Cognitive Neural Processes Linking Childhood Behavioral Inhibition and Psychological Disorders

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Date: 2019-12-24T00:00:00+00:00

Abstract

Behaviorally inhibited children are more susceptible to psychological disorders during middle childhood and adolescence compared to typical children, representing a population with elevated developmental risk. Such children exhibit greater withdrawal motivation, prolonged anticipatory and pre-attentive processes, diminished attentional control, heightened attentional bias toward negative information, hyperactive response monitoring processes, and slower habituation relative to non-inhibited children. These shared cognitive-neural processes constitute important pathways linking childhood behavioral inhibition to psychological disorders and can serve as predictive indicators for screening individuals at higher developmental risk. Beyond children' s inherent traits, environmental factors can also influence developmental outcomes by shaping cognitive-neural processes. Therefore, future research should examine cross-cultural differences in the cognitive-neural processes underlying the association between behavioral inhibition and psychological disorders in children, and subsequently identify protective factors for the development of behaviorally inhibited children from both individual trait and environmental perspectives.

Full Text

Preamble

Cognitive Neurological Process Associated with Behavioral Inhibition and Psychopathology in Children

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Abstract

Children with behavioral inhibition are more likely to develop psychological disorders during middle childhood and adolescence, representing a higher developmental risk. Such children exhibit greater withdrawal motivation, longer anticipatory and pre-attention processes, reduced attention control, increased attention bias toward negative information, abnormally active response monitoring, and slower habituation compared to non-inhibited children. These similar cognitive-neural processes constitute important pathways linking childhood behavioral inhibition to psychopathology and can serve as predictive indicators for screening individuals at higher developmental risk. Beyond children' s inherent traits, environmental factors can also influence developmental outcomes by shaping cognitive-neural processes. Therefore, future research should consider differences in the cognitive-neural processes linking behavioral inhibition and psychopathology across cultural contexts, thereby identifying protective factors for behaviorally inhibited children from both individual temperament and environmental perspectives.

Keywords: Behavioral inhibition; Psychopathology; Cognitive neural processes; Withdrawal motivation; Pre-attention processes; Attention processes; Response processes; Habituation processes

Behavioral inhibition refers to a temperament or response pattern exhibited by infants and young children when facing novel situations or unfamiliar adults and peers [?, ?, ?, ?, ?, ?]. During initial encounters with unfamiliar people, objects, or situations, children respond in distinct ways: behaviorally inhibited children display withdrawal behaviors such as interrupting ongoing activities, retreating to familiar caregivers, or leaving the location where the unfamiliar event occurs, whereas non-inhibited children show no significant change in their ongoing activities and may even actively approach unfamiliar people or objects [?, ?]. Rubin, Coplan, and Bowker [?] proposed a developmental trajectory for inhibited children: the temperament of behavioral inhibition emerges in infancy, characterized by low arousal thresholds and difficulty in soothing; during toddlerhood, it manifests as inhibited and anxious features associated with social withdrawal, negative self-concept, and related problems in early and middle childhood. Behaviorally inhibited children are more prone to psychological disorders in middle childhood and adolescence compared to typical children.

Existing research has found that childhood behavioral inhibition is associated with a broad range of subsequent psychological disorders. Social anxiety represents the most frequently studied developmental outcome for behaviorally inhibited children, with numerous studies demonstrating that inhibited children have a significantly higher likelihood of developing social anxiety symptoms in middle childhood compared to non-inhibited children [?, ?, ?, ?, ?, ?]. Clauss and Blackford [?] conducted a meta-analysis revealing that behaviorally inhibited children are 7.5 times more likely to develop social anxiety than their non-inhibited counterparts. Additionally, behavioral inhibition serves as a risk factor for other psychological disorders. A five-year longitudinal study indicated that behavioral inhibition correlates with social anxiety, social phobia, panic disorder, obsessive-compulsive symptoms, and depression during middle childhood and adolescence [?, ?]. Behaviorally inhibited children also show greater likelihood of substance abuse during adolescence [?, ?].

The neural basis linking behavioral inhibition and psychopathology primarily involves two aspects: brain structure and cognitive-neural processes. Structurally, individuals identified as behaviorally inhibited in childhood or as highly reactive in infancy exhibit larger amygdalae, caudate nuclei [?, ?, ?, ?], and medial prefrontal cortex volumes, but smaller left orbitofrontal cortex [?, ?, ?], hippocampal [?, ?, ?], and dorsal anterior cingulate cortex volumes [?, ?, ?]. Regarding cognitive-neural processes, previous studies using EEG, ERP, and fMRI have revealed that behaviorally inhibited children show greater withdrawal motivation, longer anticipatory and pre-attention processes, reduced attention control, increased attention bias toward negative information, hyperactive error detection processes, and slower habituation—processes similar to those observed in patients with psychological disorders, thereby illuminating the connection between behavioral inhibition and psychopathology. This review primarily summarizes the characteristics of behavioral inhibition and its association with psychopathology from the perspective of cognitive-neural processes and their relationship with social-cognitive processes.

2 Cognitive Neural Processes of Behavioral Inhibition

According to Sylvester and Pine' s [?] threat system theory, the threat reaction system and threat response system constitute the biological mechanisms linking behavioral inhibition and psychopathology. The reaction system, involving the hypothalamus, primary sensory cortex, amygdala, bed nucleus of the stria terminalis, and hippocampus, rapidly detects and automatically categorizes stimuli, projecting to other brain regions to trigger broader organismic changes. The amygdala within the response system influences individuals' responses to stimuli and is modulated by lateral prefrontal and medial cortical regions. Behaviorally inhibited individuals and those with anxiety disorders tend to identify stimuli as threatening and exhibit exaggerated responses. These emotional reactions to novel stimuli encompass various cognitive-neural processes including attention, working memory, and conscious awareness. The following sections elaborate on the pathways linking behavioral inhibition and psychopathology from more specific cognitive-neural process perspectives.

2.1 Withdrawal Motivation

Withdrawal motivation represents a primary characteristic distinguishing behaviorally inhibited from non-inhibited children. This difference is reflected in frontal EEG asymmetry patterns, representing functional differences in approach and avoidance motivational systems corresponding to distinct hemispheric functions [?, ?]. Left frontal asymmetry is associated with approach-related emotions promoting digestion and engagement, whereas right frontal asymmetry relates to withdrawal and stimulus avoidance [?, ?, ?]. Frontal asymmetry patterns in behaviorally inhibited children can be observed as early as infancy. Fox and Davidson [?] used EEG to examine ten-month-old infants' responses to stranger approach and maternal separation, finding that infants already exhibited frontal asymmetry patterns associated with different motivational states during their first year. Additional research found that highly reactive infants at four months showed greater right frontal activation and more fear expressions at fourteen and twenty-one months [?, ?, ?, ?]. Furthermore, studies continue to observe relationships between frontal asymmetry and behavioral inhibition beyond infancy into childhood and adolescence, with children classified as highly reactive in early childhood showing greater right frontal asymmetry at ages 10-12 [?, ?, ?, ?, ?].

Frontal asymmetry explains the association between behavioral inhibition and psychopathology from both trait and state perspectives. Coan, Allen, and Harmon-Jones [?] proposed that frontal asymmetry serves dual functions regarding emotion and motivation: (1) as an individual difference variable related to temperament, associated with individuals' anticipated emotional responses; and (2) as a state-dependent variable related to emotional states. As an individual difference variable, frontal asymmetry may increase or decrease the likelihood of certain emotional responses. According to Gray' s psychopathology model, emotions represent reactions to motivational systems. High activity in the behavioral inhibition system predisposes individuals to anxiety disorders, whereas low activity in the behavioral activation system predisposes individuals to specific types of depressive symptoms [?, ?]. Genetic studies have also found that differences in system activity have heritable bases and are associated with anxiety disorders, depression, and obsessive-compulsive disorder [?, ?, ?, ?, ?, ?, ?, ?, ?, ?], supporting behavioral inhibition as a risk temperament associated with psychopathology. From a state perspective, when frontal asymmetry functions as a state-dependent variable, it reflects changes in psychological and emotional states. Research has shown that children of depressed mothers exhibit reduced left prefrontal cortical activity, predicting higher depression risk in children [?, ?], suggesting that right frontal asymmetry itself reflects a more negative state associated with psychopathology development.

