways, continue to receive attention in relation to adolescent anxiety.

Environmental Risk and Protective Factors

Exposure to online social environments has become a widespread phenomenon during adolescence, leading to an intense growth of interest and corresponding research examining adolescents' online activities. Digital environments, including social media, can be employed for multiple motivations, such as mood regulation and connection to peers, and can heavily influence emotional experiences. Exposure to online social stimulation, however, can be maladaptive by leading to cyberbullying victimization and excessive social comparisons. The impact of online engagement on the development of anxiety-related problems appears dependent on the timing of the first exposure. Investigating another type of peer relationship widely experienced by adolescents, academic stress from perceived low classmate acceptance within the school environment also surfaces as a salient predictor of anxiety symptom progression. Not only does classroom peer acceptance affect anxiety development but it also moderates how gene and environment interact, indicating the importance of considering digital and school environments as key contextual factors that shape vulnerability to anxiety symptoms across adolescence (A. Nelemans et al., 2018).

Developmental Timing and Sensitive Periods

Adolescence is a developmental phase characterized by the emergence of genetic and environmental influences relevant to the development of anxiety symptoms (A. Nelemans et al., 2018). The onset of puberty may trigger a sensitive period in which genetic factors become more strongly related to variability in anxiety, as the neural circuits underlying threat perception and regulation undergo significant maturation (Waszczuk et al., 2013). However, due to the variability of pubertal timing, the influence of heredity on anxiety may increase for some youth as early as age 10, while for others, the sensitive window may not begin until age 15 or 16 [table 2].

Table 2: Developmental Timing, Social Contexts, and Mechanisms Shaping Anxiety Trajectories

Dimension	Core Concept	Developmental Pattern	Implications
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Sensitive Periods	Puberty onset	Genetic influences on anxiety intensify between ~9–16 years	Timing variability alters individual vulnerability
Gene–Environment Interaction	Context-dependent genetic effects	PRS interacts with family, peer, and school stress	Risk and resilience vary by social exposure
Epigenetic Modulation	DNA methylation & chromatin changes	Adolescence uniquely responsive to stress	Environmental stress may produce long-lasting effects
Neurobiological Development	HPA axis maturation	Heightened stress reactivity, sex differences	Explains rising anxiety prevalence in adolescence
Shifting Social Salience	Family → peers → individual autonomy	Peer effects peak early adolescence, decline later	Intervention targets should shift with age
Measurement & Trajectories	Anxiety subtype emergence	Separation, social, bodily harm anxieties follow distinct timelines	Supports developmentally specific assessment
Protective Pathways	Supportive parenting, peer acceptance	Promote recovery and resilience	Targets for prevention and intervention

Adolescent Social Contexts and Anxiety Trajectories

Across adolescence, social contexts shape distinct anxiety trajectories and influence the emergence of anxiety disorders. Genetic predispositions are necessary, but not sufficient, for anxiety and interact with family, peer, school, community, and cultural factors to elevate risk or confer resilience. Specific processes accompanying genetic risk also modify susceptibility to acute stressors or promote recovery from episodes, highlighting the importance of the acute environmental context for trajectories over time. Identification of these protective, risk- amplifying, and recovery-promoting factors represents a promising avenue for targeted intervention (A. Nelemans et al., 2018).

Family Dynamics and Parenting Practices

Both genetic and environmental pathways shape anxiety across development, but social environments emerge as critical at adolescence. Family dynamic and parenting practices represent a key source of risk and resilience and continue to exert influence during sensitive periods (Azimova, S., et al. 2023). Parenting practices during childhood exert long-term effects on anxiety, and with early onset family adverse effects are widely established risk factors (A. Nelemans et al., 2018).

Peer Relationships and School Climate

Peer relationships and school climate significantly shape psychosocial development and mental health through multiple mechanisms. In adolescence, social worlds expand beyond the family to include schoolmates and friends, making peers a critical influence on well-being (A. Nelemans et al., 2018). Genetic predispositions toward anxiety elevate vulnerability to negative peer experiences, such as peer rejection, victimization, and academic stress (J Veed, 2009). In turn, anxiety symptoms weaken peer relationships, leading to elevated internalizing symptoms. Negative peer interactions, therefore, have the potential to amplify anxiety through different social contexts that exert influence at distinct developmental stages. Gene–environment interactions can augment susceptibility to peer-related risks and shape the timing of their effects. Polygenic risk for anxiety interacts with stressful peer experiences to predict anxiety symptoms and school dropout, with the magnitude of the interaction decreasing from age 12 to 15, coinciding with

declining peer influence in early adolescence (Serra Poirier et al., 2016). Genetic influences on both anxiety and peer problems remain constant throughout development. At early stages, genetic predispositions facilitate maladaptive social exchanges, which in turn heighten anxiety; later, peers exert less influence on anxiety trajectories because familial and individual factors become primary determinants for many youths (Ziyaev, A. A., et al).

