

Cognitive Risks in Developmental Psychopathology

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In D. Cicchetti (Ed.) *Developmental Psychopathology*, Wiley.

Introduction

“Why should we think upon things that are lovely? Because thinking determines life.” William James.

Psychology is standardly defined as the study of thoughts, behaviors, and emotions.

Developmental psychopathology includes the investigation of both normal and abnormal phenomena and processes in psychology. This chapter centers on thoughts, that is, cognition, in developmental psychopathology. Cognition means different things to different scholars. We focus on, and organize our review around, central mental *processes*, including attention, memory, and executive functioning, alongside core cognitive (sometimes considered social-cognitive) *products*, including attributions, attitudes, rumination, and reappraisal (see (R. E. Ingram, Miranda, & Segal, 1998), for discussion of differentiating cognitive products and processes as risks to psychopathology). First, we start with a brief modern historical overview concerning the progression of how cognitive factors and processes have been conceptualized and examined from both a normal and abnormal perspective. Next, we introduce some logical conceptual models to organize our ensuing review of evidence. Then, we review the dominant cognitive risks, structured by processes and products, for several prevalent and impairing psychopathologies (internalizing disorders of depression and bipolar disorder (BD) as well as anxiety disorders, externalizing disorders of attention deficit hyperactivity disorder (ADHD) and conduct disorders, and schizophrenia). Space limitations did not permit a review of every cognitive risk; rather, we summarize knowledge on those main theoretically specified and empirically investigated products and processes. Finally, we discuss several points that emerged from this review and present some future research directions, including: (1) developmental issues, (2) the integration of cognitive products and processes within cognitive/affective neuroscience and genetics frameworks, (3) transdiagnostic versus disorder-specific models of psychopathology, and (4) translational implications.

History

The history of cognitive approaches in psychopathology has followed a curious path, best illustrated as mostly parallel play, between two predominantly independent scientific traditions: clinical psychology/psychiatry and experimental psychology/cognitive science. This theme of parallel play between clinical and cognitive science is largely reflected up to the present, and is also mirrored in the organization of our review of cognitive influences in developmental psychopathology. More specifically, cognitive clinical scientists have tended to emphasize and study more easily accessible, self-reportable cognitive products (e.g., attributions, automatic thoughts and dysfunctional attitudes), whereas experimental cognitive scientists have predominantly focused on cognitive processes (e.g., memory and attention) that are typically assessed via experimental tasks. Moreover, cognitive clinical scientists have tended to be more interested in and study individual differences in cognitive risks and associations with psychopathology, whereas experimental cognitive scientists have leaned more toward group differences in cognitive processes.

Both clinical and cognitive scientists followed the paradigmatic shift observed in the broad history of psychology, with the transition from psychodynamic to behaviorism and then the cognitive revolution. Broadly speaking, cognitive approaches to psychopathology have progressed through five stages: (1) first starting with the descriptions and observations of how cognition is perturbed in psychopathology (Beck, 1967), (2) then to experiments focused on cognitive processes in aggression (e.g., Bandura, Ross, & Ross, 1961; Bandura, 1977), (3) followed later by an emphasis on cognitive products (e.g., Abramson, Seligman, & Teasdale, 1978; Dodge, 1980), (4) leading to investigations using cognitive science tasks and methods (R. E. Ingram et al., 1998), and a focus on information processing grounded in social psychology (e.g., Bandura, 1986) and emotion (e.g., J J Gross & Thompson, 2007) and (5) finally and most recently, an emphasis seeking to integrate genetics (Feder,

Nestler, & Charney, 2009; Gibb, Beevers, & McGeary, 2013; Swanson et al., 2007) as well as cognitive and affective neuroscience models and approaches (Beck, 2008; e.g., Bishop, 2007; De Raedt & Koster, 2010; Nigg & Casey, 2005; J. Posner, Russell, & Peterson, 2005).

Perhaps the most recognized clinical scientist to advance cognitive approaches to psychopathology, Aaron Beck, initiated clinical cognitive studies of psychopathology via description and observation. As a psychodynamically trained psychiatrist, he was searching for classic psychoanalytic themes of anger turned inwards among the dreams of his depressed patients, but instead accurately recognized that these patients described themes of loss, failure, hopelessness, and pessimism. Beck's work, based on his astute clinical observations with depressed adults revealing negative thoughts of the self, world, and future, led to his ground-breaking book on Cognitive Therapy (Beck, 1967) and initial theorizing focusing on etiology and treatment approaches involving cognitive schemas, content, and process in depression. About this same time, Albert Ellis was promoting his cognitive approach—Rational Emotive Behavior Therapy (Ellis, 1957).