2.2 Anticipatory Process

Anticipatory process is considered a biased cognitive process that influences emotion regulation. Research has found that during the anticipation of threatening stimuli, behaviorally inhibited children fail to show strong prefrontal engagement; however, when viewing social stimuli, they exhibit increased activation in medial prefrontal and dorsolateral prefrontal regions, confirming the role of prefrontal functional differences in anticipatory processing variations among behaviorally inhibited children [?, ?, ?, ?, ?]. Another study found that for behaviorally inhibited individuals, strong prefrontal system activation during anticipation coupled with functional connectivity between the cingulate and amygdala can protect individuals and reduce their likelihood of developing social anxiety [?, ?, ?]. Overall, behaviorally inhibited individuals show reduced activation in emotion regulation-related brain regions (anterior cingulate, medial prefrontal cortex, dorsolateral prefrontal cortex) but increased activation in limbic systems during anticipatory processes [?, ?].

The differences in brain activity during anticipatory processes among behaviorally inhibited individuals are associated with anxiety disorders and social anxiety. Regarding the duration of anticipatory processing, individuals with high social anxiety show longer anticipatory periods than those with low social anxiety. Since this process involves rumination about upcoming, frequent, and intrusive social events, it increases personal anxiety and interferes with attention [?, ?]. Regarding the type of anticipatory process, maladaptive anticipation (stagnant thinking and outcome fantasizing) also correlates with social anxiety [?, ?, ?, ?, ?]. Behaviorally inhibited individuals fail to appropriately prepare the prefrontal cortex for threatening stimuli during the anticipation phase while simultaneously showing excessive amygdala activation [?, ?, ?, ?], resulting in enhanced initial threat responses related to the threat reaction system and insufficient emotion regulation strategies related to the threat response system, thereby triggering anxiety.

2.3 Pre-attention Process

Pre-attention process itself does not contain attentional components but is related to the automatic initiation of attention [?, ?, ?, ?, ?]. When stimulus materials deviate from standard materials, the brain rapidly responds to unexpected auditory stimuli [?, ?], generating mismatch negativity (MMN). MMN originates in the right parietal and left middle frontal gyrus regions [?, ?, ?, ?, ?] and serves as a reliable index of auditory cognitive pre-attention processes. Generally, MMN latency is considered attention-related, but MMN itself is independent of attentional processes, reflecting the brain' s capacity for automatic comparison [?, ?, ?, ?]. Researchers using standard and deviant auditory stimuli observed that behaviorally inhibited children showed smaller MMN amplitudes and longer MMN latencies in early cortical auditory processing compared to socially competent control children, supporting the role of frontal regions in social withdrawal [?, ?, ?, ?, ?, ?].

Pre-attention processes also represent an important focus in psychopathology research. The smaller MMN amplitudes and longer latencies observed in behaviorally inhibited children relative to typical children mirror findings from studies using standard and deviant auditory stimuli with anxious and depressed participants, suggesting these features reflect a fundamental cognitive process deficit linking to psychopathology [?, ?, ?, ?, ?, ?]. However, some studies have reported contradictory results. Using altered acoustic features inserted in musical sequences as stimuli, researchers found that higher MMN was associated with greater depression risk in depressed patients, attributed to their lower tolerance for unexpected deviations in musical sequences [?, ?, ?, ?, ?, ?]. This material-dependent discrepancy also appears in facial expression experiments [?, ?]. Although previous pre-attention research has yielded inconsistent results, behaviorally inhibited children show similar prefrontal dysfunction-induced alterations in pre-attention processes as anxiety and depression patients when experimental stimuli are comparable, suggesting this shared process may underlie the development of psychopathology in behaviorally inhibited children.

2.4.1 Attention Control

Attention control represents a regulatory capacity that researchers have examined across multiple dimensions. Eysenck, Derakshan, Santos, and Calvo [?] distinguished between stimulus-driven bottom-up control and goal-driven top-down control systems. Anxiety disrupts the balance between these systems, affecting central executive functions including attentional inhibition and shifting. Derryberry and Reed' s [?] attention scale focuses on overall attention control capacity, primarily dividing attention control into focused attention and attention shifting processes [?, ?, ?, ?]. The source of attention control lies in the frontoparietal network, which belongs to the threat response system and is thought to provide flexible threat responses in primates by altering attention toward or away from potential threat stimuli [?, ?]. Focused attention capacity relates to superior and inferior parietal systems [?, ?], while attention shifting capacity relates to ventrolateral prefrontal cortex [?, ?].

Attention control plays a crucial role in childhood behavioral inhibition. Children' s heightened reactivity to novel stimuli and failed cognitive control contribute to the emergence of behavioral inhibition [?, ?, ?]. Previous ERP research on attention processes in behaviorally inhibited individuals has focused on P1 and N2 components. The P1 component reflects early automatic attentional processes, occurring 100-130 ms after stimulus presentation with maximum amplitude at occipital sites [?, ?]. Jetha et al. [?] found that shy adults showed reduced P1 when viewing emotional faces, opposite to non-shy individuals, interpreted as cognitive avoidance and attentional inhibition. This finding, consistent with the amygdala sensitivity model, has been replicated in children, with Thai, Taber-Thomas, and Pérez-Edgar [?] observing P1 enhancement for angry face cues only in non-behaviorally inhibited children using a dot-probe paradigm. Beyond the P1 component, the N2 component also receives significant attention in ERP research. N2 is associated with conflict monitoring, response inhibition, and attention control processes [?, ?]. Research has demonstrated that behavioral inhibition at age two predicts N2 activity at age seven in Go/No-Go tasks, with behavioral inhibition at age two relating to social reticence at age seven only in individuals with high N2 [?, ?].

The role of attention control in linking childhood behavioral inhibition and psychopathology is well-established. Better attention control and the capacity to mobilize cognitive resources to regulate negative emotions serve as protective factors against negative developmental outcomes in behaviorally inhibited children, as flexible attention shifting can modulate maladaptive response patterns [?, ?, ?, ?, ?, ?]. In contrast, children and adolescents with fearful temperaments tend to focus attention on threat stimuli, a cognitive process that increases emotional vulnerability and anxiety symptoms [?]. Consequently, early behavioral inhibition relates to preschool anxiety problems only in individuals with poor attention shifting capacity [?, ?].

2.4.2 Attention Bias

Attention bias constitutes an important factor influencing attention control [?, ?, ?, ?, ?, ?]. Behaviorally inhibited individuals exhibit attention biases in both reward and punishment tasks. Knutson, Westdorp, Kaiser, and Hommer [?] used a monetary incentive delay task to examine attention bias in reward and punishment contexts, finding that striatal activation enhances anticipation of monetary stimuli and improves performance. Specifically in behaviorally inhibited individuals, striatal activation increases with incentive magnitude, an effect present in both reward and punishment anticipation tasks and larger than in non-inhibited groups [?, ?]. Lahat, Benson, Pine, Fox, and Ernst [?] similarly found enhanced striatal activation in behaviorally inhibited individuals, along with stronger caudate activation during reward tasks, which is modulated by the DRD4 gene [?, ?] and associated with social anxiety symptoms [?, ?].

