Investigating the Cognitive and Neurobiological Factors in Adolescent Depression Through the Lens of the Adolescent Information Processing Model

Hannah Kim

Received October 26, 2024
Accepted January 24, 2025
Electronic access January 31, 2025

Depression in young people is a multi-faceted condition, involving both emotional, behavioral, psychological, and neurobiological factors. While maladaptive neuroplasticity may increase the susceptibility to depression, the same neuroplasticity can also present a remarkable opportunity for recovery. This review paper employs Rice and Dolgin's Adolescent Information Processing Model to explore the cognitive and neurobiological factors contributing to depression in adolescents. The present research conducts a narrative review of existing scientific literature on information processing, cognitive biases, and neurobiological alterations associated with depression in individuals aged 9 to 18. The findings support the hypothesis that cognitive impairments play a crucial role in the development of adolescent depression, and neurobiological impairments, linked to these cognitive impairments, compound its severity. These impairments disrupt various stages of information processing in adolescents including: 1) selection, 2) interpretation, 3) memory, 4) inference, 5) thinking, and 6) reasoning. Through a stage-focused analysis, the paper nests the cognitive and neurobiological complexity of depression into these individual steps. This study concludes by emphasizing the need for future research to address several limitations, including the consideration of additional factors influencing depression, such as social and emotional elements, the exploration of the interconnectedness between cognitive and neurobiological alterations across different information processing steps, and the investigation of causal relationships between cognitive and neurobiological factors through longitudinal studies. Such longitudinal studies can guide the development of stage-specific therapeutic interventions, such as Cognitive Behavioral Therapy, for adolescent depression.

Keywords: Adolescent Depression, Adolescent Information Processing Model, Cognitive Impairment, Neurobiological Alteration

Introduction

Adolescence, a life stage typically spanning between the ages of 9 to 18[1], is marked by vast possibilities, unconventional thinking, and idealistic dreams[2]. However, depression can disrupt these hallmarks of adolescent behavior[3], leading to self-critical thoughts[4], feelings of hopelessness[5], and suicidal behavior[6]. Adolescent depression is a significant concern: In 2021, for example, 20.1% of adolescents aged 12 to 17 experienced a major depressive episode (MDE), with 14.7% reporting severe impairment[7]. Depression not only stands as a leading cause of disability among adolescents[8] but is also a major contributor to suicide rates in this age group[9]. This issue highlights the urgent need to understand and address depression in adolescents to mitigate severe outcomes such as suicide.

In the broader context of behavioral, emotional, and social processes[3], cognitive and biological alterations play a significant role in informing depression. Impaired cognitive alterations are argued to be a potentially key underlying cause of the onset of depression[10]. During these cognitive changes, neurobiological alterations have been observed to simultaneously occur, exacerbating depressive symptoms in adolescents[11]. Research shows that patients exhibiting neurobiological alterations may indicate a higher severity of depression[12], as spike-timing-dependent plasticity suggests that the prolonged reinforcement of negative thinking can reshape synaptic connections and the brain's anatomy[13,14]. Current research shows that cognitive and neurobiological changes provide clear measurable and mechanistic insights[15], involving the examination of "how specific components interact and lead to observed effects" (pg. 1) demonstrating high potential to inform more targeted interventions.

From the onset of depression, individuals often exhibit negative cognitive biases, which causes a pervasive sense of pessimism[16]. These biases trap individuals in a cycle of negativity, such as dwelling on past challenges[17] or anticipating future problems[18]. As these biases become deeply integrated and almost intuitive, they simultaneously amplify the neural pathways associated with negative emotions[19], escalating depressive

symptoms with feelings of being trapped in persistent, overwhelming sadness[20].

In adolescents, the brain's greater neuroplasticity allows these biases to strengthen faster over time[21], making them more prone to experiencing core symptoms of depression such as sadness, hopelessness, and worthlessness[22,23]. These cognitive biases also often operate outside of conscious awareness[24], making it challenging for individuals to break free from depression[25] and reducing the effectiveness of treatments like cognitive-behavioral therapy that target negative thoughts[26,27].

Extensively studying specific biases in adolescent depression can be beneficial for improving detection rates. In current literature, depressive disorders among adolescents are largely underdetected[28], which requires a timely solution, as untreated depression is strongly associated with the development of suicidal thoughts and behaviors[29]. Hankin et al.[30] state that cognitive biases characterize various psychiatric disorders, with the content or focus of these biases being disorder-specific. Therefore, with their unconscious and disorder-specific nature, understanding negative cognitive biases and their associated neurobiological changes is essential for improving detection and developing more effective interventions.

Information Processing Models as an Intervention for Adolescent Depression

Given the multifaceted changes associated with depression and other mental health illnesses, one effective way to examine these alterations in brain structure and function is by studying how individuals process information. Cognitive scientists have proposed that an individual's negative style of information processing—encompassing their ways of noticing, interpreting, and remembering events in their lives[31]—may contribute to the development and maintenance of psychopathology, particularly depression[32]. Research shows that negative cognitive style can bias or disrupt each step of the information processing model, leading to negative information processing, contributing to depression[33].

Adolescents are particularly vulnerable to the effects of negative information processing. During adolescence, the brain undergoes significant developmental changes, particularly in areas related to emotion regulation and decision-making[34]. This period of neuroplasticity makes adolescents more susceptible to the long-term impact of negative experiences and thought patterns.

Research by Platt et al.[35] highlights the importance of understanding how negative information processing contributes to the development of depressive symptoms in adolescents. Because information processing reflects the coordinated interaction of diverse cognitive functions[36], by assessing the specific cognitive processes and the neural mechanisms through the lens of an information processing model, interventions can be designed to target and mitigate these risk factors early on[25].

Current information processing models primarily include parallel distributed processing (PDP) and serial processing frameworks[37]. PDP emphasizes the overall interaction and interconnectedness of the steps in information processing[38] and is considered to be a traditional framework for understanding information processing in adults[39]. Meanwhile, serial information processing models are focused on individualized steps and showcase a single sequence of processing operations performed at a time[40].

A variety of serial information processing models have been posed by cognitive scientists[41]. Historically, Atkinson and Shiffrin[42] came up with the first serial information processing model, and further research produced modifications that incorporated novel steps, such as thinking and reasoning[41]. Most notably, these frameworks have been used in application to understanding the mental processes of specific populations, such as adolescents and youth[43]. Current literature shows the usefulness of the serial information processing model, in particular, in understanding mental disorders and strategizing treatment interventions[44].

The well-validated nature of serial processing models in application to understanding adolescent mental health shows their potential to provide critical insights into the progression of depressive symptoms and, thus, successful treatments[45]. Of these models, the Adolescent Information Processing Model by Rice and Dolgin[43] offers a comprehensive framework to investigate cognitive and neurobiological disruptions. This information processing model, adapted to address the unique mental processes of the adolescent population, offers great potential for investigating the disruptions associated with depression in youth.