Also about this time, an instrumental theoretical and empirical figure, Bandura, was advancing beyond psychoanalytic doctrine and was initiating his classic line of inquiry investigating behavioral (e.g., Bandura et al., 1961), and what ultimately became social-cognitive (e.g., Bandura, 1977), processes using a more experimental approach.

Progressing firmly as the cognitive revolution was in full swing, several theoretical advances in clinical cognitive understanding of psychopathology unfolded with an emphasis on cognitive products. Building on the basic behavioral understanding of learned helplessness approaches to human depression (W. R. Miller & Seligman, 1975), Abramson and colleagues underscored how attributions can affect depression (e.g., Abramson et al., 1978). Dodge and colleagues also applied attributional models to explicate aggression and conduct disorder (e.g., Crick & Dodge, 1994; Dodge, 1980).

About this same time, various experimental cognitive scientists were conducting more controlled experiments with core cognitive processes, including attention, memory, and executive functioning, using between groups comparison designs, sometimes with psychopathologically disordered groups. Relatedly, other experimentalists, using more social psychology and emotion traditions (e.g., J J Gross & Thompson, 2007) were similarly investigating cognitive processes and information processing, using experimental tasks developed in cognitive psychology, with clinical applications in aggression, anxiety, and mood (Joormann, 2010a). At the present time, the state of the field is focused on integrating cognitive processes and products with either genetic influences or neuroscientific principles and findings (e.g., Beck, 2008; Disner, Beevers, Haigh, & Beck, 2011; Nigg & Casey, 2005).

Logical models

Before initiating our review of cognitive processes and products in common psychopathologies, it is important to discuss several ways that can be used to more formally model the relationship between cognitive factors and psychopathology. The following section will discuss several logical models that could be used to conceptualize the role of cognitive influences in developmental psychopathology: correlate, consequence, risk factor/ vulnerability, causal risk factor. We draw on prior work articulating logical models that can more formally describe relations between a risk and psychopathology (e.g., Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). As we discuss later, a general shortcoming of the broad field of cognitive risks in developmental psychopathology across the lifespan is the lack of consideration and application of such logical models to more formally organize cognitive risks and try to rule out, or in, particular models based on the evidence. This is a goal of the chapter and our review. In addition, this section will also describe how cognitive factors might work together with other influences to contribute to

psychopathology through the following mechanisms: moderation, mediation, and dynamic/transactional.

The simplest type of relationship characterizes a cognitive factor as a *correlate* of psychopathology, in that the two are associated (Kazdin et al., 1997). In addition, cognitive influences may also be a *consequence* of maladjustment, such that they accompany or follow directly from psychopathology. Correlates can be identified using cross-sectional or retrospective study designs. Establishing cognitive correlates of mental health outcomes can act as the first step to inform future research designs, as discussed later, which are better able to address the temporal or potential causal relationship among cognitive influences and developmental trajectories of psychopathologies.

Within the conceptualization of a correlate, a cognitive construct may also act as a risk factor, or vulnerability, to psychopathology, such that the cognitive influence is associated with an increase in the probability of the outcome over the population base rate of the outcome (Kazdin et al., 1997). A risk factor is therefore a specific type of correlate that must temporally precede the outcome. Risk factors can only be identified using a prospective longitudinal design. Within risk factors, there are two categories: variable and fixed risk factors. Variable risk factors may change within an individual (e.g. age, height), whereas fixed risk factors do not change (e.g. race, sex). Cognitive influences are mutable variables, and therefore fall under the category of variable risk factors. For example and as we review later, a body of research that has utilized multiple time point designs has found that a negative inferential style predicts, especially in interaction with stressors, depression. In addition, as discussed in the literature review, prospective longitudinal studies suggest that impairments in EF, memory, attention and related brain systems predict later psychosis, ADHD, and PTSD, suggesting that cognitive deficits may be a risk factor for many disorders. A recent, relatively specific type of risk factor is an endophenotype (Gottesman & Gould, 2003), which is an intermediate trait (e.g. cognitive products and

processes) that is relatively stable over time (e.g., present even during remission from illness) and present in healthy family members, thus linking genetic risk to later psychopathology.