Attention bias may underlie the relationship between childhood behavioral inhibition and social anxiety [?, ?], influenced by fear circuits including the amygdala and prefrontal cortex, and affecting the stability of behavioral inhibition. Research indicates that attention bias toward threat-related information represents a primary cause for anxiety onset and maintenance [?, ?, ?, ?, ?, ?] and moderates the relationship between behavioral inhibition and social withdrawal/anxiety symptoms [?, ?, ?]. Behaviorally inhibited children show enhanced dorsolateral prefrontal activation after viewing negative faces [?, ?, ?, ?] and greater engagement of cingulate, striatal, and dorsal prefrontal regions during high attentional control trials [?, ?]. The dorsolateral prefrontal region, a crucial component of the threat response system, implements emotion regulation strategies such as cognitive reappraisal and active suppression, and its activation correlates with anxiety symptoms [?, ?], revealing the role of attention bias in linking behavioral inhibition and psychopathology.

2.5 Response Process

Error-related response monitoring includes error detection and subsequent adjustment processes [?, ?, ?, ?, ?]. Response processes relate to cingulo-insular network function [?, ?, ?]. The dorsal insula and cingulate cortex belong to the threat response system, with research confirming that cingulate cortex modulates responses to perceived threats, while insular abnormalities in activity, functional connectivity, and cortical thickness also relate to behavioral inhibition [?, ?]. In ERP experiments, response processes are primarily reflected in the ERN, CRN, and Pe components. Previous studies have found that behaviorally inhibited children show larger ERN amplitudes than non-inhibited children, with children aged 11-13 showing more anxiety symptoms also exhibiting larger ERN amplitudes [?, ?]. Enhanced response monitoring demonstrates continuity, with individuals who were behaviorally inhibited in childhood continuing to show larger ERN amplitudes in adolescence [?, ?]. Additionally, the difference between ERN and CRN reflects characteristics of behaviorally inhibited children, with researchers finding larger amplitude differences between these components in highly fearful toddlers [?, ?]. In stressful contexts, larger Pe amplitudes also reflect timidity in children [?, ?, ?, ?].

The enhanced response processes observed in behaviorally inhibited individuals relate to psychopathology. In child samples, larger ERN amplitudes correlate with obsessive-compulsive symptoms [?, ?] and mediate the relationship between early behavioral inhibition and adolescent social anxiety [?, ?]. In adolescent samples, individuals meeting diagnostic criteria for anxiety disorders also show increased ERN components [?, ?]. This ERN enhancement may result from overactive fear systems in behaviorally inhibited children requiring greater inhibitory control [?, ?, ?, ?, ?, ?]. Beyond ERN, two other ERP components related to error processing also connect to psychopathology, with higher anxiety levels associated with stronger CRN [?, ?, ?, ?] and lower Pe amplitudes related to depression [?, ?, ?, ?, ?, ?]. fMRI research has further explored the underlying mechanisms of response processes, observing medial frontal cortex activation including cingulate cortex during task errors [?, ?, ?, ?, ?], particularly cingulate hyperactivity in severe obsessive-compulsive disorder patients [?, ?]. These findings suggest that error-related processes may constitute a cognitive pathway linking behavioral inhibition and psychopathology.

2.6 Habituation Process

High reactivity comprises two distinct processes: greater initial responses to novel stimuli and slower habituation to repeated stimuli. Habituation refers to the disappearance of responses to repetitive stimuli—specifically, the process by which individuals reduce attentional resource allocation when a novel stimulus is identified as identical, non-threatening, and non-rewarding [?, ?, ?, ?]. Behaviorally inhibited children exhibit withdrawal responses to novel stimuli, whereas non-inhibited children quickly engage with new environments and unfamiliar peers. This difference stems not only from behaviorally inhibited children' s tendency to identify ambiguous stimuli as threatening but also from their greater difficulty with habituation [?, ?]. Blackford et al. [?] used fMRI to demonstrate that behaviorally inhibited individuals show faster and greater amygdala activation when viewing novel stimuli and sustained amygdala responses to recently familiarized faces, leading to habituation differences [?, ?, ?, ?, ?]. Additionally, research has identified a subgenual anterior cingulate cortex-centered network that plays an important role in habituation, incorporating experience about whether previous stimuli remain threatening, thereby facilitating threat or non-threat identification [?, ?]. These findings support Sylvester and Pine's [?] threat system theory, indicating that behaviorally inhibited individuals' responses to threat information include not only initial amygdala-mediated reactions related to the threat reaction system but also cingulate-mediated habituation processes related to the threat response system that connect to psychopathology. Fried, MacDonald, and Wilson [?] suggested that failure to quickly adapt to repeated stimuli represents uncertainty or unfamiliarity. The familiarization process relates to delayed security and increased anxiety [?, ?, ?, ?], with recent research also finding slower habituation in depressed patients compared to healthy populations [?, ?]. However, this conclusion has not been universally supported. Sladky et al. [?] found that anxiety patients showed habituation in amygdala, orbitofrontal cortex, and pulvinar thalamus when processing emotional faces during emotion discrimination tasks, contrary to healthy populations. Other studies using card-guessing and monetary reward tasks found faster ventral striatum habituation in depressed patients compared to typical populations [?, ?]. Avery and Blackford [?] proposed that these inconsistencies may stem from different task demands: when participants simply view emotional materials, anxiety and depression patients show slower habituation; however, when task performance is simultaneously required, patients habituate faster than healthy controls. Therefore, the slower habituation observed in behaviorally inhibited individuals during passive emotional stimulus viewing may relate to increased anxiety and depressive symptoms.

3 Cognitive Neural Processes and Social Cognitive Processes

Social-cognitive processes constitute another important pathway linking childhood behavioral inhibition and psychopathology, encompassing environmental influences on children' s social cognition including peer relationships, social expectations, and parenting [?, ?]. The excessive fear in behaviorally inhibited children may originate from either differential perception of social situations or similar social perception coupled with deficient regulation capacity when facing high-arousal contexts.

Cognitive-neural and social-cognitive processes exert not only independent influences on child development but also joint and indirect effects. For behaviorally inhibited children, cognitive-neural processes represent individual-level temperamental risk factors, while social-cognitive processes constitute environmental risk factors. Together they form influences on developmental outcomes. Simultaneously, social-cognitive processes indirectly affect developmental outcomes by shaping cognitive-neural processes.

3.1 Joint Influence of Temperament and Social-Cognitive Processes on Psychopathology

In the joint influence pathway, individual behavior is affected by both temperament and specific environmental factors, with their interaction jointly predicting adaptive outcomes. Therefore, individuals with certain physiological characteristics are more likely to thrive in their adapted environments—for example, low arousal characteristics represent risk factors only in Western cultures emphasizing high arousal but facilitate good adaptation in Eastern cultures [?, ?, ?, ?, ?]. This joint pathway manifests in behaviorally inhibited children as the combined influence of temperament and social-cognitive processes on psychopathology.

Parenting environment plays a significant role in the development of behaviorally inhibited children. Rubin, Burgess, and Hastings [?] found that intrusive and derisive maternal parenting leads to more internalizing problems (anxiety or depression) in behaviorally inhibited children, and parental behavioral inhibition also predicts the relationship between child behavioral inhibition and anxiety disorders [?, ?].

Peer relationships represent another influence on the social development of behaviorally inhibited children. Rubin identified peer difficulties including peer rejection, bullying, and low friendship quality as negative developmental outcomes for behaviorally inhibited children [?, ?]. Research indicates that perceived peer bullying predicts subsequent anxiety symptoms [?, ?, ?, ?] and mediates the relationship between fearful temperament and anxiety symptoms [?, ?, ?, ?]. Additionally, children' s self-blame and avoidant coping when facing peer difficulties contribute to psychological disorders including depression and anxiety, creating a cyclical reinforcement of negative socioemotional and social-cognitive functioning [?, ?, ?, ?, ?, ?, ?].