The Present Study

Previous adolescent depression research has often focused primarily on the singular stages of cognitive factors in the context of adolescent depression[46]. For example, Platt et al.[35] investigated negative attentional bias, suggesting that targeting the initial stage of information processing could disrupt the cycle of negative cognition. However, the broader role of negative information processing biases has not been thoroughly examined. These include specific cognitive distortions such as negative attentional bias, the tendency to focus excessively on negative stimuli[35]; interpretation biases, the habitual interpretation of ambiguous situations in a negative light[47]; memory biases that preferentially recall negative events[48]; and future biases that predict future events in a negatively[18]. While these biases have been studied individually, limited literature review studies have examined their effects in the context of the entire cognitive processing model, particularly in adolescents who are uniquely

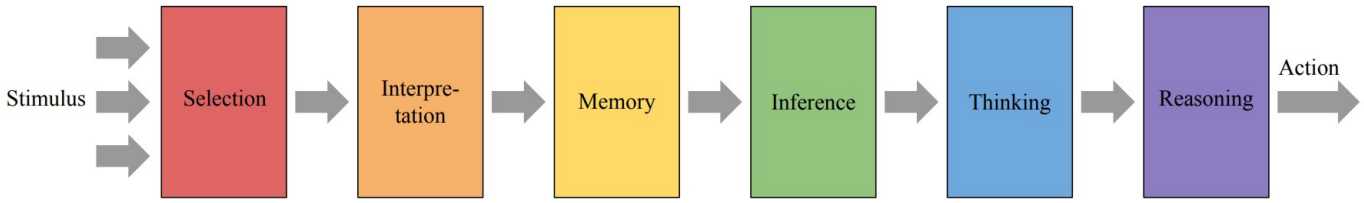

Figure 1: Adolescent Information Processing Model. Obtained from Rice & Dolgin (2005).

vulnerable to emotional and cognitive disruptions during this developmental period.

To address this gap, the present study utilizes the Adolescent Information Processing Model by Rice and Dolgin[43] to explore the cognitive and neurobiological disruptions in adolescent depression, addressing each individual stage. This model, featured in their seminal work "The Adolescent: Development, Relationships, and Culture,"[43] represents a comprehensive approach to serial processing models, updated numerous times to incorporate the latest research and theoretical advancements in adolescent development. This model evolves from existing frameworks, offering an adolescent-specific lens. Meanwhile, considering the established benefits of serial processing models, Rice and Dolgin's framework emphasizes the sequential nature of cognitive stages and their unique manifestations in adolescent depression.

This focus of the model is significant for understanding the specific ways that adolescents process negative stimuli, which may differ significantly from adult cognitive patterns due to ongoing developmental changes in the brain and heightened neuroplasticity[49].

The present study examines how disruptions across all stages of information processing contribute to the persistence of depressive symptoms through cognitive and neurobiological perspectives. While this study does not fully encompass the intricate relationships between all the cognitive and neurobiological factors, it demonstrates how disruptions in each stage contribute to a persistent negative cognitive style that exacerbates depressive symptoms in adolescents. As such, by analyzing disruptions in each stage of the Adolescent Information Processing Model (Figure 1), this paper argues that alterations in the stages of information processing are linked to depression in adolescents.

As seen in Figure 1, an informative model of information processing in adolescents developed by Rice and Dolgin[43] is presented. This model includes the following steps that lead to the execution of stimuli through action: 1) selection, 2) interpretation, 3) memory, 4) inference, 5) thinking, and 6) reasoning.

In these stages, first, various stimuli are presented to the adolescent individual, but only a select few proceed to the interpretation stage, filtered through the selection stage. Once a stimulus reaches the interpretation stage, the individual makes sense of it, drawing upon memory recall, such as past experiences with similar information. These recalled memories may influence their understanding and even lead to predictions about future outcomes. Next, in the thinking stage, individuals focus on synthesizing all the information involved in this sequence. Following this, the reasoning stage acts as a reevaluation process, allowing individuals to revisit their understanding and correct any flaws or misconceptions. Upon completing all these stages, the individual responds to the stimulus with an action, which may occur internally as thoughts and emotions or externally as observable actions.

This paper examines how each step of the proposed model is disrupted by negative cognitive biases, contributing to depression in adolescence. In response to the gap in the existing literature that frequently investigates stages in isolation, this study employs a narrative review methodology, intentionally focusing on research involving adolescents aged 9 to 18 as relevant to all stages of the information processing model. To ensure relevance and accuracy, inclusion criteria required that studies be conducted within the last 25 years, thereby limiting the scope to 21st-century research. This approach was designed to provide a comprehensive and up-to-date understanding of adolescent depression within the defined age range, creating a cohesive picture of the current state of research in this field. This paper integrates neurobiological findings to reveal structural and functional brain changes that correspond with these cognitive biases, offering deeper insights into the maintenance of adolescent depression.

In summary, examining information processing steps provides a window into the cognitive factors that contribute to the development of depression in adolescents, as well as the neurobiological changes that intensify the condition. This knowledge is essential for advancing our understanding of psychiatric disorders, optimizing treatment approaches, and improving outcomes for individuals affected by depression and related conditions.

Discussion

Selection: Negative Attentional Bias

The first stage of the information processing model is information selection[43]. Whether or not afflicted by depression, outside of conscious awareness, the brain selectively filters informa-

tion based on social norms, personal preferences, and cognitive style[50,51]. Stevens and Bavelier[52] define selective attention as "the processes that allow an individual to select and focus on particular input for processing while simultaneously suppressing irrelevant or distracting information" (pg. 30). Selection is an essential step in the information processing model because our senses are bombarded with countless stimuli that our brains simply cannot process and comprehend.

Selection is typically biased in healthy individuals due to confirmation bias[53]—where steps of information processing are tailored to confirming existing personal preference, experience, and belief[54]. However, in the case of depressed individuals, this bias shifts from merely a tendency to select information that confirms existing beliefs to a negative bias[55]. Thus, depressed individuals may have a propensity for deliberately selecting negative information[56]. This negative attentional bias[56] is defined as "the tendency of depressed individuals to focus on negative stimuli and thoughts more than healthy individuals" (pg. 1).

The dot-probe task is a common method of measuring these attentional biases in the information selection stage[57]. Hankin et al.[30] gathered 161 children aged 9 to 17, including those with MDD, anxiety, comorbid MDD and anxiety combined, and healthy controls. They used the dot-probe task to measure attentional biases by having participants quickly identify the location of a dot that appears after viewing pairs of emotional and neutral faces, with faster response times indicating preferential attention to the emotional stimuli. This study revealed that youth with MDD oriented more quickly to sad faces, indicating preferential selective attention to sad stimuli and thus demonstrating a skewed prioritization of negative information.

Another study using a dot-probe measure of attentional bias with 105 adolescents aged 13 to 17 proposed a significant association between negative attentional bias and increased symptoms of depression[35]. Findings indicating a causal relationship between negative attentional bias and the severity of depression highlight the distinct role of the altered selection stage within the information processing model in contributing to depression.

Using a more precise methodology—an emotional visual search to examine how each person sustains attention on emotional stimuli while managing distractions[58], alongside a dot-probe task—researchers tested negative attentional bias in a sample of 681 adolescents. With higher levels of depression displaying higher negative attentional bias scores[46], this study demonstrated that depressive symptoms are correlated to negative attention bias, revealing its direct potential association with aggravating depression.