Finally, a subset of risk factors may act as causal risk factors, such that the manipulation of these risk factors changes the probability of the outcome (Kazdin et al., 1997). The only study designs that are sufficient to identify causal risk factors include naturalistic experiments, laboratory experiments (e.g. randomized control trials, animal studies), or studies that examine dose-response relationships. Studies that have manipulated cognitive reappraisal in a laboratory setting, for instance, have demonstrated that this cognitive construct is associated with less sadness and general negative affect among both clinical and nonclinical participants (Perry & Henry, 2012). Likewise, training to reduce attentional bias towards negative information has been shown to reduce risk for depression recurrence (e.g., Browning, Holmes, Charles, Cowen, & Harmer, 2012a), and experimental modification of cognitive emotion recognition at the perceptual level reduced anger and aggression in ambiguous contexts (Penton-Voak et al., 2013).

As part of a developmental, interdisciplinary approach to the study of cognitive risks to psychopathology, it is safe to assume that cognitive risk factors do not operate in isolation, but rather work together with other influences to engender risk to psychopathology. One possibility is moderation. In this type of relationship, a moderator “B” changes the relationship between independent variable “A” and the outcome “O” (Kraemer et al., 2001). It is important to note that, strictly speaking, the moderator “B” does not affect the level of “A” or the probability of “A”. Statistical analysis that examines moderators may seek to evaluate hypotheses of differential susceptibility to psychopathology as well as resiliency that buffers against these maladaptive outcomes.

A second mechanism that models how risk factors work together is mediation. In this type of relationship, a mediator “B” is caused to vary by the independent variable “A”. “B”, in turn, causes

variation in the outcome “O” (Kraemer et al., 2001). Therefore, unlike in moderation, “A” does indeed affect the mediator “B”. Statistical analysis that examines mediators may seek to identify an intermediate variable in the causal pathway from “A” to a negative mental health outcome, thereby explaining how and why “A” influences that outcome.

A final model that explains how risk factors work together is the transactional model (e.g., Sameroff, 1975). In this model, variables are “recursive”, such that variables can serve both as an antecedent and as an outcome. For example, in the cognitive vulnerability-transactional stress model for depression (Hankin & Abramson, 2001), a negative life event contributes to initial elevations in negative affect (e.g. sadness, anger), which interacts with cognitive risk factors (e.g., negative cognitive style) to maintain and amplify negative affect, thereby increasing risk for depression, which in turn contributes to future dependent negative life events (e.g. fight with a friend), perpetuating this recursive cycle. It is important to note that these logical models are not mutually exclusive. Indeed, it is highly likely that different models will hold true for different cognitive products and processes, or even for the same cognitive process or product at different times or for different individuals.

Overview and Plan for the Literature Review

The following sections review the evidence of core cognitive risks to psychopathology. There are several initial points to highlight. First, we do not review every single cognitive risk (process or product) that has ever been investigated. Rather, we concentrate this review on the main theoretically specified and empirically investigated factors and processes. Second, we do not review all forms of psychopathology, but rather focus on several of the most investigated and prevalent forms of psychopathologies—depression, BD, schizophrenia, anxiety (primarily posttraumatic stress disorder (PTSD), social anxiety disorder (SAD), generalized anxiety disorder (GAD), panic, and obsessive compulsive disorder (OCD), and externalizing disorders (primarily ADHD) and conduct disorders). Third, we organize this review mainly around prior

reviews and meta-analyses, as well as essential individual papers, that establish associations between these cognitive influences and those selective psychopathologies. It is important to emphasize, as became evident in conducting this review of core cognitive risks and psychopathologies, that not all of the cognitive products and processes have been studied with all of the main psychopathological disorders we reviewed. As such, our review covers associations between cognitive risks and psychopathology for those links for which there is a sufficient evidence base.

From this review, certain general conclusions can be reached. First, evidence is consistent with the view that cognitive processes are perturbed across disorders such that these processes may function as transdiagnostic risks to many of the psychopathologies reviewed, whereas cognitive products may operate as relatively more specific risks to particular psychopathologies based on the content and affective tone of the cognition and disorder. Still, this relatively broad conclusion requires additional research to substantiate, and it is a point to which we return to and expand upon later in the Discussion. For example, while the data suggest many processes may be relatively general risks to psychopathology transdiagnostically, future research may find greater specificity in cognitive processes when examined in greater detail and at multiple levels of analysis (e.g., different neurotransmitter systems implicated in the underlying neural processes, or different strategies employed in cognitive tasks assessing these core processes). Likewise, seemingly specific cognitive products (e.g., rumination and worry) may share common underlying processes.

Second, the data reveal significant associations between cognitive risks and psychopathology, but the current state of knowledge is not consistently and systematically organized around the logical conceptual models presented in the previous section. To the extent possible, we present the evidence in this review in a manner that attempts to adjudicate among these potential logical models. Can certain ones be ruled out (e.g., purely concomitant association) for certain cognitive risks and particular forms of

psychopathology? Are other models (e.g., risk factor/vulnerability) presently more favored and supported by the evidence?