Contemporaneous cultural background differences and temporal cultural shifts also affect developmental outcomes for behaviorally inhibited children. According to previous Western research, behavioral inhibition and social withdrawal negatively impact children' s social development and adaptation, with such children showing more social anxiety and depressive symptoms, more negative self-evaluation, and greater peer bullying and rejection compared to non-inhibited children [?, ?]. However, inhibited temperament was accepted and adaptive in 1990s Chinese cultural contexts. Chen, Rubin, and Sun [?] compared Chinese and Canadian samples, finding that in Chinese cultural contexts, children' s shyness correlated with positive peer nominations, unlike the association with negative peer nominations reported in Canadian samples, demonstrating cultural context effects. Furthermore, temporal shifts importantly influence developmental outcomes. Research shows that in Chinese cultural contexts, shyness in 1990 correlated positively with positive social nominations, teacher-rated competence, leadership, academic achievement, and social cooperation, whereas by 2002, shyness correlated positively with negative social nominations and depressive symptoms [?, ?, ?, ?].

Although current research only demonstrates interactions between temperament and social-cognitive processes, temperamental differences between behaviorally inhibited and non-inhibited children are manifested at the cognitive-neural level. Therefore, we can anticipate potential interactions between cognitive-neural and social-cognitive processes in the pathways linking behavioral inhibition and psychopathology, representing a future research direction.

3.2 The Shaping of Cognitive-Neural Processes by Social-Cognitive Processes

In the indirect influence pathway, social-cognitive processes shape cognitive-neural processes, thereby affecting developmental outcomes. Kitayama and Uskul [?] propose that individual behavior is influenced not only by neural mechanisms but also by social norms. For behaviorally inhibited children, peer relationships, parenting, and sociocultural expectations constitute important social-cognitive factors affecting developmental outcomes.

3.2.1 The Shaping of Cognitive-Neural Processes by Parenting Environment Previous research confirms that parenting environments shape cognitive-neural processes including withdrawal motivation, attention processes, and response processes in children.

From the perspective of withdrawal motivation, frontal asymmetry features may serve as physiological links between environmental factors and psychopathology. Studies show that maternal postpartum depression and parenting behaviors such as sensitivity, responsiveness, and emotional expression affect infants' frontal asymmetry patterns, with maternal depressive symptoms and more negative parenting associated with right frontal asymmetry in children [?, ?, ?]. Moreover, infants of mothers with stable psychological disorders also show greater right frontal EEG asymmetry, typically accompanied by increased negative emotion and more withdrawal behaviors [?, ?, ?, ?]. Thus, adverse environments may shape children' s right frontal asymmetry features, leading to greater behavioral inhibition and association with multiple psychological disorders.

From the attention process perspective, children' s attention control capacity and attention bias are both influenced by parenting styles. Regarding attention control, directly critical parenting correlates with low attention control levels [?, ?]. Regarding attention bias, attention bias toward angry faces mediates the relationship between authoritarian parenting, negative emotion, and childhood social anxiety disorder [?, ?, ?, ?], with authoritarian parenting representing an important factor influencing the development of behavioral inhibition [?, ?], demonstrating parenting' s role in linking childhood behavioral inhibition and psychopathology.

Response processes also relate to parenting environments, reflecting both genetically based response monitoring [?, ?] and environmental influences. Brooker and Buss [?] found that harsh parenting and fearful emotion predicted greater fearful emotion and larger ERN amplitudes two years later in children, indicating that response monitoring constitutes a cognitive process linking parenting, fearful emotion, and anxiety problems. Both genetically and environmentally, error-related response monitoring and cognitive control processes provide explanations for potential psychological disorders in behaviorally inhibited children.

3.2.2 The Shaping of Neural Processes by Cultural Factors Cultural factors shape neural processes through indirect environmental pathways influencing developmental outcomes. Domínguez Duque, Turner, Lewis, and Egan [?] propose that when people repeatedly participate in cultural practices, cultural behaviors systematically impact the brain, affecting neural activity at multiple levels and dimensions, including low-level perception and attention as well as high-level cognition, emotion, attribution, self-concept, and consciousness. Cultural experience can modulate and determine pre-existing neural activity patterns [?, ?]. Overall, joint and indirect influence pathways are not independent, as interactions themselves demonstrate brain shaping by culture, since genes adapted to environments are more likely to be preserved [?, ?].

Specifically regarding behaviorally inhibited children, current research shows joint effects of social-cognitive and cognitive-neural processes. In Western cultural contexts, behavioral inhibition and social withdrawal negatively affect children' s social development and adaptation, whereas in traditional Chinese cultural contexts, children' s shyness and withdrawal are accepted by parents and peers [?, ?]. Beyond these joint effects, culture and associated differences in parenting styles and peer attitudes may indirectly influence developmental outcomes by shaping cognitive-neural processes, thereby revealing cross-cultural differences. Currently, research on indirect cultural influence pathways in behavioral inhibition remains limited, representing a future research direction.

4.1 The Predictive Role of Cognitive-Neural Processes in the Association Between Behavioral Inhibition and Psychopathology

This review has summarized cognitive-neural processes linking childhood behavioral inhibition and psychopathology. Behaviorally inhibited children are at greater risk for future psychological disorders due to similar neural processes or structural features shared with certain patient populations. Clarifying these neural process associations establishes a foundation for using cognitive-neural processes as predictive indicators of developmental risk in behaviorally inhibited children. Existing research shows that within behaviorally inhibited populations, only individuals displaying these similar processes exhibit higher psychopathology risk, while other behaviorally inhibited individuals do not show greater risk than typical populations.

From the withdrawal motivation perspective, children who consistently show behavioral inhibition exhibit right frontal asymmetry as early as nine months, whereas those who later transition from inhibited to non-inhibited status do not show this feature [?, ?, ?, ?, ?, ?]. Another longitudinal study found that high negative reactivity at nine months correlated with social wariness at age four only in individuals showing right frontal asymmetry [?, ?, ?]. Furthermore, in highly behaviorally inhibited children, right frontal asymmetry correlates with larger ERN amplitudes in social contexts [?, ?], demonstrating the predictive value of early withdrawal motivation for socioemotional development in behaviorally inhibited children and its potential as a screening indicator for individual developmental risk.

From the attention process perspective, the link between behavioral inhibition and social anxiety is influenced by attention control and attention bias. Among ERP components, increased P2 amplitude relates to reduced social anxiety symptoms and moderates the relationship between behavioral inhibition and social anxiety [?, ?]; higher N2 activation or greater dorsal prefrontal cortex and dorsal anterior cingulate cortex activation relates to subsequent social reticence [?, ?]. fMRI research has also identified unique neural system features in high-risk individuals within behaviorally inhibited populations, such as lower default mode network connectivity in striatal and bilateral sensorimotor cortices—regions closely related to attention processes—in behaviorally inhibited subjects from high depression-risk families [?, ?]; behaviorally inhibited young adults with anxiety disorders also show special features like larger amygdala volumes [?, ?, ?]. Roy et al. [?] examined the extended amygdala system, proposing that dysfunction in amygdala, prefrontal cortex, striatum, anterior insula, and cerebellum represents risk indicators for anxiety disorders in behaviorally inhibited individuals.

From the response process perspective, the relationship between response monitoring, inhibitory control, and anxiety symptoms is moderated by behavioral inhibition. In high behavioral inhibition groups, smaller ERN amplitudes correlate with lower anxiety symptoms, indicating that individuals sharing similar neural processes with psychopathology patients show higher developmental risk. Moreover, children exhibiting both behavioral inhibition and enhanced response monitoring may face greater anxiety disorder risk than those showing only behavioral inhibition or only enhanced response monitoring [?, ?]. Panic disorder also relates to response processes. In Flanker tasks, highly behaviorally inhibited children show more error detection than low inhibition children, with higher ERN and CRN amplitudes at age seven predicting panic disorder risk in behaviorally inhibited children [?, ?].

These cognitive-neural processes screen high-risk individuals at the trait level, and future research should consider longitudinal studies to validate these predictive relationships. Meanwhile, developmental outcomes for behaviorally inhibited children are also influenced by environmental factors. Thus, after further clarifying interactions between social-cognitive processes such as culture and parenting with cognitive-neural processes, and the shaping effects of social-cognitive processes on cognitive-neural processes, researchers can more deeply explain mechanisms linking behavioral inhibition and psychopathology.