Negative attentional bias intensifies adolescent depression by not only prioritizing negative information but also diminishing attention to positive stimuli, thereby accelerating its progression. In one study of 26 adolescent girls, participants gave a speech in front of both a critical and positive judge while their eye movements were tracked[59]. In contrast to healthy participants, girls with higher depressive symptoms looked at the potentially critical judge for a longer period of time. These findings support that adolescent depressive symptoms are correlated with sustained attention toward critical evaluation and exclusion of positive evaluation[59], which clearly demonstrates the extensive role of negative attentional bias in the progression of adolescent depression.

These research findings convey that negative stimuli in adolescents with depression are not only prioritized but actively reinforced. Evidence highlights how depression disrupts the selection stage of the information processing model, interfering with adaptive responses to the environment.

Negative Attentional Bias: White Matter Reduction

This negative attentional bias, as applied to the information processing model, shows clear links to the neurobiological mechanisms of sustained adolescent depression. In cases of persistent depression resulting from repeated use and deep integration of negative attentional bias, research shows co-occurring neurobiological changes in adolescent patients[60].

White matter, in particular, has been observed to play a critical role in the filtration of irrelevant or unwanted information by supporting the connectivity between the visual cortex and the parietal cortex, allowing individual attentional selectivity[61]. In adolescents with depression, this normative neurobiological process is disrupted[60]. Barch et al.[62] demonstrated in a recent diffusion tensor imaging study that depressed adolescents showcased the emergence of decreased white matter in regions key for information processing—potentially reflecting altered neurobiology that further contributes to the progression of depression.

Such findings illustrate the connection between cognitive biases and structural brain changes, emphasizing that dysfunctions in attention have clear correlations to depression in adolescents. As denoted by the white matter reduction in adolescent depression, neurobiological changes related to attentional bias serve as a compounding factor that disrupts the selection stage of the information processing model.

By prioritizing negative stimuli and neglecting positive or neutral information, adolescents experience increased depressive symptoms. Neurobiological evidence additionally conveys how these attentional biases are associated with structural changes, further impairing one's ability to filter stimuli and thus reinforcing depression. These insights show the significance of understanding the selection stage of the Adolescent Information Processing Model in shaping adolescent depression.

Interpretation: Negative Interpretative Bias

The second stage of the information processing model is interpretation[43]. After the selection of stimuli, the information advances to the actual processing stages, where the individual interprets

and assigns meaning to it by drawing on past memories, existing beliefs, and other cognitive schemes[63]. Like any other stages of information processing, personal biases inevitably influence interpretation[53].

As for depressed adolescents, they not only exhibit personal biases but also show a consistent tendency to interpret ambiguous information more negatively than others. This tendency is called negative interpretative bias[64], and is seen to be prevalent in adolescents with depression. In one study, for example, Orchard and Reynolds[65] assessed interpretative bias scores through the Ambiguous Scenarios Test (AST), where adolescents aged 12 to 18 verbally described how they interpreted an ambiguous scenario. Comparing depressed and non-depressed clinical groups, clinically referred adolescents with depression tended to interpret ambiguous scenarios more negatively compared to healthy controls. This finding highlights that negative interpretative bias is uniquely associated with the interpretation stage in adolescents with depression.

Similarly, Sfärlea et al.[66] employed both the AST and the Scrambled Sentences Task (SST) to measure explicit and implicit negative interpretative biases in adolescents diagnosed with major depression and those identified as high-risk as children of depressed parents. They found that negative interpretative bias existed in both groups, including those without a diagnosis, showing that negative interpretive bias predates the diagnosis of adolescent depression. These results revealed the potential for interpretative bias to be a contributor to the disorder.

Additional research shows that these negative interpretative biases in depressed adolescents also significantly affect how they perceive ambiguous scenarios as they extend to their sense of self-perception[67]. Low self-esteem, a facet of self-interpretation, is a prevalent issue among adolescents with depression, as indicated by studies linking self-esteem levels to depressive symptoms[68]. One study explored how training individuals to focus on either negative or positive social outcomes could impact their self-image[69]. The study found that adolescents trained to see negative outcomes developed a significantly more negative self-image, whereas those trained to see positive outcomes improved their self-perception. This indicates a clear correlation between interpretative biases and self-interpretation.

For instance, Ibrahim et al.[4] conducted a study involving 461 students aged 13 to 17, where the Rosenberg Self-esteem Scale (RSE) was used to assess self-esteem. The findings showed that 47.5% of adolescents with low self-esteem experienced depression, contrasting with only 2% of those with high self-esteem, suggesting an association between depressive symptoms and self-esteem. Meanwhile, Shah et al.[70] examined 600 students aged 12 to 18 using both the RSE and the Beck Depression Inventory Scale, and findings revealed an inverse relationship between self-esteem scores and depressive symptoms. These results underscore the critical role of self-esteem in adolescent mental health, providing insight into how negative interpretative bias coexists with depression.

Negative Interpretative Bias: Abnormal Precuneus

Negative interpretative bias not only perpetuates adolescent depression, but is also closely linked to neurobiological changes that heighten the disorder. From a neurobiological perspective, negative self-perception, or self-interpretation, is linked to abnormal precuneus that leads to increased connectivity to the prefrontal cortex and short-term memory systems, which are areas involved in ruminative processes associated with low self-esteem[71].

In a study involving 32 unmedicated patients with early-onset chronic depression and 40 healthy controls, Rubart et al.[72] found significant differences in brain connectivity patterns. Healthy controls exhibited robust connectivity between the precuneus and right pre-supplementary motor area, whereas individuals with depression showed weaker connectivity between the subcallosal anterior cingulate and the precuneus. As the subcallosal cingulate cortex is known for its role in regulating emotional behavior and feelings of sadness[73], and the precuneus is crucial for self-processing operations[74], the weaker connectivity may reflect impaired self-interpretation, particularly in relation to emotional regulation.

Another study highlighted differences in precuneus activity during self-judgements between control and depressed adolescents[75]. Adolescents with depression exhibited increased activity in the precuneus and posterior cingulate cortex (PCC) regions, while healthy adolescents showed deactivations in these areas. This suggests that the brain's heightened activation in the precuneus and PCC are linked to negative self-judgements, a maladaptive self-interpretation, and are linked to existing states of depression.

Within the framework of the Adolescent Information Processing Model, the interpretation stage involves assigning meaning to stimuli using past experiences, beliefs, and biases. Negative self-interpretation, an outcome of negative interpretative bias, is seen to potentially play a contributing role alongside the abnormal neurobiological function of the precuneus.

Memory: Negative Memory Bias

Following the stage of interpretation is memory, the process of recalling past experiences[43]. Through memory, humans, as empirical beings, depend on past experiences to make sense of the world[76].

Generally, people tend to prioritize the retrieval of negative memories over positive ones[77]. This tendency is particularly pronounced in individuals with depression, who exhibit a heightened form of this prioritization known as negative memory bias[78]. Research has shown a direct correlation between the

severity of depression symptoms and the extent of negative memory bias[79].

A study involving 578 adolescents aged 11 to 15 found that those who recalled more self-referential negative words and fewer self-referential positive words exhibited more severe depressive symptoms[80]. This result demonstrates the clear association between negative memory bias and mental health symptoms.

Negative memory bias is also shaped by the tendency of depressed adolescents to disproportionately store negative memories, creating a memory pool that is predominantly negative[81]. This was evidenced by research conducted with 155 adolescents aged 12 to 16, which measured memory forgetfulness using the item-method directed forgetting paradigm[82]. Findings showed that participants with higher depression scores exhibited greater forgetfulness of positive memories. This highlights that negative memory bias extends beyond the prioritization of memories during retrieval but is rooted in a cognitive impairment that selectively stores negative experiences.