Third, throughout the review we will point out the age group of the samples (e.g., child, adolescent, adult), some basic design features (e.g., cross-sectional, longitudinal; case-control), and methods (e.g., questionnaires/ self-reports in much of the product literature; cognitive tasks in most of the process studies). From the review it became clear that the majority of the work has focused on adult samples, although this generality varies and depends on the particular cognitive process and product investigated along with disorder. We return to discuss and comment on developmental/age features and design considerations later in the chapter.

Finally, a key theme that emerged from this review is that there has generally been an independent, “silo” approach underlying inquiries into cognitive risks to development of psychopathology. To our knowledge no prior review has covered *both* cognitive products and processes together *and* development of several main psychopathologies, including both internalizing and externalizing problems. Many investigators focus on one product or process and one disorder only, and there has been little connection across different levels of analysis in cognition (i.e., lack of process and product integration) and across different forms of psychopathology (i.e., possibly transdiagnostic vs. specific disorder prediction). Just as young children interact together in parallel play, there has not been much cross-talk between the cognitive products and process literature, or between literatures on cognition in different clinical disorders. We organized our review by cognitive processes and products, rather than by clinical disorder, purposefully to begin to facilitate this conceptual shift focused on cognitive influences, which may be transdiagnostic risks to development of psychopathologies. We hope that this review will stimulate future research to move beyond the current silo approach and begin to integrate studies across cognitive products and processes and examine how these multiple cognitive influences predict development of several psychopathologies.

Cognitive Processes

Executive Function

Executive function (EF) processes enable us to respond flexibly to the environment and regulate their thoughts and behaviors, allowing us to break out of habits, make decisions and evaluate risks, plan for the future, prioritize and sequence our actions, and cope with novel situations. EF is comprised of a set of cognitive control processes, mainly supported by the prefrontal cortex (PFC), which regulate lower level cognitive processes (e.g., perception, motor responses) and thus enable self-directed behavior towards a goal (e.g., Miyake & Friedman, 2012). Given the breadth of the EF construct, many different tasks have been used to assess EF in the laboratory (Table 1). EF appears to be especially vulnerable to disruption, as evidenced by EF impairments associated with most forms of psychopathology, as discussed below. Thus, it has been proposed that EF deficits may be transdiagnostic intermediate phenotypes or risk factors for emotional, behavioral, and psychotic disorders (Susan Nolen-Hoeksema & Watkins, 2011). There is strong evidence that multiple aspects of EF are indeed impaired across most diagnostic groups, although the magnitudes of these deficits vary, both across disorders and across aspects of EF.

Core Components of EF: Shifting, Updating, and Inhibition. EF is best characterized as consisting of separable but related cognitive processes, with both unique and shared individual differences, genetic influences, and neural substrates (Miyake & Friedman, 2012). One influential model which has been applied to understanding the effects of psychopathology on EF, the *unity/diversity model* has identified three fundamental aspects of EF: (1) shifting, (2) inhibition, and (3) updating working memory, and as well as a common EF ability which is involved in all aspects of EF and may subsume inhibition (Miyake & Friedman, 2012). Shifting is defined as switching between task sets or response rules. For example, you may need to shift from reading this chapter to responding to an

urgent email and back again. Inhibition is defined as suppressing or resisting a prepotent (automatic) response in order to make a less automatic but task-relevant response. For example, you may want to resist the automatic response of checking those not-so-urgent emails in order to complete reading the chapter. Updating is defined as monitoring and coding incoming information for task-relevance, and replacing no longer relevant information with newer, more relevant information. For example, as you read this chapter, you may be monitoring for a relevant piece of information you are looking for (say, the definition of updating), hold this information in working memory while you write it down, then replace it with the next relevant piece of information. Common EF is defined as what is shared across all EF tasks, and is posited to be the ability to monitor for and maintain goal and context information (e.g., the goal of finishing this section of the chapter; (Miyake & Friedman, 2012). This theory is compatible with the view that the central role of the frontal lobes is active maintenance of goals, plans and other task-relevant information, which may be essential for all aspects of EF (E. K. Miller & Cohen, 2001).