4.2 The Role of Cultural Factors

Rubin et al. [?] propose that cultural differences in adaptive outcomes for behaviorally inhibited children arise because parents transmit sociocultural orientations and expectations to children during parenting. In traditional collectivist cultures, restrained, quiet, low-initiative behaviors are socially accepted, leading to better developmental outcomes for inhibited temperament children. In market economies and individualist cultures, however, inhibited temperament represents a maladaptive pattern, with children receiving more negative parental evaluations and peer rejection/bullying, forming negative self-concepts and low self-worth, ultimately leading to emotional and adjustment problems.

Self-concept structures themselves differ substantially between Western and Chinese cultures. Individuals in Asian cultures possess an interdependent self-concept, maintaining interconnectedness with others, whereas Western culture encourages independence from others to express unique internal attributes, de-emphasizing such connections [?, ?]. Zhu and Zhang [?] used the self-reference effect in memory to study Chinese individuals'self-personality traits, similarly revealing Chinese interdependent self-features. This self-concept difference means shy, sensitive children better meet interpersonal harmony requirements in traditional Chinese contexts, showing greater adaptation than in Western cultures advocating individual independence [?, ?].

These studies demonstrate the substantial impact of cultural factors on individual development. Such cultural factors may influence neural functions, thereby indirectly affecting developmental outcomes. Therefore, self-concept differences between Chinese and Western cultures may also manifest in children' s cognitive-neural processes, leading to different pathways linking behavioral inhibition and psychopathology across cultures. However, cross-cultural research on cognitive-neural processes in behaviorally inhibited children remains insufficient, representing a future research direction.

4.3 Prevention of Negative Socioemotional Developmental Outcomes in Behaviorally Inhibited Children

Behavioral inhibition, as a temperament characteristic in infancy and early childhood, manifests as vigilance, quietness, and withdrawal from social stimuli. This early temperament relates to subsequent socioemotional developmental outcomes, with consistently high levels of behavioral inhibition predicting high levels of social withdrawal in early childhood, particularly specific types of social withdrawal. Social withdrawal, along with multiple psychological disorders described above, constitutes negative socioemotional developmental outcomes in children, and social withdrawal itself can lead to anxiety and depression [?, ?].

Social withdrawal has been subtyped by Coplan et al. [?] into three categories corresponding to different developmental outcomes. Anxious-withdrawn children show both high approach and high avoidance motivation, expressing the most anxiety among withdrawn subtypes; unsociable-withdrawn children show low approach motivation with fewer internalizing problems than the anxious-withdrawn type; and active-isolated children do not avoid social interaction and express the most positive emotion among all subtypes. From a motivational perspective, behaviorally inhibited children also experience the conflict state of high approach and high avoidance motivation [?, ?], corresponding to the anxious-withdrawn subtype of social withdrawal—the subtype with the most internalizing problems, showing more anxiety, depression, and social anxiety symptoms [?, ?, ?]. However, current explanations for the relationship between behavioral inhibition and social withdrawal derive only from behavioral observations of children' s motivation and emotion; neuroscientific exploration of underlying neural mechanisms remains limited. Future research should further validate and explore associated cognitive-neural processes, such as using EEG frontal asymmetry to measure children' s motivational components from neuroscientific and longitudinal perspectives to verify behavioral observations, or employing ERP and fMRI to examine characteristics of attention and response processes to further explain mechanisms underlying socioemotional developmental outcomes in behaviorally inhibited children and explore connections among behavioral inhibition, social withdrawal, and psychopathology.

Furthermore, helping behaviorally inhibited children better adapt socially, reducing social withdrawal and even social anxiety, can increase peer acceptance and reduce psychopathology risk from an environmental perspective. Currently, a few preventive interventions target behavioral inhibition. Tang, Reeb-Sutherland, Romeo, and Mcewen [?] used novel stimulus exposure to promote habituation in rats, effectively reducing fear of novel environments and anxiety risk. Week [?] applied cognitive-behavioral therapy targeting social anxiety as a preventive intervention for behaviorally inhibited individuals, finding it effectively reduced inhibition levels and decreased social anxiety likelihood. Future research should explore effective interventions based on characteristics of cognitive-neural processes in behaviorally inhibited individuals to reduce children' s risk of social withdrawal and psychopathology.

References:

Zhu, Y., & Zhang, L. (2001). Experimental study on self-memory effect. *Science in China*, 31(6), 537-543.

Aarts, K., Vanderhasselt, M. A., Otte, G., Baeken, C., & Pourtois, G. (2013). Electrical brain imaging reveals the expression and timing of altered error monitoring functions in major depression. *Journal of Abnormal Psychology*, 122(4), 939-950.

Affrunti, N. W., Geronimi, E. M. C., & Woodruff-Borden, J. (2014). Temperament, peer victimization, and nurturing parenting in child anxiety: A moderated mediation model. *Child Psychiatry & Human Development*, 45(4), 483-492.

Allen, J. J. B., & Reznik, S. J. (2015). Frontal EEG asymmetry as a promising marker of depression vulnerability: Summary and methodological considerations. *Current Opinion in Psychology*, 4, 93-97.

Asendorpf, J. B. (1990). Beyond social withdrawal: Shyness, unsociability, and peer avoidance. *Human Development*, 33(4-5), 250–259.

Asendorpf, J. B., & Asendorpf, J. B. (1991). Development of inhibited children' s coping with unfamiliarity. *Child Development*, 62(6), 1460–1474.

Avery, S. N., & Blackford, J. U. (2016). Slow to warm up: the role of habituation in social fear. *Social Cognition Affect Neuroscience*, 11(11), 1832–1840.

Barhaim, Y., Marshall, P. J., Fox, N. A., Schorr, E. A., & Gordon-Salant, S. (2003). Mismatch negativity in socially withdrawn children. *Biological Psychiatry*, 54(1), 17–24.

Barhaim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & Van Ijzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: a meta-analytic study. *Psychological Bulletin*, 133(1), 1–24.

Barker, T. V., Reeb-Sutherland, B., Degnan, K. A., Walker, O. L., Chronis-Tuscano, A., & Henderson, H. A., ···Fox, N. A. (2015). Contextual startle responses moderate the relation between behavioral inhibition and anxiety in middle childhood. *Psychophysiology*, 52(11), 1544–1549.

Bellgowan, J. F., Molfese, P., Marx, M., Thomason, M., Glen, D., & Santiago, J., ···Hamilton, J. P. (2015). A neural substrate for behavioral inhibition in the risk for major depressive disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 54(10), 841–848.

Blackford, J. U., Buckholtz, J. W., Avery, S. N., & Zald, D. H. (2010). A unique role for the human amygdala in novelty detection. *Neuroimage*, 50(3), 1188–1193.

Blackford, J. U., Avery, S. N., Cowan, R. L., Shelton, R. C., & Zald, D. H. (2011). Sustained amygdala response to both novel and newly familiar faces characterizes inhibited temperament. *Social Cognitive and Affective Neuroscience*, 6(5), 621–629.

Blackford, J. U., Allen, A. H., Cowan, R. L., & Avery, S. N. (2013). Amygdala and hippocampus fail to habituate to faces in individuals with an inhibited temperament. *Social Cognitive and Affective Neuroscience*, 8(2), 143–150.

Blackford, J. U., Clauss, J. A., & Benningfield, M. M. (2018). The Neurobiology of Behavioral Inhibition as a Developmental Mechanism. In K. Pérez-Edgar & N. A. Fox (Eds.), *Behavioral Inhibition* (pp. 113–134). Cham, Gewerbestrasse, Switzerland: Springer, Cham.

Bonetti, L., Haumann, N. T., Vuust, P., Kliuchko, M., & Brattico, E. (2017). Risk of depression enhances auditory pitch discrimination in the brain as indexed by the mismatch negativity. *Clinical Neurophysiology*, 128(10), 1923–1936.

Brooker, R. J., Buss, K. A., & Dennis, T. A. (2011). Error-monitoring brain activity is associated with affective behaviors in young children. *Developmental Cognitive Neuroscience*, 1(2), 141–152.