In addition, adolescents with depression also tend to interpret their memories more negatively during recall. One study shows that adolescents diagnosed with MDD are prone to recalling distorted or false memories, particularly negative lures —irrespective of whether the original information was negative, neutral, or positive[83]. This abnormal pattern of memory recall and interpretation is evident in individuals with depression, as commonly experienced symptoms include memory deficits and loss[84]. This suggests that memory processing in individuals with MDD is skewed, characterized by deviations and atypical patterns.

Negative Memory Bias: Hippocampal Imbalance

The cognitive impairments observed in negative memory bias are closely linked to neurobiological changes, particularly in the hippocampus, a brain critical for memory formation and retrieval. The hippocampus is a vital part of the human brain, in charge of both the formation and retrieval of memories, and its involvement in both depression and memory is well demonstrated in human neuroimaging studies[85].

For example, MacMaster and Kusumakar[86] found that there was a significant difference in the left hippocampus compared to the right when examining the hippocampal structures of 17 patients with MDD—13 to 18 years old—with 17 healthy controls. The left hippocampus is in charge of episodic verbal memory—subjective information that can be interpreted in many different ways—while the right hippocampus oversees spatial memory—information that is far more objective[87]. These findings suggest that memories open to various interpretations are more likely to be distorted and viewed through a highly subjective lens in individuals with depression compared to healthy individuals. Consequently, adolescents with depression often experience increased significant alterations in specific regions of the brain, affecting their subjective recollections.

Negative memory bias, occurring within the memory stage of the Adolescent Information Processing Model, contributes to adolescent depression. Furthermore, hippocampal imbalances compound these impairments by altering the brain's structural ability to process memories effectively, leading to persisting symptoms of depression.

Inference: Negative Future Bias

The fourth stage of the Adolescent Information Processing Model is inference, which involves making future predictions based on interpreted information[43]. This stage enables complex analysis of stimuli, generating insights, and anticipating potential outcomes[88]. Future predictions are essential to human life, with positive anticipation of the future serving as a driving force for survival and development[89]. In itself, many adolescent studies have accounted for positive anticipation towards the future behind student motivation and development of youth[90–92]. However, in depressed youth, future predictions are often negatively skewed, a phenomenon frequently referred to as negative future bias[93].

This negatively oriented future thinking is associated with a sense of hopelessness, a key factor in suicidality in depression[94]. Horwitz et al.[95] define hopelessness as "a set of cognitive schemas oriented toward negative views/expectations about the future" (pg. 169). Identifying hopelessness early on is crucial, given that more than 90% of youth who die by suicide have been found to be diagnosed with at least one mental illness[6]. Thus, it is essential to understand this tendency towards negatively oriented future thinking in depression. Hopelessness plays a profound role in shaping cognitive schemas and contributing to the heightened risk of suicidal behavior among adolescents.

Research demonstrates the prevalence of negative future bias in depressed adolescents, where their future-oriented thoughts become predominantly negative, less vivid, and accompanied by reduced motivation[18]. A longitudinal study conducted with 19 adolescents aged 16 to 19, all with a history of clinical or subclinical depression, explored how mental health struggles impacted their future-oriented thinking[96]. While engaging in future-related thoughts during periods of poor mental health, participants reported a dominance of negative thoughts about the future, reinforcing their feelings of despair.

Further, in a review paper of 22 longitudinal studies[18], authors identified negative future bias as a significant predictor of adolescent depression, with a direct correlation between higher negative future bias and the severity of depressive symptoms. These findings reinforce the link between negative future bias and depression in adolescents, shedding light on how this cognitive alteration may contribute to the persistence of depression.

Negative Future Bias: Prefrontal Cortex Dysfunction

Neurobiological alterations also account for the disruptions observed in the inference stage, which compound the symptoms of depression. One research study, for example, conducted on 94 adolescents with a mean age of 15.2 years used functional magnetic resonance imaging (fMRI) scans to compare the neural activity between the two groups [97]. Results revealed lower neural value signaling in the prefrontal cortex (PFC) in adolescents with higher depressive symptoms compared to their healthy counterparts. As such, when the PFC becomes abnormally dysfunctional, emotions can become hyperactive and out of control, often exacerbating depression [98]. PFC dysfunction is especially detrimental, as the anterior lateral PFC plays a vital role in estimating future success [99] and the lateral PFC in regulating and keeping negative emotions under control—showing a clear connection between neurobiological alterations and disruptions in the inference stage of information processing.

Similarly, a review paper examined 28 fMRI studies of adolescents aged 13 to 18 with depression, revealing disruptions in the medial prefrontal cortex (mPFC), a critical region within the PFC responsible for self-referential thinking [100]. The mPFC is also linked to emotional regulation in future scenarios, and its dysfunction can impair adolescents' ability to think about and emotionally process future events, a key function impacting depression [101]. This neurobiological evidence further heightens the depressive symptoms resulting from the negatively impaired inference stage in the Adolescent Information Processing Model.

Thinking: Rumination

Thinking is the fifth stage of the information processing model, where individuals deeply engage with memories recalled during the memory stage [43]. Individuals reflect on and scrutinize these memories, extracting meaning and understanding past experiences.

Persistent negative feelings are one of the most common symptoms of depression in adolescents [30]. Rumination is a key characteristic of thinking in depressed individuals, reinforcing these negative sensations [96,102]. In fact, individuals with depression tend to stay alone and isolated, which leads them to linger in their thoughts longer, amplifying the impacts of rumination [103].

Rumination is defined as "focusing passively and repetitively on one's symptoms of distress" [104] (pg. 216). For individuals experiencing depression, their recall of memories tends to focus primarily on negative ones. These negative memories act as triggers, leading to repetitive dwelling. Individuals with depression struggle to disengage from negative stimuli [105], becoming stuck in a loop of replaying distressing memories. This inability to move on perpetuates rumination, turning thinking into a continuous cycle of negativity.

Adolescents with depression are particularly susceptible to rumination, which reinforces negative emotions and amplifies their depressive symptoms. For instance, Hankin [106] studied 350 teenagers and discovered that greater rumination levels were predicted. Repeated negative thinking patterns reinforce a cycle of rumination, continuously dwelling on negative thoughts and emotions without finding solutions. This cycle amplifies negative neural pathways, heightening the risk of depression. Repeated thinking of memories can solidify them into long-term memories, making them more likely to recur [107].

In a similar vein, but with a larger sample size and a greater accuracy, Wilkinson et al. [108] tracked the symptoms and severity of depression in 658 healthy teenagers at elevated risk for psychopathology over the course of a year. High levels of rumination were found to be a significant predictor of the emergence of depression during the follow-up period. This emphasizes the importance of rumination as a critical cognitive susceptibility component in adolescent depression, where repeated engagement with negative thoughts strengthens neural pathways reinforcing depression.