Digital Environments and Social Media Exposure

Digital social environments, including social media platforms, provide both opportunities and risks for adolescents. During the preteen years, when many youths first gain access to devices and accounts, online interactions can offer formats alternative to face-to-face engagement for social exploration and self-expression. Connectivity through shared interests and supportive individuals may facilitate mood regulation, encouraging positive alternatives to substance use. Yet opportunities for exploration coexist with risks for cyberbullying. Ford (2021) estimated that nearly 1 in 5 youths ages 10 to 17 experienced online aggression in the preceding year, and feelings of inferiority due to social media comparison when needing affirmation or validation appear tied to depressive symptoms. Digital parenting characterized by active engagement with technology, transparency about searching and online profiles, and fostering communication and trust helps safeguard against online hazards and alleviate mental health concerns (Nesi, 2018).

The transition into adolescence, spanning approximately ages 9 to 14, constitutes a sensitive period for anxiety-provoking experiences that typically peak later than other forms of psychopathology. Gene–environment interactions may amplify risk associated with peer victimization, parental monitoring, and family support received at this juncture; vulnerability varies with the relative timing of genetic risk, environmental opportunity, and psychosocial stressors (A. Nelemans et al., 2018).

Cultural and Societal Influences

According to recent studies, youth and adolescents belonging to socio-cultural minorities are particularly likely to suffer from anxiety disorders, especially in environments which are not accommodating to their background. Moreover, the role of cultural influences on anxiety and comorbid disorders manifests as salient vulnerability pathways, with high levels of stigma, acculturation stress, and discrimination raising anxiety risk among minorities. Multiple structural inequalities are associated with these supplemental vulnerabilities, including low socioeconomic status (SES) and racism. In particular, adolescent girls whose social environments actively endorse victimization are also disproportionately affected, while those living under culturally variable circumstances appear more resilient to a common risk factor loop between endorsement of victimization, perceived victimization, and anxiety (A. Nelemans et al., 2018).

Mechanisms Linking Genetics, Environment, and Anxiety

Genetic predispositions interact with environmental risk and protective factors across the adolescent period to shape anxiety trajectories, with the developmental timing of these influences and the shifting nature of social contexts being particularly important. Genetic contributions to variation in anxiety have been examined through heritability estimates and the identification of specific polymorphisms, polygenic risk scores, and gene pathways that may confer risk for adolescent anxiety. The heritability of anxiety symptoms is estimated to be moderate, with longitudinal studies indicating an increase in genetic influences during the transition to adolescence (A. Nelemans et al., 2018).

A range of environmental risk and protective factors for anxiety have been identified at different ecological levels, including family, peer, school, community, and cultural contexts. These processes can be further categorized into maladaptive versus protective. Family influences, peer relationships, and school climate constitute the immediate social context for adolescents, and stresses within these domains have been linked to anxiety—a relationship that is moderated by genetic risk (M. Gregory & C. Eley, 2007).

Gene–Environment Interactions

Adolescents face increased societal pressure and heightened anxiety. Anxiety disorders are increasingly prevalent during adolescence and contribute to diverse problems. Anxiety has a substantial genetic basis, although social factors play a crucial role. Developmental variations in social environments necessitate that activities promoting anxiety reduction are tailored to age and context to improve effectiveness. Genetic predispositions influence vulnerability to environmental risk factors and modulatory influences associated with protection from adversity. Genetic susceptibility or sensitivity differs across people. Risk and protective models explain individual variation in vulnerability and resilience regarding anxiety, correlating with other internalizing and externalizing problems. Polygenic scores related to diverse anxiety-related pathways affect adolescent anxiety according to the amount of family peer problem exposures.