Brooker, R. J., & Buss, K. A. (2014a). Harsh parenting and fearfulness in toddlerhood interact to predict amplitudes of preschool error-related negativity. *Developmental Cognitive Neuroscience*, 9(2014), 148–159.

Brooker, R. J., & Buss, K. A. (2014b). Toddler fearfulness is linked to individual differences in preschool error-related negativity. *Developmental Neuropsychology*, 39(1), 1–8.

Buzzell, G. A., Troller-Renfree, S. V., Barker, T. V., Bowman, L. C., Chronis-Tuscano, A., Henderson, H. A., ···& Fox, N. A. (2017). A neurobehavioral mechanism linking behaviorally inhibited temperament and later adolescent social anxiety. *Journal of the American Academy of Child & Adolescent Psychiatry*, 56(12), 1097–1105.

Carrasco, M., Hong, C., Nienhuis, J. K., Harbin, S. M., Fitzgerald, K. D., Gehring, W. J., & Hanna, G. L. (2013). Increased error-related brain activity in youth with obsessive-compulsive disorder and other anxiety disorders. *Neuroscience letters*, 541(2013), 214–218.

Chakalov, I., Paraskevopoulos, E., Wollbrink, A., & Pantev, C. (2014). Mismatch negativity to acoustical illusion of beat: how and where the change detection takes place? *NeuroImage*, 100(2014), 337–346.

Chen, X., Rubin, K. H., & Sun, Y. (1992). Social reputation and peer relationships in Chinese and Canadian children: A cross-cultural study. *Child Development*, 63(6), 1336–1343.

Chen, X., Hastings, P. D., Rubin, K. H., Chen, H., Cen, G., & Stewart, S. L. (1998). Child-rearing attitudes and behavioral inhibition in Chinese and Canadian toddlers: a cross-cultural study. *Developmental Psychology*, 34(4), 677–686.

Chen, X., Cen, G., & He, L. Y. (2005). Social functioning and adjustment in Chinese children: the imprint of historical time. *Child Development*, 76(1), 182–195.

Chen, X., & French, D. C. (2008). Children' s social competence in cultural context. *Annual Review of Psychology*, 59(1), 591–616.

Chung, K., & Park, J. Y. (2018). Beyond the real world: Attention debates in auditory mismatch negativity. *Neuroreport*, 29(6), 472–477.

Clauss, J. A., Ronald L. Cowan, & Jennifer Urbano Blackford. (2011). Expectation and temperament moderate amygdala and dorsal anterior cingulate cortex responses to fear faces. *Cognitive Affective & Behavioral Neuroscience*, 11(1), 13–21.

Clauss, J. A., & Blackford, J. U. (2012). Behavioral inhibition and risk for developing social anxiety disorder: a meta-analytic study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(10), 1066–1075.

Clauss, J. A., Avery, S. N., Vanderklok, R., Cowan, R. L., Benningfield, M. M., & Blackford, J. U. (2014). Neurocircuitry underlying risk and resilience to social anxiety disorder. *Depression and Anxiety*, 31(10), 822–833.

Clauss, J. A., Seay, A. L., Vanderklok, R. M., Avery, S. N., Cao, A., Cowan, R. L., ···Blackford, J. U. (2014). Structural and functional bases of inhibited temperament. *Social Cognitive and Affective Neuroscience*, 9(12), 2049–2058.

Clauss, J. A., Benningfield, M. M., Rao, U., & Blackford, J. U. (2016). Altered prefrontal cortex function marks heightened anxiety risk in children. *Journal of the American Academy of Child & Adolescent Psychiatry*, 55(9), 809–816.

Coan, J. A., Allen, J. J. B., Harmon-Jones, E. (2001). Voluntary facial expression and hemispheric asymmetry over the frontal cortex. *Psychophysiology* 38(6), 912–925.

Coplan, R. J., Rose-Krasnor, L., Weeks, M., Kingsbury, A., Kingsbury, M., & Bullock, A. (2013). Alone is a crowd: social motivations, social withdrawal, and socioemotional functioning in later childhood. *Developmental Psychology*, 49(5), 861–875.

Davidson, R. J. (1992). Anterior cerebral asymmetry and the nature of emotion. *Brain and Cognition*, 20(1), 125–151.

Derryberry, D. & Reed, M. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology*, 111(2): 225–243.

Domínguez Duque, J. F., Turner, R., Lewis, E. D., & Egan, G. (2010). Neuroanthropology: a humanistic science for the study of the culture-brain nexus. *Social Cognitive & Affective Neuroscience*, 5(2-3), 138–147.

Dosenbach, N. U., Visscher, K. M., Palmer, E. D., Miezin, F. M., Wenger, K. K., Kang, H. C., ···Petersen, S. E. (2006). A core system for the implementation of task sets. *Neuron*, 50(5), 799–812.

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: attentional control theory. *Emotion*, 7(2), 336–353.

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on tutorial. *Biological significance: a functional reaction errors and their psychology*, 51(2-3), 87–107.

Fitzgerald, K. D., Welsh, R. C., Gehring, W. J., Abelson, J. L., Himle, J. A., & Liberzon, I., ···Taylor, S. F. (2005). Error-related hyperactivity of the anterior cingulate cortex in obsessive-compulsive disorder. *Biological Psychiatry*, 57(3), 287–294.

Fox, N. A., & Davidson, R. J. (1987). Electroencephalogram asymmetry in response to in 10-month-old the approach of a stranger and maternal separation infants. *Developmental Psychology*, 23(2), 233–240.

Fox, N. A., (1991). If it' s not left, it' s right: electroencephalograph asymmetry and the development of emotion. *American Psychologist*, 46(8), 863–872.

Fox, N. A., Calkins S. D., & Bell, M. A. (1994). Neural plasticity and development in the first two years of life: evidence from cognitive and socioemotional domains of research. *Development and Psychopathology*, 6 (4), 677–696.

Fox, N. A., Henderson, H. A., Rubin, K. H., Calkins, S. D., & Schmidt, L. A. (2010). inhibition and exuberance: Continuity and discontinuity of behavioral psychophysiological and behavioral influences across the first four years of life. *Child Development*, 72(1), 1–21.

Fried, I., MacDonald, K. A., & Wilson, C. L. (1997). Single neuron activity in human hippocampus and amygdala during recognition of faces and objects. *Neuron*, 18(5), 753–765.

Fu, X., Taber-Thomas, B. C., & Pérez-Edgar, K. (2017). Frontolimbic functioning during threat-related attention: Relations to early behavioral inhibition and anxiety in children. *Biological Psychology*, 122(2017), 98-109.

Garnefski, N., Kraaij, V., & Etten, M. V. (2005). Specificity of relations between internalizing and adolescents' cognitive emotion regulation strategies and externalizing psychopathology. *Journal of Adolescence*, 28(5), 619–631.

Giard, M. H., Perrin, F., Pernier, J., & Bouchet, P. (2010). Brain generators implicated in the processing of auditory stimulus deviance: a topographic event-related potential study. *Psychophysiology*, 27(6), 627–640.

Goldstein, B. L., Shankman, S. A., Kujawa, A., Torpey-Newman, D. C., Olino, T. M., & Klein, D. N. (2016). Developmental changes in electroencephalographic frontal asymmetry in young children at risk for depression. *Journal of Child Psychology & Psychiatry*, 57(9), 1075.

Gulley, L. D., Oppenheimer, C. W., & Hankin, B. L. (2014). Associations among to anger, and social anxiety among negative parenting, attention bias youth. *Developmental Psychology*, 50(2), 577–585.

Guyer, A. E., Nelson, E. E., Pérez-Edgar, K., Hardin, M. G., Robersonnay, R., Monk, C. S., ···Ernst, M. (2006). Striatal functional alteration in adolescents characterized by early childhood behavioral inhibition. *Journal of Neuroscience the Official Journal of the Society for Neuroscience*, 26(24), 6399–6405.