Rumination: Abnormal Default Mode Network

In severe depression, rumination becomes deeply automatic. In the context of MDD, rumination is often seen as a self-referential thinking style linked to disruptions in the brain's Default Mode Network (DMN) [109]. The DMN consists of various brain regions, including the mPFC, medial and lateral parietal cortex, and temporal lobe [110]. This network is primarily responsible for mind wandering and task-unrelated thoughts, such as focusing on memories and processing secondary information elicited during the interpretation stage [111].

In adolescents with MDD, disruptions in the DMN contribute to inflexibly elevated connectivity, as observed in a study by Ho and her colleagues [112]. Using fMRI to measure functional connectivity within the DMN in 26 medication-free adolescents with MDD (aged 13 to 17) and 37 healthy controls, the study revealed that these disruptions exacerbate persistent negative thought patterns, such as rumination, exacerbating symptoms of depression. Thereby, neurobiological abnormality further exemplifies evidence of rumination, unique to the thinking stage of the Adolescent Information Processing Model in depression.

Reasoning: Cognitive Impairments

Reasoning is the sixth stage in the Adolescent Information Processing Model [43]. In this stage of the information processing model, individuals reevaluate the information that they have processed.

It is generally understood that depression can impair rational thinking and lead individuals to become more emotionally driven, which may affect their ability to think logically [113]. Irrational beliefs are a known risk factor for depression [114], demonstrating that depression alters individuals' ability to validate and

re-evaluate the information they have processed. Hu et al.[115] asserted that "depression has a negative effect on reasoning in adolescents" (pg. 5).

These authors supported this assertion by assessing the reasoning abilities of 1,961 Chinese adolescents aged 12 to 18 with depression, revealing a significant negative impact of depression on their cognitive reasoning abilities. These findings further confirm the association between cognitive impairments in reasoning and adolescent depression.

Cognitive Impairments: Hemispheric Imbalance

The limited rationality of individuals with depression's minds is further aggravated by neurobiological alterations, particularly through the hemispheric imbalances. Specifically, depression is associated with an overactive right hemisphere and a relatively underactive left hemisphere[116]. The left hemisphere governs quantitative and analytical functions, while the right hemisphere is responsible for intuition and creativity, leading to a more subjective experience of the world[117]. This interplay underscores how neurobiological imbalances contribute to diminished rationality in individuals with depression, resulting in altered reasoning.

While little is known about the hemisphere imbalance linked to adolescent depression, research on the corpus callosum has provided significant new knowledge. The corpus callosum is a crucial structure that sits between the two hemispheres of the brain and plays a major role in effective signaling and communication between them[118]. MacMaster et al.[119] discovered that adolescents with depression have a smaller corpus callosum compared to their healthy peers. This finding, based on magnetic resonance imaging measurements of the corpus callosum volume in 16 patients with MDD aged 14 to 18, compared with age- and sex-matched healthy individuals, highlights a limited connection between hemispheres. When the corpus callosum is smaller, the brain tends to process information through the stronger side[120].

Adolescence is inherently guided by emotions, as evidenced by both behavioral observations and brain development science[121]. This emotional orientation likely results in information being processed predominantly by the right hemisphere, which is stronger in many adolescents[122]. Consequently, the limited rationality observed in adolescents, including those who are healthy, may lead to flawed or overly emotional responses that they fail to recognize when re-evaluating processed information.

In adolescents with depression, these tendencies are amplified by cognitive and neurobiological impairments, such as imbalances and reduced inter-hemispheric communication. These impairments interact within the reasoning stage of the Adolescent Information Processing Model, compounding difficulties in re-evaluating processed information and, thus, contributing to depressive symptoms in adolescents.

Conclusion

This review paper underscores the critical lens Adolescent Information Processing Model offers in the context of examining the cognitive and neurobiological foundations of depression. By dissecting the Adolescent Information Processing Model, this paper has discussed how cognitive biases disrupt each stage—selection, interpretation, memory, inference, thinking, and reasoning.

These disruptions not only lead to a persistent negative cognitive style, but are also compounded by neurobiological alterations that heighten depressive symptoms in adolescents. Such neurobiological insights provide a more comprehensive understanding of how cognitive biases are not merely psychological phenomena but are deeply rooted in the brain's structure and functioning.

This narrative review, however, is not without limitations. Adolescent depression is not merely a mental disorder rooted in cognitive and neurobiological factors; it involves a broader, more complex interplay of influences, including emotional, sociocultural, mood regulation, and external stressors[123]. This narrow focus limits the generalizability of the findings, suggesting a need for future studies to adopt a more comprehensive approach, like integrating emotional and social factors to deepen insights into adolescent depression. For example, studies could explore how cultural norms or family stressors interact with cognitive thinking styles or neurobiological alterations to influence depressive symptoms in the context of the Adolescent Information Processing Model.

Furthermore, the present study's reliance on a localized and sequential analysis of cognitive and neurobiological alterations within a serial information processing model reveals inherent limitations. This approach does not sufficiently account for the interconnectedness and dynamic interplay among these factors. The disruptions in each stage of information processing are discussed as linear and discrete; however, the interactions between cognitive biases and neurobiological changes are likely more fluid and cumulative. For example, the cognitive biases that emerge at one stage may not only persist but also interact with neurobiological alterations in ways that exacerbate depressive symptoms at later stages. Future research should furthermore consider an approach that explores these complex interactions across the entire model. Longitudinal studies, examining how disruptions in one cognitive stage influence subsequent stages, and how neurobiological changes progress simultaneously, on populations of ages 9 to 18 would provide a more nuanced and integrative perspective on adolescent depression. By combining diverse methodologies, such as mixed-methods designs that incorporate both qualitative and quantitative data, additional research can capture not only the measurable cognitive and neurobiological changes, but also the experiential social, emotional, and environmental aspects of adolescents' lives.

Furthermore, while literature established a clear explanation of how neurobiological alterations worsen depressive symptoms in each stage of the Adolescent Information Processing Model, there is limited information available about the causal relationship between these alterations and impaired cognitive processing. For example, it continues to be relatively unclear whether neurobiological changes precede cognitive biases, if impaired cognitive processes themselves lead to neurobiological changes, or if pre-existing neurobiological conditions cause adolescents to be more susceptible to developing impaired cognitive processes. Additional longitudinal studies that track adolescents over a period of time may be valuable in better understanding this relationship. Such studies could reveal the mechanisms that drive the onset and progression of depressive symptoms and help identify potential biomarkers for early detection and intervention.

Early identification and intervention are paramount in mitigating the progression of depression in adolescents. Cognitive-behavioral therapies (CBT), which specifically target negative cognitive biases, have shown promise in disrupting these harmful cognitive cycles [124]. By addressing these biases early, CBT can promote healthier cognitive patterns, improve mental health outcomes, and reduce the risk of severe consequences such as suicide [125].

This present study, which examines stage-specific cognitive and neurobiological changes through the lens of the Adolescent Information Processing Model, provides valuable insights that could further refine interventions. For instance, understanding how negative biases manifest in distinct stages—such as selection, interpretation, and memory—can inform more targeted, stage-specific therapeutic strategies. Tailored CBT modules designed to address specific cognitive disruptions, can significantly enhance treatment effectiveness [126].

Moreover, integrating the study's findings into early detection programs could help identify at-risk adolescents before symptoms become severe. School-based mental health initiatives could incorporate assessments to evaluate stage-specific cognitive vulnerabilities and provide early, personalized support. Similarly, neurobiological findings could inform the development of biomarkers for depression, aiding in early diagnosis and monitoring treatment progress.