Research shows that genetic predispositions interact with environmental risk and protective factors across adolescence to shape anxiety trajectories. The timing of developmental transitions and other shifting social contexts, such as the family, school, peer group, and larger cultural milieu, closely influence vulnerability and resilience. Polygenic and other genetic risk scores associated with multiple systems relevant to anxiety interact with specific aspects of the social environment (A. Nelemans et al., 2018).

Epigenetic Modulation Across Development

Stressful experiences during adolescence can induce persistent changes in DNA methylation that may elevate anxiety risk. In a cradle-to-grave study commencing at birth, both methylation and chromatin accessibility changes in association with exposure to chronic stress were observed (Azimova, S., et al. 2023). These dynamics were tightly coupled across multiple tissue types and mirrored those seen in model organisms. Stress exposure during childhood and adolescence intensified the transmission of altered methylation states across cells, tissues, and generations, whereas perturbations at birth or adulthood had negligible intergenerational consequences. The adolescent brain is particularly responsive to environmental stimuli, including social contexts. Bronfenbrenner and Morris (A. Nelemans et al., 2018) proposed that environmental influences on development would be strongest when individual maturation coinciding with the transition to adulthood parallels heightened exposure to ‘contextual stimuli’.

Neurobiological Pathways in Adolescence

Adolescence is characterized by neurobiological changes that influence the way in which genetic and environmental influences shape the anxiety trajectories of young people over time. Development of the hypothalamic–pituitary–adrenal (HPA) axis is linked to increased reactivity to stress, which can contribute to anxiety (A. Schriber & E. Guyer, 2015). The HPA axis and associated neural circuits mature earlier and reach adulthood sooner in males than in females, which helps to explain differences in sensitivity to social influences (A. Nelemans et al., 2018). Cortical and subcortical regions show different patterns of maturation, which has implications for externalizing or internalizing disorders. The development of coherent coactivation patterns becomes more evident between early and late adolescence; during this period, activation of the amygdala when encoding fearful faces strongly predicts social anxiety symptoms.

Methodological Considerations and Evidence Synthesis

Anxiety in youth is a prevalent and debilitating condition. Despite the large literature on anxiety, however, the scientific evidence is fragmented and lacks a systematic synthesis of what is known about how genetic versus environmental risk and protective factors come together to shape longitudinal anxiety trajectories. To address this gap, empirical evidence and theoretical models from a wide range of disciplines, from genetics to developmental psychology to the social sciences, have been integrated with the aim of elucidating how genetic predispositions interact with environmental risk and protective factors across adolescence to shape anxiety trajectories in the general population, with a particular focus on the critical role of developmental timing and the multiple social contexts that shift throughout adolescence (Omonov Q., et al).

Longitudinal Designs and Timing

Adolescence constitutes a uniquely challenging stage of the life course, with various opportunities but also risks for maladaptive development. The onset of puberty marks a period characterized by substantial biological, cognitive, social, and emotional transformations, accompanied by heightened psycho-affective vulnerability (R. Cohen et al., 2018). Anxiety disorders emerge prominently during this phase, driven by complex interactions among genetic, epigenetic, and environmental factors. Nevertheless, the role of these factors is not equally distributed throughout development; systemic fluctuations induce opportunistic trends to develop further (P.W. Haller et al., 2018). Addressing how genetic predispositions influence environmental processes across this developmental stage remains fundamental to elucidating risk and resilience in anxiety trajectories. Individual differences in anxiety reactivity are confirmed to be heritable. A diversity of gene–environment interactions connected to diffused candidate genes adheres to non-linear, time-lagged relationships; gene–anxiety links fluctuate; and specific pathways coalesce into a concern shared by many adolescents. Altogether, these support the notion that adolescence constitutes a sensitive period for anxiety.