The largest deficits on these core aspects of EF are found for individuals with schizophrenia. Meta-analyses have found large impairments on measures of shifting, inhibition and updating (Table 2). A recent meta-analysis also found that adolescents with psychosis risk syndrome have small but significant deficits in EF, which are somewhat larger in those who go on to convert to schizophrenia (Giuliano et al., 2012). EF deficits increase during the prodrome, and increase again with the first psychotic episode, then appear to be fairly stable over time (e.g., see Lewandowski, Cohen, & Ongur, 2011 for review), with little evidence that individuals with schizophrenia experience greater age-related declines in EF than healthy individuals (Irani, Kalkstein, Moberg, & Moberg, 2011). However, there is some evidence that EF deficits are sensitive to symptom levels. Specifically, EF impairments in schizophrenia are associated with negative symptoms and disorganization, but not with positive

symptoms (Dibben, Rice, Laws, & McKenna, 2009).

These EF processes are also impaired in individuals with mood disorders, although the magnitude of these deficits is somewhat smaller than those in schizophrenia. Meta-analytic evidence demonstrates that individuals with depression are significantly impaired on measures tapping shifting, inhibition, and updating, with medium effect sizes, while individuals with BD have somewhat larger impairments in shifting and inhibition, while there is little research on updating in individuals with BD (Table 2). More research is needed to determine how EF deficits change over the lifespan in individuals with depression and BD. A meta-analysis of children with BD found fairly comparable effect sizes to those in adults (Walshaw, Alloy, & Sabb, 2010), suggesting that EF deficits are already present early in BD and may be relatively stable across the lifespan. EF deficits in individuals with depression appear to be relatively stable from adolescence through older adulthood (Snyder, 2013), but there has been little research in younger adolescents or children with depression. EF deficits in BD and depression also appear to be fairly stable across current mood state, as most measures of EF remain equally impaired euthymic individuals with BD (Kurtz & Gerraty, 2009)(Table 2) and individuals with depression in remission (Snyder, 2013). On the other hand, some EF deficits increase with symptom severity (e.g., see McDermott & Ebmeier, 2009 for meta-analysis). However, longitudinal research is needed to determine whether EF tracks changes in depression severity, or whether preexisting EF abilities affect depression severity.

Individuals with OCD have impaired performance across these core EF domains, with small effect sizes for shifting and inhibition, but large effect sizes for updating (Table 2). Importantly, while depression frequently co-occurs with OCD, EF deficits in OCD are not driven by co-occurring depression, as even those with low levels of depressive symptoms show the same level of EF deficits (Snyder, Kaiser, Warren, & Heller, 2013). While there have been few studies of EF in children and

adolescents with OCD, they appear to have EF deficits similar to those of adults with OCD (e.g., Andrés et al., 2007; Shin et al., 2008), suggesting that EF deficits are present from soon after illness onset. EF deficits appear to be fairly stable through adulthood (Snyder et al., 2013), but there has been little research on older adults with OCD.

Evidence for impairments in these core EF domains is less consistent for other anxiety disorders. A recent meta-analysis found that compared to trauma-exposed individuals who did not develop PTSD, individuals with PTSD had worse performance on measures of shifting, with a medium effect size, but not the Stroop measure inhibition (Table 2; (Polak, Witteveen, Reitsma, & Olf, 2012). However, a review of the literature including a wider range of inhibition tasks suggests that individuals with PTSD do experience inhibition deficits (Aupperle, Melrose, Stein, & Paulus, 2012). EF impairments in individuals with PTSD do not seem to be clearly influenced by current mood state, suggesting that they are trait-like (Aupperle et al., 2012). However, unlike OCD, co-occurring depression may account for EF deficits in individuals with PTSD, although more research in individuals without severe depressive symptoms is needed to confirm this finding (Polak et al., 2012). While most research has focused on adults, a few studies in children and adolescents have also found EF impairments associated with trauma and PTSD (Carrion, Wong, & Kletter, 2012). However, in adults there is some evidence that EF deficits associated with PTSD increase with age from early to middle adulthood, although this finding is based on cross-sectional studies, leaving the possibility that cohort effects (e.g., in type of trauma exposure) could account for these findings (Polak et al., 2012).

In contrast with OCD and PTSD, there is not strong evidence for broad EF impairments associated with other anxiety disorders, and there have been no published meta-analyses. While a few studies have found impairments in shifting associated with PD, SAD and GAD in adults (Airaksinen, Larsson, & Forsell, 2005; L. J. Cohen et al., 1996; Mantella et al., 2007), others have found no

evidence of impairment in shifting (Airaksinen et al., 2005; Boldrini et al., 2005; Purcell, Maruff, Kyrios, & Pantelis, 1998), or inhibition (Price & Mohlman, 2007a; van den Heuvel et al., 2005; Van der Linden, Ceschi, Zermatten, Dunker, & Perroud, 2005). In sum, there is limited and inconsistent evidence for EF impairments on traditional neuropsychological tasks in individuals with anxiety disorders other than OCD and PTSD.