By addressing cognitive vulnerabilities early, we can foster resilience, enhance mental health, and improve the overall well-being of young people. This comprehensive approach is essential for ensuring that adolescents navigate this critical developmental period with greater psychological strength and stability, laying the foundation for healthier adult lives. The significance of this research lies in its potential to transform how we understand, prevent, and treat adolescent depression, offering hope for a brighter, more resilient future for our youth.

References

1 N. Kaneshiro, *Adolescent development*, https://medlineplus.gov/ency/article/002003.htm.

2 R. Frankel, *The Adolescent Psyche*.

3 A. Thapar, S. Collishaw, D. Pine and A. Thapar, *Depression in adolescence*.

4 M. Ibrahim, W. Wan Ismail, N. Nik Jaafar, U. Mohd Mokhtaruddin, H. Ong, N. Abu Bakar and H. Mohd Salleh Sahimi, *Depression and Its Association With Self-Esteem and Lifestyle Factors Among School-Going Adolescents in Kuala Lumpur, Malaysia*.

5 I. Rădulescu, A. Drăgoi, S. Trifu and M. Cristea, *Neuroplasticity and depression: Rewiring the brain's networks through pharmacological therapy*.

6 E. Galaif, S. Sussman, M. Newcomb and T. Locke, *Suicidality, depression, and alcohol use among adolescents: A review of empirical findings*.

7 *Highlights for the 2021 National Survey on Drug Use and Health*, https://www.samhsa.gov/data/sites/default/files/2022-12/2021NSDUHFFRHighlights092722.pdf.

8 V. Zonca, *Preventive strategies for adolescent depression: What are we missing? A focus on biomarkers*.

9 *Depression and Suicide*, https://www.hopkinsmedicine.org/health/conditions-and-diseases/depression-and-suicide#:~:text=Most%20people%20who%20commit%20suicide,or%20a%20substance%20use%20disorder.

10 B. Platt, A. Sfärlea, J. Löchner, E. Salemink and G. Schulte-Körne, *The role of cognitive biases and negative life events in predicting later depressive symptoms in children and adolescents*.

11 M. Rot, S. Mathew and D. Charney, *Neurobiological mechanisms in major depressive disorder*.

12 F. Zhang, W. Peng, J. Sweeney, Z. Jia and Q. Gong, *Brain structure alterations in depression: Psychoradiological evidence*.

13 C. Keysers and V. Gazzola, *Hebbian learning and predictive mirror neurons for actions, sensations and emotions*.

14 D. Dow-Edwards, F. MacMaster, B. Peterson, R. Niesink, S. Anderson and B. Braams, *Experience during adolescence shapes brain development: From synapses and networks to normal and pathological behavior*.

15 *mechanistic insight*.

16 X. Fan, M. Mocchi, B. Pascuzzi, J. Xiao, B. Metzger, R. Mathura, C. Hacker, J. Adkinson, E. Bartoli, S. Elhassa, A. Watrous, Y. Zhang, A. Allawala, V. Pirtle, S. Mathew, W. Goodman, N. Pouratian and B. K. R, *Brain mechanisms underlying the emotion processing bias in treatment-resistant depression*.

17 L. Hilt and S. Pollak, *Getting Out of Rumination: Comparison of Three Brief Interventions in a Sample of Youth*.

18 P. Tang, K. Kostyrka-Allchorne, A. Butura, J. Phillips-Owen and E. Sonuga-Barke, *Reciprocal developmental pathways between future-related thinking and symptoms of adolescent depression and anxiety: A systematic review and meta-analysis of longitudinal studies*.

19 S. Disner, C. Beevers, E. Haigh and A. Beck, *Neural Mechanisms of the Cognitive Model of Depression*.

20 S. Chand and H. Arif, *Depression*, https://www.ncbi.nlm.nih.gov/books/NBK430847/.

21 K. Konrad, C. Firk and P. Uhlhaas, *Brain Development During Adolescence*.

22 J. Roiser, R. Elliott and B. Sahakian, *Cognitive Mechanisms of Treatment in Depression*.

23 Y. Toender, L. Velzen, I. Heideman, B. Harrison, C. Davey and L. Schmaal, *Neuroimaging predictors of onset and course of depression in childhood and adolescence: A systematic review of longitudinal studies*.

24 I. Gotlib and D. Neubauer, *Information-Processing Approaches to the Study of Cognitive Biases in Depression*.

25 A. Wenzel and A. Beck, *A cognitive model of suicidal behavior: Theory and treatment*.

26 S. Nolen-Hoeksema, V. Vine and K. Gilbert, *Rumination and emotions*, http://www.scopus.com/inward/record.url?scp=84932615604andpartnerID=8YFLogxK.

27 A. Pucci, *When Cognitive-Behavioral Therapy Fails: The Five Most Common Reasons*, https://www.nacbt.org/when-cognitive-behavioral-therapy-fails-the-five-most-common-reasons/.

28 E. Twivy, M. Kirkham and M. Cooper, *The lived experience of adolescent depression: A systematic review and meta-aggregation*.

29 G. Maslow, K. Dunlap and R. Chung, *Depression and Suicide in Children and Adolescents*.

30 B. Hankin, B. Gibb, J. Abela and K. Flory, *Selective Attention to Affective Stimuli and Clinical Depression Among Youths: Role of Anxiety and Specificity of Emotion*.

31 J. Margolis, S. May-Varas and T. Mead, *Educational Learning Theories 3rd Edition*, https://openoregon.pressbooks.pub/educationallearningtheories3rd/front-matter/introduction/.

32 I. Gotlib and J. Joormann, *Cognition and Depression: Current Status and Future Directions*.

33 C. Beevers, M. Mullarkey, J. Dainer-Best, R. Stewart, J. Labrada, J. Allen, J. McGeary and J. Shumake, *Association Between Negative Cognitive Bias and Depression: A Symptom-Level Approach*.

34 J. Silvers, *Adolescence as a pivotal period for emotion regulation development*.

35 B. Platt, S. Murphy and J. Lau, *The Association Between Negative Attention Biases and Symptoms of Depression in a Community Sample of Adolescents*.

36 E. Roy, *Cognitive Factors*.

37 W. Verwey, C. Shea and D. Wright, *A cognitive framework for explaining serial processing and sequence execution strategies*.

38 J. Townsend and M. Fifić, *Parallel versus serial processing and individual differences in high-speed search in human memory*.

39 D. Rumelhart, G. Hinton and J. McClelland, *A General Framework for Parallel Distributed Processing*, https://stanford.edu/~jlmcc/papers/PDP/Volume%201/Chap2_PDP86.pdf.

40 *serial processing*, https://dictionary.apa.org/serial-processing.

41 J. Jarecki, J. Tan and M. Jenny, *A framework for building cognitive process models*.

42 R. Atkinson and R. Shiffrin, *Human Memory: A Proposed System and its Control Processes*.

43 F. Rice and K. Dolgin, *The adolescent: Development, relationships and culture*.

44 D. Ahorsu, K. Chung, H. Wong, M. Yiu, Y. Mok, K. Lei and T. H. H, *The Effects of Major Depressive Disorder on the Sequential Organization of Information Processing Stages: An Event-Related Potential Study*.