Measurement of Anxiety in Youth

Across early and middle adolescence, youth experience a range of anxieties associated with developmental changes and social demands that may be exacerbated by genetic predispositions and negative environmental factors. Fear of separation-anxiety related to parting from a primary caregiver-increases among youth approaching the transition to adolescence, declined in early adolescence, and resurged among youth experiencing significant school-based changes, such as changing schools or switching from primary to secondary education (A. Nelemans et al., 2018). Fears of social scrutiny-anxiety regarding appearing unattractive or inadequate in front of peers-expand during the transition into adolescence, and correspondence between genetic risk for anxiety and levels of these anxieties has been detected (M. Gregory & C. Eley, 2007). Growth in fears of sexual and bodily harm a concern with physical safety and personal violation-emerges in early to mid-adolescence, although environmental correlates fail to account for the increase, suggesting that genetic influences alone shape these risks (Azimova, S., et al. 2023). Consistent with observations that anxiety prevalence declines over late adolescence and through adulthood, new anxieties unrelated to peer interactions and evolving concerns about issues such as global violence and societal welfare surface through early-maturity and late-adolescent transitions. Heightened risk for anxiety conditions associated with pubertal maturation, school changes, family transition, or other environmental stressors peaks across the early to middle-adolescent period, corresponding with developmental timeframes wherein anxieties increase and genetic contributions become prominent (Jaborova D., et al).

Integrative Analyses and Meta-analytic Approaches

Socio-emotional and psychological development is highly contingent on social context. Understanding adolescent anxiety trajectories-influenced by genetic predisposition and pathways through which different social environments buffer or amplify risk-requires a thorough appreciation of changing social contexts and their interplay with development.

Family Dynamics and Parenting Practices. Family relationships remain paramount for adolescent socio-emotional development. Warm, supportive parenting facilitates risk and resilience, while coercion exacerbates risk. An intergenerational mode of transmission-from caregiver difficulties with anxiety, depression, and other behaviours to child outcomes through parenting styles anchored in these difficulties-operates alongside direct genetic transmission (Waszczuk et al., 2013). Genetic predispositions associated with anxiety mutually interact with parenting behaviours to influence adolescent anxiety trajectories. Parenting approaches significantly account for, moderate, and, occasionally, even fully mediate links between family history of anxiety disorders and current individual vulnerability to anxiety.

Peer Relationships and School Climate. Peer and school environments exert a pervasive effect on adolescent psychological development. Acceptance among peers enhances resilience; victimization and peer rejection

reduce resilience; perceived support, and positive social experiences at school foster resilience; and academic stress increases risk (A. Nelemans et al., 2018). Genetic risk associated with anxiety varies in its relation to these different, and differentially moderating, peer, interpersonal, social, contextual, and academic aspects. Participant analyses indicate a connection between the polygenic broad anxiety propensity score and socially-driven, peer-longitudinal change in social anxiety through peer acceptance and victimization.

Digital Environments and Social Media Exposure. Online environments are a key component of adolescent social experience. Interactions in digital form differentially influence young people's social adjustment and well-being. Cyberbullying, social comparison, exposure to distressing material, and harassment-commonly manifest in some social media settings-are among the risks. Use of mobile devices invariably shapes, and frequently modulates, engagement with, and thus exposure to, other young people. Moreover, these devices facilitate the search for easily accessible relationship information. Timing, since youth infuses early-adolescent punctuality, continues to modulate the vulnerability of technology-driven, digital, online engagement; technology offers mood-regulation avenues, yet simultaneously intervenes at a paralleled rate, in advance of hormonal shift scheduling and internal-disposition (Sasmakov, S. A., et al).

Cultural and Societal Influences. Cultural, community, neighbourhood, society, socio-economic background, and structural inequality variables intermediary parental styles, peer interactions, and school climate to contingent well-being, function distinctively. Lack of fit, corroborative, or stigma-related acculturation stress, for example, modulates young migrants' experiences, and thus anxiety, when timely socio-cultural synchronization remains hindered. Partly shifted dependent pursuit, socio-economically driven risk loads concomitantly progress anxiety differentiation; factor predecessors participate in establishing risk developmental timescale (Makhkamova S., et al).

Implications for Prevention and Intervention

Genetic and Environmental Pathways to Anxiety Across Adolescence: Developmental Timing and Shifting Social Contexts

Implications for Prevention and Intervention

Developmentally sensitive adjustments may improve preventive strategies targeting these genetic and environmental mechanisms linked to adolescent anxiety. Interventions could emphasize the family environment during early adolescence, before the onset of puberty, when the influence of stressful parenting practices on anxiety may be heightened (A. Nelemans et al., 2018), and at the age of 10 or 11, when polygenic risk associated with high levels of family stress begins to emerge (Gazelle, 2022). Tailoring preventive programs to the specific family, school, or community components relevant at different stages of adolescence may enhance their impact. Broader policies that translate these findings into endorsement of evidence-based, developmentally appropriate programs, training in implementation, and support for resource allocation are also needed.