However, research in non-clinical samples suggests that trait anxiety, and especially anxious apprehension (worry) is associated with impairments in a specific aspect of EF, inhibiting competing responses. A number of studies have found that high trait anxiety is associated with impaired inhibition task performance (e.g., Bishop, 2008; Eysenck & Derakshan, 2011; Snyder et al., 2010), although others have not (e.g., Avila & Parcet, 2001), likely due to differences in the types of tasks, and potentially most critically, the measures of anxiety used. Recent research suggests that anxious apprehension, but not anxious arousal, is associated with an impaired ability to inhibit competing verbal responses (Snyder et al., 2010). Since anxious apprehension and anxious arousal can both be present to different degrees in individuals with anxiety disorders, more specific measures of anxious apprehension, as well as more sensitive and specific measures of inhibition, may be beneficial in detecting EF deficits in such individuals.

Finally, ADHD is associated with impairments in shifting and inhibition, while updating has not been widely studied. While earlier theories posited a core inhibitory deficit that secondarily disrupts other aspects of EF (e.g., Barkley, 1997), recent meta-analyses demonstrate that only motor response inhibition tasks (stop signal and go/no-go) show substantial deficits, while the Stroop measure of inhibition shows only a small effect size (Table 2). EF is also impaired in other externalizing disorders, including ODD/CD, but these deficits may be accounted for at least in part by co-occurring ADHD (see Ogilvie, Stewart, Chan, & Shum, 2011 for meta-analysis). EF deficits occur in individuals of all ages

with ADHD, from preschoolers (Pauli-Pott & Becker, 2011) through mid-adulthood (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005). However, effect sizes are somewhat larger in meta-analyses of adults with ADHD than those with children and adolescents (Table 2). It is possible that the subset of individuals who continue to experience ADHD symptoms into adulthood have a more severe form of the disorder, contributing to greater EF deficits, or that poor EF is a risk factor for continued ADHD symptoms in adulthood. However, this must be interpreted with caution because different tasks or task variants are often used with different age ranges, precluding direct comparisons.

Working Memory. While shifting, inhibition and updating are important aspects of EF, this model in no way posits that these are the only components of EF. For example, working memory is often considered a component of EF. Working memory (WM) is defined as actively maintaining (i.e., ‘holding on line’) or manipulating information across a short delay. Manipulating information in WM places heavier demands on EF (i.e., the *central executive* component of WM) than simple maintenance (Repovs & Baddeley, 2006). WM maintenance can further be divided into verbal (e.g., words, letters and numbers) and visuospatial (e.g., shapes, patterns and spatial locations) stores), while the central executive component of WM is believed to be domain-general (e.g., Repovs & Baddeley, 2006).

Working memory deficits are widespread across forms of psychopathology, but vary in magnitude both across disorders and across components of WM. Visuospatial WM is consistently impaired, with large effect sizes for individuals with schizophrenia, and medium-small effect sizes for BD and ADHD, and small but significant effect sizes for depression and OCD (children and adolescents for ADHD, mainly adults for all other disorders; Table 2). Verbal WM is also widely impaired, but deficits are smaller for simple verbal working memory maintenance (e.g., digit span forward) than verbal working memory manipulation (e.g., digit span backward). Specifically, meta-analyses show verbal WM manipulation deficits with large effect sizes for schizophrenia, medium

effect sizes for BD, MDD, and ADHD (somewhat larger for children/adolescents than adults), and small but significant effect sizes for PTSD and OCD. In contrast, simple verbal WM maintenance tasks show only medium effect sizes for schizophrenia, small effects for BD, depression, and ADHD (only adults meta-analyzed), and no significant impairment in OCD (Table 2). There has been very little research on WM in individuals with anxiety disorders other than OCD, but there have been reports of impaired visuospatial WM in individuals with PD (Boldrini et al., 2005), and impaired verbal WM manipulation, but not maintenance, in individuals with GAD (Christopher & MacDonald, 2005).

The finding that manipulation is more impaired than maintenance, along with evidence that visuospatial and verbal WM manipulation are equally impaired, both support the view that working memory deficits in these disorders are due to impairment in the central executive aspect of working memory, rather than the content-specific maintenance systems (Barch, 2005). This suggests that verbal WM deficits may arise from difficulty with encoding and/or manipulating information in working memory, rather than maintenance of information. Supporting this, a meta-analysis found that working memory deficits associated with schizophrenia do not increase with the delay interval over which material must be maintained (J. Lee & Park, 2005), while studies have found that increasing presentation time or stimulus saliency to aid encoding improves working memory performance in individuals with schizophrenia (J. Lee & Park, 2005). However, this more fine-grained analysis of the factors driving WM impairments has not been performed for other forms of psychopathology.