45 D. Brinker, Y. Zhou, M. Callejas and E. MacGeorge, *Increasing Information Seeking About Antibiotic Risks: Testing a Clinical Intervention Message Using the Risk Information Seeking and Processing Model*.

46 A. Klein, L. Voogd, R. Wiers and E. Salemink, *Biases in Attention and Interpretation in Adolescents With Varying Levels of Anxiety and Depression*.

47 J. Joormann, C. Waugh and I. Gotlib, *Cognitive Bias Modification for Interpretation in Major Depression: Effects on Memory and Stress Reactivity*.

48 S. Grant, A. Huskey, J. Faunce and B. Friedman, *Chapter 8 - Negative memory biases in health and psychiatric disorders*.

49 J. Kanwal, Y. Jung and M. Zhang, *Brain Plasticity during Adolescence: Effects of Stress, Sleep, Sex, and Sounds on Decision Making*.

50 T. DeAngelis, *Psychologists are taking aim at misinformation with these powerful strategies*.

51 P. Agarwal, *Sway: Unravelling Unconscious Bias*, https://www.bloomsbury.com/uk/sway-9781472971388/.

52 C. Stevens and D. Bavelier, *The Role of Selective Attention on Academic Foundations: A Cognitive Neuroscience Perspective*.

53 D. Vedejová, *Confirmation bias in information search, interpretation, and memory recall: evidence from reasoning about four controversial topics*.

54 C. McAndrew and J. Gore, *Understanding Preferences in Experience-Based Choice: A Study of Cognition in the 'Wild'*.

55 T. Kube, J. Glombiewski, J. Gall, L. Touissant, T. Gärtner and W. Rief, *How to modify persisting negative expectations in major depression? An experimental study comparing three strategies to inhibit cognitive immunization against novel positive experiences*.

56 A. Mennen, K. Norman and N. Turk-Browne, *Attentional Bias in Depression: Understanding Mechanisms to Improve Training and Treatment*.

57 N. Thigpen, L. Gruss, S. Garcia, D. Herring and A. Keil, *What does the dot-probe task measure? A reverse correlation analysis of electrocortical activity*.

58 M. Calvo, L. Nummenmaa and P. Avero, *Visual Search of Emotional Faces*.

59 M. Woody, D. Rosen, K. Allen, R. Price, E. Hutchinson, M. Amole and J. Silk, *Looking for the negative: Depressive symptoms in adolescent girls are associated with sustained attention to a potentially critical judge during in vivo social evaluation*.

60 H. Li, D. Wei, J. Sun, Q. Zhang and J. Qiu, *Fronto-Limbic Alterations in Negatively Biased Attention in Young Adults with Subthreshold Depression*.

61 C. Seiler, K. Jones, D. Shera and C. Armstrong, *Brain Region White Matter Associations With Visual Selective Attention*.

62 D. Barch, X. Hua, S. Kandala, M. Harms, A. Sanders, R. Brady, R. Tillman and J. Luby, *White Matter Alterations Associated With Lifetime and Current Depression in Adolescents: Evidence for Cingulum Disruptions*.

63 J. Zepp, C. Dubé and D. Melcher, *A direct comparison of central tendency recall and temporal integration in the successive field iconic memory task*.

64 C. Berna, T. Lang, G. Goodwin and E. Holmes, *Developing a Measure of Interpretation Bias for Depressed Mood: An Ambiguous Scenarios Test*.

65 F. Orchard and S. Reynolds, *The Combined Influence of Cognitions in Adolescent Depression: Biases of Interpretation, Self-Evaluation, and Memory*.

66 A. Sfärlea, C. Buhl and B. Platt, *'I Am a Total... Loser' – The Role of Interpretation Biases in Youth Depression*.

67 A. Barkai and N. Rappaport, *A Psychiatric Perspective on Narratives of Self-Reflection in Resilient Adolescents*.

68 M. Masselink, E. Roekel and A. Oldehinkel, *Self-Esteem in Early Adolescence as Predictor of Depressive Symptoms in Late Adolescence and Early Adulthood: The Mediating Role of Motivational and Social Factors*.

69 C. Hirsch, A. Mathews and D. Clark, *Inducing an Interpretation Bias Changes Self-Imager: A Preliminary Investigation*.

70 S. Shah, F. Dhaheri, A. Albanna, N. Jaberi, S. Eissaee, N. Alshehhi, S. Shamisi, M. Hamez, S. Abdelrazeq, M. Grivna and T. Betancourt, *Self-Esteem and Other Risk Factors for Depressive Symptoms Among Adolescents in United Arab Emirates*.

71 W. Cheng, E. Rolls, H. Ruan and J. Feng, *Functional Connectivity of the Precuneus in Unmedicated Patients With Depression*.

72 A. Rubart, B. Zurowski, I. Veer, D. Schön, M. Göttlich, J. Klein, E. Schramm, J. Wenzel, C. Haber, D. Schoepf, J. Sommer, C. Konrad, K. Schnell and H. Walter, *Precuneus Connectivity and Symptom Severity in Chronic Depression*.

73 B. Dunlop, J. Rajendra, E. Craighead, M. Kelley, C. McGrath and K. Choi, *Functional Connectivity of the Subcallosal Cingulate Cortex and Differential Outcomes to Treatment With Cognitive-Behavioral Therapy or Antidepressant Medication for Major Depressive Disorder*.

74 A. Cavanna and M. Trimble, *The Precuneus: A Review of Its Functional Anatomy and Behavioural Correlates*.

75 K. Bradley, S. Colcombe, S. Henderson, C. Alonso, M. Milham and V. Gabbay, *Neural correlates of self-perceptions in adolescents with major depressive disorder*.

76 G. Malcolm, I. Groen and C. Baker, *Making Sense of Real-World Scenes*.

77 S. Williams, J. Ford and E. Kensinger, *The Power of Negative and Positive Episodic Memories*, https://doi.org/10.3758/s13415-022-01013-z.

78 J. Vrijsen, C. Amen, B. Koekkoek, I. Oostrom, A. Schene and I. Tendolkar, *Childhood Trauma and Negative Memory Bias as Shared Risk Factors for Psychopathology and Comorbidity in a Naturalistic Psychiatric Patient Sample*.

79 F. Duyser, P. Eigndhoven, M. Bergman, R. Collard, A. Schene, I. Tendolkar and J. Vrijsen, *Negative Memory Bias as a Transdiagnostic Cognitive Marker for Depression Symptom Severity*.

80 J. Bone, G. Lewis, J. Roiser, S. Blakemore and G. Lewis, *Recall Bias During Adolescence: Gender Differences and Associations With Depressive Symptoms*.

81 S. Connolly and L. Alloy, *Negative Event Recall as a Vulnerability for Depression: Relationship between Momentary Stress-Reactive Rumination and Memory for Daily Life Stress*.

82 S. Ho, J. Cheng, D. Dai, T. Tam and O. Hui, *The effect of positive and negative memory bias on anxiety and depression symptoms among adolescents*.

83 J. Joormann, B. Teachman and I. Gotlib, *Sadder and Less Accurate? False Memory for Negative Material in Depression*.

84 J. Yin, A. John and D. Cadar, *Bidirectional Associations of Depressive Symptoms and Cognitive Function Over Time*.

85 B. Wiltgen, M. Zhou, Y. Cai, S. Parivash, W. Li and A. Silva, *Hippocampal Memory Trace and the Neural Mechanisms of Memory Persistence*.