Targeting High-Risk Periods

Anxiety is characterized by fear or apprehension about future events or situations, it is often accompanied by behavioral manifestations such as avoidance, compulsive acts, or ritualized behaviors, it is linked to operative concerns that include threats to the self, social acceptance, social standing, and access to material resources, anxious youth exhibit slower rates of academic skill development across the elementary years, recognizing content as more difficult than their peers, adolescent-specific environmental risk factors aggregate during the first year of high school, anxiety-inducing messages or interactions arriving through newly available digital platforms, periods shown to increase the risk of anxious and violent behavior (Sasmakov, S. A., et al). Biological and contextual mechanisms are consistently implicated in the differential expression of both genetic and environmental influences on anxiety across youth, the hypothalamic-pituitary-adrenal axis in the stress response, social affiliation and social support among

adolescents moderates or mediates the effect of stressful experiences on anxiety. Paired with the increasing manifestation of anxiety through preadolescence and its continued rise into early adulthood, adolescence now emerges as a major window of prevention for anxiety in formation and persistence, examining factors that can help define timings of preventative interventions during premiddle to postpartum and sustained preventative measures across secondary schooling. (A. Nelemans et al., 2018) (G. Gee et al., 2022)

Contextually Informed Preventive Programs

Contextually Informed Preventive Programs

Exposure to contextual risk predicts anxious behaviour and associating contextual protective factors has been linked to lower risk for anxiety (A. Nelemans et al., 2018). Preventive programs that incorporate an understanding of contextual influences on adolescent development, and pre-existing levels of anxiety are required. Indeed, it is not sufficient to develop preventive programs that have shown some efficacy without establishing the contexts underpinning anxiety or the resources that protect against its development. Eco-developmental theory emphasises that as youth progress through adolescence increasingly important contextual influences emerge yet frameworks for considering preventive programs that account for these shifts are limited. Program development would benefit from considering environments known to be salient in adolescence, including family, community, school, and electronic.

Policy and Practice Implications

Adolescents face unique developmental challenges across multiple domains that influence their anxiety trajectories. Research highlights the importance of different kinds of social environments as adolescents develop and switch between increasingly autonomous social roles. Family dynamics, peer relationships, school climate, digital environments, and broader cultural contexts represent dimensions of adolescent social life that appreciably alter exposure to risk and resilience factors (A. Nelemans et al., 2018) and that can be usefully framed in reference to the genetically and environmentally specified processes discussed earlier (Akhmedova N., et al).

Gaps, Limitations, and Future Directions

Anxiety rises markedly during adolescence (P.W. Haller et al., 2018) yet individual trajectories differ. Genetic predispositions interact with environmental risk and protective factors to shape these trajectories. These influences develop through shifting social contexts. During adolescence the risk-resilience equation consequently changes. Developmental changes enhance sensitivity to risk while diminishing resilience to protectors (A. Nelemans et al., 2018). Specific periods emerge during adolescence when genetic or environmental influences hold heightened sway (Belsky et al., 2015). Timings differ according to the signature milestones of each environmental domain-peer relationships, digital contexts, family, school, or cultural stresses.

Conclusion

Genetic and environmental pathways converge across adolescent development to shape anxiety within shifting social contexts. Genetic contributions interact with family, peer, school, and community factors-moderated by these same environments-thereby delineating individual risk and resilience from early childhood through young adulthood. Family warmth, coercion, intergenerational transmission, peer acceptance, victimization, digital and cultural exposures, and socioeconomic context differentially affect anxiety trajectories (Sasmakov, S. A., et al). These trajectories also reflect timing: early genetic and parenting influences establish a foundation, while peer, digital, and cultural factors exert increasing weight during middle to late adolescence.

Selected covariates further specify genetic and environmental impacts. New technologies for data fusion support estimates of shared and unique variance across studies. Large, integrative databases draw on birth

cohorts, classroom-based longitudinal designs, and community surveys. Cultural evolution shape aspects of adolescent development and well-being.