Complex Tasks: Verbal Fluency & Planning. Many complex tasks may also tap multiple aspects of EF. For example, verbal fluency tasks (generating words starting with a certain letter or from a category) likely tap several cognitive processes (Rende, Ramsberger, & Miyake, 2002). Planning tasks are also complex, involving multiple cognitive demands (Goel & Grafman, 1995), and so may not represent a single EF ability. Notably, verbal fluency and planning tasks are frequently used in clinical

studies. Such tasks may be commonly implemented in clinical research because they are viewed as more ecologically sensitive: the complexity of verbal fluency and planning tasks may make them more relatable to real-world tasks that require similar skills. Thus, there are both disadvantages (in terms of interpretability) and advantages (in terms of ecological validity) in the use of such complex EF tasks.

Deficits in verbal fluency are widespread across disorders. Indeed, meta-analyses show that the largest deficit for adults with schizophrenia and depression is found on the semantic verbal fluency task, with large and medium effect sizes respectively (Table 2). Semantic verbal fluency is also impaired in individuals with BD (all ages, with medium effect sizes), OCD (mainly adults, with small effect sizes), and ADHD (all ages, with small effect sizes), while there is inconsistent evidence for verbal fluency in individuals with PTSD (Aupperle et al., 2012)(Table 2). For schizophrenia, BD, and MDD, effect sizes for phonemic verbal fluency are somewhat smaller than those for semantic, although still significant (Table 2). In contrast, individuals with OCD have equal impairments in the two forms of verbal fluency, and verbal fluency deficits associated with ADHD appear to be larger for phonemic verbal fluency than semantic verbal fluency (Table 2). There has been little research on verbal fluency in anxiety disorders other than OCD: one study reported impaired phonemic verbal fluency in individuals with PD (Gladsjo et al., 1998), while others found no impairment in individuals with GAD (Airaksinen et al., 2005) or SAD (Hood et al., 2010). However, conclusions are premature given the paucity of evidence.

Why might semantic and phonemic verbal fluency tasks be differentially affected in different disorders? Verbal fluency tasks impose multiple EF demands (e.g., shifting among subcategories, monitoring for repeated words, memory retrieval). One possibility as to why semantic verbal fluency is more impaired in individuals with schizophrenia, BD, and depression is that it may place heavier demands on shifting, and particularly on selecting what to switch to, since category cues are likely to

lead to the activation of many category members, which then compete for production (Snyder & Munakata, 2010). Another possibility is that deficits in semantic memory retrieval may contribute to semantic verbal fluency impairment, particularly in individuals with schizophrenia. For example, a meta-analysis found that individuals with schizophrenia have large deficits on semantic verbal fluency both for switching between subcategories, an index of EF ($d = 1.02$), and semantic clustering, and index of semantic memory ($d = 0.93$) (Doughty & Done, 2009). In contrast, the larger effect for phonemic verbal fluency in individuals with ADHD could potentially be due to deficits in phonological processing in many individuals with ADHD, since ADHD and reading disabilities frequently co-occur (Willcutt, Pennington, Olson, & DeFries, 2007). Thus, deficits in verbal fluency may arise from a variety of sources, and illustrate the difficulty of interpreting results from complex tasks.

Planning has been much less studied. Individuals with BD have significant impairments in planning, with one meta-analysis finding a small effect size in adults and another finding a large effect size in children and adolescents (Table 2). Adults with depression also have significant, but small, impairments on planning tasks (Table 2). In individuals with ADHD, two meta-analyses, both in children and adolescents, found quite different effect sizes for planning tasks, one small, one medium (Table 2). Finally, there is inconsistent evidence for planning deficits associated with PTSD (Aupperle et al., 2012). Thus, while planning tasks in theory tap multiple aspects of EF, standard measures of planning may be less sensitive than other EF tasks in detecting more subtle EF deficits associated with some disorders.