86 F. MacMaster and V. Kusumakar, *Hippocampal Volume in Early Onset Depression*.

87 A. Ezzati, M. Katz, A. Zommit, M. Lipton, M. Zimmerman, M. Sliwinski and R. Lipton, *Differential Association of Left and Right Hippocampal Volumes With Verbal Episodic and Spatial Memory in Older Adults*.

88 J. Tarieh, *Feelings of inadequacy: The relationships between overthinking and anxiety*.

89 H. Engberg, B. Jeune and K. Christensen, *Optimism and survival: does an optimistic outlook predict better survival at advanced ages? A twelve-year follow-up of Danish nonagenarians*.

90 S. Stoddard and J. Pierce, *Promoting Positive Future Expectations During Adolescence: The Role of Assets*.

91 K. Schmid and S. Lopez, *Chapter 4 - Positive pathways to adulthood: The role of hope in adolescents' constructions of their futures*.

92 J. Husman and W. Lens, *The role of the future in student motivation*.

93 P. Greene, A. Latham and K. Miller, *One Preferring that Overall, Things are Worse: Future-Bias and Unequal Payoffs*.

94 J. Reeve, *Understanding Motivation and Emotion*.

95 A. Horwitz, J. Berona, E. Czyz, C. Yeguez and C. King, *Positive and Negative Expectations of Hopelessness as Longitudinal Predictors of Depression, Suicidal Ideation, and Suicidal Behavior in High-Risk Adolescents*.

96 P. Tang, G. Pavlopoulou, K. Kostyrka-Allchorne, J. Phillips-Owen and E. Sonuga-Barke, *Links Between Mental Health Problems and Future Thinking From the Perspective of Adolescents With Experience of Depression and Anxiety: A Qualitative Study*.

97 E. Palacios-Barrios, J. Hanson, K. Barry, W. Albert, S. White, A. Skinner, K. Dodge and J. Lansford, *Lower Neural Value Signaling in the Prefrontal Cortex Is Related to Childhood Family Income and Depressive Symptomatology During Adolescence*.

98 M. Yeung, T. Lee and A. Chan, *Depressive and Anxiety Symptoms Are Related to Decreased Lateral Prefrontal Cortex Functioning During Cognitive Control in Older People*.

99 K. Miyamoto, N. Trudel, K. Kamermans, L. Verhagen, M. Wittmann and M. Rushworth, *Identification and Disruption of a Neural Mechanism for Accumulating Prospective Metacognitive Information Prior to Decision-Making*.

100 R. Kerestes, C. Davey, K. Stephanou, S. Whittle and B. Harrison, *Functional brain imaging studies of youth depression: A systematic review*.

101 J. Mitchell, M. Banaji and C. Macrae, *The Link between Social Cognition and Self-referential Thought in the Medial Prefrontal Cortex."MIT Press Direct*.

102 P. Hertel, C. Wahlheim, W. Price, E. Crusius and C. Patino, *Stuck in the past? Rumination-related memory integration*.

103 T. Elmer and C. Stadtfeld, *Depressive Symptoms Are Associated With Social Isolation in Face-to-Face Interaction Networks*.

104 S. Nolen-Hoeksema, *The Other End of the Continuum: The Costs of Rumination*.

105 S. Levens and I. Gotlib, *Updating positive and negative stimuli in working memory in depression*.

106 B. Hankin, *Rumination and Depression in Adolescence: Investigating Symptom Specificity in a Multiwave Prospective Study*.

107 B. Rasch and J. Born, *Maintaining Memories by Reactivation*.

108 P. Wilkinson, T. Croudace and I. Goodyer, *Rumination, Anxiety, Depressive Symptoms and Subsequent Depression in Adolescents at Risk for Psychopathology: A Longitudinal Cohort Study*.

109 X. Chen, N. Chen, Y. Shen, H. Li, L. Li, B. Lu, Z. Zhu, Z. Fan and C. Yan, *The subsystem mechanism of default mode network underlying rumination: A reproducible neuroimaging study*.

110 C. Gillespie, S. Szabo and C. Nemeroff, *Chapter 36 - Unipolar depression*.

111 X. Zhou and X. Lei, *Wandering Minds with Wandering Brain Networks*.

112 T. Ho, C. Connolly, E. Blom, K. LeWinn, I. Strigo, M. Paulus, G. Frank, J. Max, J. Wu, M. Chan, S. Tapert, A. Simmons and T. Yang, *Emotion-Dependent Functional Connectivity of the Default Mode Network in Adolescent Depression*.

113 K. Valdés-García, I. Monroy-Velasco, B. Pérez-Pedraza and L. Sánchez-Loyo, *Depression, suicide ideation, and irrational beliefs: Explanatory models in psychology students*.

114 C. Vassou, E. Georgousopoulou, C. Chrysohoou, M. Yannakoulia, C. Pitsavos, M. Cropley and D. Panagiotakos, *Irrational beliefs trigger depression and anxiety symptoms, and associated with increased inflammation and oxidative stress markers in the 10-year diabetes mellitus risk: the ATTICA epidemiological study*.

115 C. Hu, C. Wang, W. Liu and D. Wang, *Depression and Reasoning Ability in Adolescents: Examining the Moderating Role of Growth Mindset*.

116 D. Hecht, *Depression and the Hyperactive Right-Hemisphere*.

117 R. Shmerling, *Right Brain/Left Brain*.

118 *Corpus callosum*, https://qbi.uq.edu.au/brain/brain-anatomy/corpus-callosum#:~:text=The%20two%20hemispheres%20in%20your,send%20signals%20to%20each%20other.

119 F. MacMaster, N. Carrey and L. Langevin, *Corpus Callosal Morphology in Early Onset Adolescent Depression*.

120 J. Fritz, *When the Brain Is Out of Synch, the World Is Out of Balance*, https://www.homeeddirectory.com/blog/when-brain-out-synch-world-out-balance.

121 H. Mulholland, *Teenager emotions: What's normal? What's not?*, https://communityhealth.mayoclinic.org/featured-stories/teen-emotions.

122 D. Pine, L. Kentgen, G. Bruder, P. Leite, K. Bearman, Y. Ma and R. Klein, *Cerebral laterality in adolescent major depression*.

123 J. Lee, *The Contributing Factors to Adolescent Depression*.

124 S. Hofmann, A. Asnaani, I. Vonk, A. Sawyer and F. A, *The Efficacy of Cognitive Behavioral Therapy: A Review of Meta-analyses*.

125 J. Beames, K. Kikas and A. Werner-Seidler, *Prevention and early intervention of depression in young people: an integrated narrative review of affective awareness and Ecological Momentary Assessment*.

126 L. Kiropoulos, T. Kilpatrick, T. Kalincek, L. Cherulov, E. McDonald, T. Wijeratne, J. Threader, V. Rozenblat, N. Simpson-O'Brien, A. Walt and L. Taylor, *Comparison of the effectiveness of a tailored cognitive behavioural therapy with a supportive listening intervention for depression in those newly diagnosed with multiple sclerosis (the ACTION-MS trial): protocol of an assessor-blinded, active comparator, randomised controlled trial*.