Harmonjones, E., & Allen, J. J. B. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology*, 106(1), 159–163.

Harrewijn, A., Buzzell, G. A., Debnath, R., Leibenluft, E., Pine, D. S., & Fox, N. A. (2019). Frontal alpha asymmetry moderates the relations between behavioral inhibition and social-effect ERN. *Biological psychology*, 141(2019), 10–16.

Heeren, A., Mogoaşe, C., Mcnally, R. J., Schmitz, A., & Philippot, P. (2015). Does attention bias modification improve attentional control? A double-blind randomized experiment with individuals with social anxiety disorder. *Journal of Anxiety Disorders*, 29(1), 35–42.

Henderson, H. A., Fox, N. A., & Rubin, K. H. (2001). Temperamental contributions to social behavior: the moderating roles of frontal EEG asymmetry and gender. *Journal of the American Academy of Child & Adolescent Psychiatry*, 40(1), 68–74.

Hirshfeld-Becker, D. R., Biederman, J., Henin, A., Faraone, S. V., Davis, S., Harrington, K., & Rosenbaum, J. F. (2007). Behavioral inhibition in preschool children at risk is a specific predictor of middle childhood social anxiety: a five-year follow-up. *Journal of Developmental & Behavioral Pediatrics*, 28(3), 225–233.

Holroyd, C. B., & Mgh, C. (2002). The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, 109(4), 679–709.

Hum, K. M., Manassis, K., & Lewis, Marc D. (2013). Neural mechanisms of emotion regulation in childhood anxiety. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 54(5), 552–564.

Ischebeck, M., Endrass, T., Simon, D., & Kathmann, N. (2014). Altered frontal EEG asymmetry in obsessive-compulsive disorder. *Psychophysiology*, 51(7), 596–601.

Jarcho, J. M., Fox, N. A., Pine, D. S., Leibenluft, E., Shechner, T., & Degnan, K. A., ···Ernst, M. (2014). Enduring influence of early temperament on neural mechanisms mediating attention-emotion conflict in adults. *Depression & Anxiety*, 31(1), 53–62.

Jarcho, J. M., & Guyer, A. E. (2018). The Neural Mechanisms of Behavioral Inhibition. In K. Pérez-Edgar & N. A. Fox (Eds.), *Behavioral Inhibition* (pp. 59–90). Cham, Gewerbestrasse, Switzerland: Springer, Cham.

Jetha, M. K., Zheng, X., Schmidt, L. A., & Segalowitz, S. J. (2012). Shyness and the first 100 ms of emotional face processing. *Social Neuroscience*, 7(1), 74–89.

Jones, N. A., Field, T., & Almeida, A. (2009). Right frontal EEG asymmetry and behavioral inhibition in infants of depressed mothers. *Infant Behavior and Development*, 32(3), 298–304.

Kagan, J., Reznick, J. S., Snidman, N., Gibbons, J., & Johnson, M. O. (1988). Childhood derivatives of inhibition and lack of inhibition to the unfamiliar. *Child Development*, 59(6), 1580–1589.

Kagan, J., & Snidman, N. (1991). Temperamental factors in human development. *American Psychologist*, 46(8), 856–862.

Killeen, L. A. (2010). Understanding parenting as a process: Frontal EEG alpha asymmetry as a measure of "online" maternal responsiveness to infant cues (Unpublished doctorial dissertation). The Pennsylvania State University.

Kitayama, S., & Uskul, A. K. (2011). Culture, mind, and the brain: Current evidence and future directions. *Annual Review of Psychology*, 62(1), 419–449.

Kitayama, S., & Huff, S. (2015). Cultural Neuroscience: Connecting Culture, Brain, and Genes. *Emerging Trends in the Social and Behavioral Sciences: An Interdisciplinary, Searchable, and Linkable Resource*, pp. 1-16. New York: Wiley.

Knutson, B., Westdorp, A., Kaiser, E., & Hommer, D. (2000). fMRI visualization of brain activity during a monetary incentive delay task. *Neuroimage*, 12(1), 20–27.

Lahat, A., Lamm, C., Chronistuscano, A., Pine, D. S., Henderson, H. A., & Fox, N. A. (2014). Early behavioral inhibition and increased error monitoring predict later social phobia symptoms in childhood. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(4), 447–455.

Lahat, A., Benson, B. E., Pine, D. S., Fox, N. A., & Ernst, M. (2018). Neural responses to reward in childhood: relations to early behavioral inhibition and social anxiety. *Social cognitive and affective neuroscience*, 13(3), 281–289.

Lamm, C., Walker, O. L., Degnan, K. A., Henderson, H. A., Pine, D. S., Mcdermott, J. M., & Fox, N. A. (2014). Cognitive control moderates early childhood temperament in predicting social behavior in 7-year-old children: An ERP study. *Developmental science*, 17(5), 667–681.

Liu, J., Chen, X., Coplan, R. J., Ding, X., Zarbatany, L., & Ellis, W. (2015). Shyness and unsociability and their relations with adjustment in Chinese and Canadian children. *Journal of Cross-Cultural Psychology*, 46(3), 1–16.

Mangun, G. R. (1995). Neural mechanisms of visual selective attention. *Psychophysiology*, 32(1), 4–18.

Markus, H. R., & Kitayama, S. (1991). Cultural variation in the self-concept. In *The self: Interdisciplinary approaches* (pp. 18–48). New York: Springer.

Mcdermott, J. M., Pérez-Edgar, K., Henderson, H. A., Chronistuscano, A., Pine, D. S., & Fox, N. A. (2009). A history of childhood behavioral inhibition and enhanced response monitoring in adolescence are linked to clinical anxiety. *Biological Psychiatry*, 65(5), 445–448.

Mcmanis, M. H., Kagan, J., Snidman, N. C., & Woodward, S. A. (2010). EEG children. *Developmental temperament asymmetry*, *Psychobiology*, 41(2), 169–177.

Moses-Kolko, E. L., Fraser, D., Wisner, K. L., James, J. A., Saul, A. T., Fiez, J. A., & Phillips, M. L. (2011). Rapid habituation of ventral striatal response to reward receipt in postpartum depression. *Biological psychiatry*, 70(4), 395–399.

Pang, X., Xu, J., Chang, Y., Tang, D., Zheng, Y., & Liu, Y., et al. (2014). Mismatch negativity of sad syllables is absent in patients with major depressive disorder. *Plos One*, 9(3), e91995.

Pérez-Edgar, K., Barhaim, Y., Mcdermott, J. M., Chronistuscano, A., Pine, D. S., & Fox, N. A. (2010). Attention biases to threat and behavioral inhibition in early childhood shape adolescent social withdrawal. *Emotion*, 10(3), 349–357.

Pérez-Edgar, K., Hardee, J. E., Guyer, A. E., Benson, B. E., Nelson, E. E., Gorodetsky, E., …Ernst, M. (2014). Drd4 and striatal modulation of the link between childhood behavioral inhibition and adolescent anxiety. *Social Cognitive & Affective Neuroscience*, 9(4), 445–453.

Pérez-Edgar, K. (2018). Attention Mechanisms in Behavioral Inhibition: Exploring and Exploiting the Environment. In K. Pérez-Edgar & N. A. Fox (Eds.), *Behavioral Inhibition* (pp. 237-261). Cham, Gewerbestrasse, Switzerland: Springer, Cham.

Reinholdt-Dunne, M. L., Mogg, K., & Bradley, B. P. (2013). Attention control: relationships between self-report and behavioural measures, and symptoms of anxiety and depression. *Cognition & Emotion*, 27(3), 430–440.

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial frontal cortex in cognitive control. *Science*, 306(5695), 443–447.

Roy, A. K., Benson, B. E., Degnan, K. A., Pérez-Edgar, K., Pine, D. S., Fox, N. A., & Ernst, M. (2014). Alterations in amygdala functional connectivity reflect early temperament. *Biological Psychology*, 103, 248–254.