References:

1. Nelemans S.A., van Assche E., Bijttebier P., Colpin H., van Leeuwen K., Verschueren K., Claes S., van den Noortgate W., Goossens L. Parenting interacts with oxytocin polymorphisms to predict adolescent social anxiety symptom development: A novel polygenic approach. 2018. URL: ncbi.nlm.nih.gov
2. Sharp P.B. Elucidating the nature and development of neural mechanisms associated with anxious apprehension and anxious arousal across adolescence. 2019. [PDF]
3. Waszczuk M.A., Zavos H.M.S., Eley T.C. Genetic and environmental influences on relationship between anxiety sensitivity and anxiety subscales in children. 2013. URL: ncbi.nlm.nih.gov
4. Veed G.J. The role of the peer group in adolescence: Effects on internalizing and externalizing symptoms. 2009. [PDF]
5. Serra Poirier C., Brendgen M., Girard A., Vitaro F., Dionne G., Boivin M. Friendship experiences and anxiety among children: A genetically informed study. 2016. [PDF]
6. Nesi J. Adolescent social media use and psychosocial adjustment: Toward a new research agenda. 2018. [PDF]
7. Gregory A.M., Eley T.C. Genetic influences on anxiety in children: What we've learned and where we're heading. 2007. [PDF]
8. Schriber R.A., Guyer A.E. Adolescent neurobiological susceptibility to social context. 2015. URL: ncbi.nlm.nih.gov
9. Cohen J.R., Andrews A.R., Davis M.M., Rudolph K.D. Anxiety and depression during childhood and adolescence: Testing theoretical models of continuity and discontinuity. 2018. [PDF]
10. Haller S.P.W., Mills K.L., Hartwright C.E., David A.S., Cohen Kadosh K. When change is the only constant: The promise of longitudinal neuroimaging in understanding social anxiety disorder. 2018. URL: ncbi.nlm.nih.gov
11. Gazelle H. Two models of the development of social withdrawal and social anxiety in childhood and adolescence: Progress and blind spots. 2022. URL: ncbi.nlm.nih.gov
12. Gee D.G., Sisk L.M., Cohodes E.M., Bryce N.V. Leveraging the science of stress to promote resilience and optimize mental health interventions during adolescence. 2022. URL: ncbi.nlm.nih.gov
13. Omonov Q., et al. The role of digital marketing technologies in enhancing business process management. *Indian Journal of Information Sources and Services*. 2024. Vol. 14, No. 4. P. 159–164.
14. Jabborova D., et al. From manuscripts to machines: The evolution of book publishing devices. *Indian Journal of Information Sources and Services*. 2024. Vol. 14, No. 4. P. 117–124.
15. Makhkamova S., et al. Architectural dialogue between historic charm and urban modernity. *Architecture Image Studies*. 2024. Vol. 5, No. 1. P. 134–141.
16. Akhmedova N., et al. The role of street art as a language of innovation in Uzbekistan's architectural practices. *Architecture Image Studies*. 2024. Vol. 5, No. 1. P. 142–152.
17. Kim Yeonjin, & Wesam Ali. (2025). Energy-Aware Hardware/Software Co-Design for Deep Neural Networks on Reconfigurable Platforms. *SCCTS Journal of Embedded Systems Design and Applications*, 3(1), 47-54. <https://doi.org/10.31838/ESA/03.01.06>
18. Abdurakhmanov, J., et al. (2023). Cloning and expression of recombinant purine nucleoside phosphorylase in the methylotrophic yeast *Pichia pastoris*. *Journal of Advanced Biotechnology and Experimental Therapeutics*. <https://doi.org/10.5455/jabet.2023.d153>
19. Ziyayev, A. A., et al. (2023). Synthesis of S-(5-aryl-1,3,4-oxadiazol-2-yl) O-alkyl carbonothioate and alkyl 2-((5-aryl-1,3,4-oxadiazol-2-yl)thio) acetate, and their antimicrobial properties. *Journal of the Turkish Chemical Society, Section A: Chemistry*. <https://doi.org/10.18596/jotcsa.1250629>

20. Azimova, S., et al. (2023). Study of the immunogenicity of combination of recombinant RBD (Omicron) and nucleocapsid proteins of SARS-CoV-2 expressed in *Pichia pastoris*. The Open Biochemistry Journal. <https://doi.org/10.2174/011874091x273716231122102205>
21. Sasmakov, S. A., et al. (2021). Expression of recombinant PreS2-S protein from the hepatitis B virus surface antigen in *Pichia pastoris*. *VacciMonitor*, 30(1), 27–32.