Rubin K., Bukowski W., & Parker J. (2006). Peer interactions, relationships, and groups. In W. Damon, R. Lerner, & N. Eisenberg (Eds.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 571-645). New York: Wiley.

Rubin, K. H., Coplan, R. J., & Bowker, J. (2009). Social withdrawal in childhood. *Annual Review of Psychology*, 60, 141–171.

Rubin, K. H., Burgess, K. B., & Hastings, P. D. (2010). Stability and social-behavioral consequences of toddlers' inhibited temperament and parenting behaviors. *Child Development*, 73(2), 483–495.

Schleider, J. L., Ginsburg, G. S., & Drake, K. (2018). Perceived peer victimization predicts anxiety outcomes in a prevention program for offspring of anxious parents. *Journal of Clinical Child & Adolescent Psychology*, 47(1), S255–S263.

Schmidt, E. T. B., & Bierman, K. L. (2015). Dimensions of parenting associated with child prekindergarten emotion regulation and attention control in low-income families. *Social Development*, 24(3), 601–620.

Schmidt, L. A., Fox, N. A., Rubin, K. H., & Sternberg, E. M. (2015). Behavioral and neuroendocrine responses in shy children. *Developmental Psychobiology*, 30(2), 127–140.

Schneider, M., Chau, L., Mohamadpour, M., Stephens, N., Arya, K., & Grant, A. (2016). EEG asymmetry and bis/bas among healthy adolescents. *Biological Psychology*, 120(2016), 142–148.

Schwartz, C. E., Kunwar, P. S., Greve, D. N., Moran, L. R., Viner, J. C., Covino, J. M., ···Wallace, S. R. (2009). Structural differences in adult orbital and ventromedial prefrontal cortex predicted by temperament at 4 months of infant age. *Neuroimage*, 47(1), S184–S184.

Schwartz, C. E., Kunwar, P. S., Hirshfeld-Becker, D. R., Henin, A., Vangel, M. G., Rauch, S. L., ···Rosenbaum, J. F. (2015). Behavioral inhibition in childhood predicts smaller hippocampal volume in adolescent offspring of parents with panic disorder. *Translational Psychiatry*, 5(7), e605.

Scott, J. H., Yap, K., Francis, Andrew, J., & Schuster, Sharynn. (2014). Perfectionism and its relationship with anticipatory processing in social anxiety. *Australian Journal of Psychology*, 66(3), 187–196.

Sladky, R., Höflich, A., Atanelov, J., Kraus, C., Baldinger, P., Moser, E., ··· Windischberger, C. (2012). Increased neural habituation in the amygdala and orbitofrontal cortex in social anxiety disorder revealed by fMRI. *PloS One*, 7(11), e50050.

Smit, D. J. A., Posthuma, D., Boomsma, D. I., & De Geus, E. J. C. (2007). The relation between frontal EEG asymmetry and the risk for anxiety and depression. *Biological Psychology*, 74(1), 26–33.

Stout, D. M., Shackman, A. J., & Larson, C. L. (2013, April 26). Failure to filter: Anxious individuals show inefficient gating of threat from working memory. *Frontiers in Human Neuroscience*, 7. Retrieved April 26, 2013 from http://search.ebscohost.com/login.aspx?direct=true&db=psyh&AN=2013-15814-001&lang=zh-cn&site=ehost-live.

Stumper, A., Danzig, A. P., Dyson, M. W., Olino, T. M., Carlson, G. A., & Klein, D. N. (2017). Parents' behavioral inhibition moderates association of preschoolers' BI with risk for age 9 anxiety disorders. *Journal of Affective Disorders*, 210, 35–42.

Sylvester, C. M., Barch, D. M., Harms, M. P., Belden, A. C., Oakberg, T. J., Gold, A. L., ···Pine, D. S. (2016). Early childhood behavioral inhibition predicts cortical thickness in adulthood. *Journal of the American Academy of Child & Adolescent Psychiatry*, 55(2), 122–129.

Sylvester, C. M., & Pine, D. S. (2018). The biological bridge between behavioral inhibition and psychopathology. In K. Pérez-Edgar & N. A. Fox (Eds.), *Behavioral Inhibition* (pp. 309–335). Cham, Gewerbestrasse, Switzerland: Springer, Cham.

Tang, A. C., Reeb-Sutherland, B. C., Romeo, R. D., & Mcewen, B. S. (2012).

Reducing behavioral inhibition to novelty via systematic neonatal novelty exposure: the influence of maternal hypothalamic-pituitary-adrenal regulation. *Biological Psychiatry*, 72(2), 150–156.

Thai, N., Taber-Thomas, B. C., & Pérez-Edgar, K. E. (2016). Neural correlates of attention biases, behavioral inhibition, and social anxiety in children: an ERP study. *Developmental Cognitive Neuroscience*, 19(2016), 200–210.

Thorell, L., Bohlin, G., & Rydell, A. M. (2004). Two types of inhibitory control: predictive relations to social functioning. *International Journal of Behavioral Development*, 28(3), 193–203.

Troller-Renfree, S. V., Buzzell, G. A., Bowers, M. E., Salo, V. C., Forman-Alberti, A., Smith, E., ···& Fox, N. A. (2019). Development of inhibitory control during childhood and its relations to early temperament and later social anxiety: unique insights provided by latent growth modeling and signal detection theory. *Journal of Child Psychology and Psychiatry*, 60(6), 622–629.

Tsai, J. L., Knutson, B., & Fung, H. H. (2006). Cultural variation in affect valuation. *Journal of Personality and Social Psychology*, 90(2), 288–307.

Van, V. V., & Carter, C. S. (2002). The anterior cingulate as a conflict monitor: fMRI and ERP studies. *Physiology & Behavior*, 77(4), 477–482.

Vassilopoulos, S. P. (2004). Anticipatory processing in social anxiety. *Behavioural & Cognitive Psychotherapy*, 32(3), 303–311.

Walker, F. R., Thomson, A., Pfingst, K., Vlemincx, E., Aidman, E., & Nalivaiko, E. (2019). Habituation of the electrodermal response–A biological correlate of resilience? *PloS One*, 14(1), e0210078.

Weeks, J. W. (2014). *The wiley blackwell handbook of social anxiety disorder*. New York: John Wiley & Sons, Incorporated.

Wen, D. J., Soe, N. N., Sim, L. W., Sanmugam, S., Kwek, K., Chong, Y. S., ···Qiu, A. (2017). Infant frontal EEG asymmetry in relation with postnatal maternal depression and parenting behavior. *Translational Psychiatry*, 7(3), e1057.

White, L. K., Mcdermott, J. M., Degnan, K. A., Henderson, H. A., & Fox, N. A. (2013). Erratum to: behavioral inhibition and anxiety: the moderating roles of inhibitory control and attention shifting. *Journal of Abnormal Child Psychology*, 41(1), 177.

White, L. K., Degnan, K. A., Henderson, H. A., Pérez-Edgar, Koraly, Walker, O. L., Shechner, T., ···Fox, N. A. (2017). Developmental relations among behavioral inhibition, anxiety, and attention biases to threat and positive information. *Child Development*, 88(1), 141–155.

Williams, L. R., Fox, N. A., Lejuez, C. W., Reynolds, E. K., Henderson, H. A., Pérez-Edgar, K. E., ···& Pine, D. S. (2010). Early temperament, propensity for risk-taking and adolescent substance-related problems: A prospective multi-method investigation. *Addictive Behaviors*, 35(12), 1148-1151.

Xiao, Z. P., Chen, X. S., Zhang, M. D., Lou, F. Y., & Chen, J. (2005). Follow-up study of three cognitive potentials of depression and anxiety disorders. *Medical Bulletin of Shanghai Jiaotong University*, 17(2), 79-82.

Zhao, M., Liu, T., & Chen, F. (2018). Automatic processing of pragmatic information in the human brain: a mismatch negativity study. *NeuroReport*, 29(8), 631-636.

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