Table 1

Examples of commonly used EF tasks

Construct	Task	Description	Outcome Measure(s)	Methods Citation¹
Shifting	Intradimensional/ Extradimensional Shift	Learn from feedback to select a stimulus based on one dimension, switch to the previously non-rewarded stimulus (intradimensional shift), then to a different stimulus dimension (extradimensional shift).	*1. Perseverative errors in intradimensional & extradimensional shifts. 2. # of shifts achieved. 3. Time to complete.	(Robbins et al., 1998)
	Trail Making B ²	Alternately connect letters and numbers in sequence (A-1-B-2 etc.) Often compared to Trail Making A (connect letters or numbers only, does not require shifting).	*1. Trail Making B–Trail Making A time. 2. Errors in B. 3. Time to complete B.	(Strauss, Sherman, & Spreen, 2006)
	Object Alternation	Find object hidden alternately under two different cups.	Errors	(Kuelz, Riemann, Zahn, & Voderholzer, 2004)
	Wisconsin Card Sorting Task (WCST)	Learn from feedback to sort cards by one dimension (e.g. color), and then switch to a different dimension (e.g. shape) when given negative feedback on the first dimension (repeats with multiple sorting rules).	*1. Perseverative errors. 2. # of rules achieved. 3. Time to complete.	(Strauss et al., 2006)
	Cued task switching	Perform one of two tasks depending on cue before each trial (e.g. color/shape, addition/subtraction, number/letter, categorize by size/ categorize by living vs. nonliving).	*1. Switch cost (switch–repeat RT). *2. Perseverative errors.	(Friedman et al., 2008)
Inhibition	Color-word Stroop (experimental version)	Identify the color ink a color word is printed in. Trials are incongruent (e.g. “red” written in blue ink) and congruent (e.g. “red” written in red ink) or neutral (non-color word) trial RTs. Trial types are randomly intermixed.	1. Stroop interference (incongruent–neutral RT) 2. Incongruent–neutral errors.	(Silton et al., 2010)
	Color-word Stroop (neuropsychological version)	Separate blocks of word reading, color naming, and incongruent (e.g. “red” written in blue ink) trials.	1. Incongruent–color naming time. 2. Incongruent block time. 3. Incongruent block errors.	(Strauss et al., 2006)
	Stop-signal	Quickly categorize and respond to stimuli (e.g. left and right pointing arrows), unless a stop signal appears, signaling to	1. Stop signal RT (time needed to stop a response).	(Band, Van der Molen, & Logan,

		withhold a response.	2. Commission errors	2003)
	Antisaccade	Look in the opposite direction of visual cue.	Errors (detected by eye tracking or failure to detect briefly presented target on correct side).	(Friedman et al., 2008)
	Go/No-Go	Quickly categorize and respond to some stimuli, and withhold a response to other stimuli.	Commission errors.	(Rubia et al., 2001)
	Hayling	Read sentences where the final word is omitted but highly predictable. First complete sentences correctly (Part A), then with an unrelated word (part B).	1. Part B – Part A RT. 2. Part B errors.	(Strauss et al., 2006)
Working Memory Updating	Keep Track	Remember to last exemplar word presented in several target categories and report these words at the end of the trial.	Accuracy	(Friedman et al., 2008)
	Letter Memory	Remember and repeat the last three letters in a letter string, adding the most recent letter and dropping the 4th letter back.	Accuracy	(Friedman et al., 2008)
	Verbal n-back	Indicate if the stimulus (usually letter) matches the stimulus <i>n</i> (e.g. 3) items back.	Accuracy	(Braver, Cohen, Nystrom, Jonides, & Smith, 1997)
	Spatial n-back	Indicate if the spatial location of a stimulus matches the location <i>n</i> (e.g. 3) items back.	Accuracy	(Friedman et al., 2008)
Visuospatial Working Memory	Corsi block tapping/ Spatial Span	Tap irregularly arranged blocks/squares in the same order as experimenter (Corsi blocks) or computer (Spatial Span).	Span (Max. length of sequence correctly performed).	(Strauss et al., 2006)
	Self-Ordered Pointing	Search an array of boxes for hidden tokens. Token is only in each location once.	1. Errors (return to previous location). 2. Strategy score (how often search is initiated from same starting box).	(Owen, Downes, Sahakian, Polkey, & Robbins, 1990)
	Sequential Comparison	View an array of colored squares & after a delay, indicate if any squares have changed colors in a test array.	Span (see reference)	(S. J. Luck & Vogel, 1997)
Verbal Working Memory	Digit Span (Forward and Backward)	Repeat sequence of numbers in forward or reverse order.	Span (Max. length of sequence correctly performed).	(Strauss et al., 2006)
	Letter-Number Sequencing	Repeat list of alternating letters and numbers, re-sequenced into numbers first, then letters.	Span (Max. length of sequence correctly performed).	(Strauss et al., 2006)

	Reading Span	Read a series of unrelated sentences, then recall the last word of each sentence.	# correctly recalled words	(Friedman & Miyake, 2004)
	Operation Span	Read aloud & verify simple math equations, then read aloud a presented word. At end of trial, recall all words.	# correctly recalled words	(Miyake et al., 2000)
Verbal Fluency	Semantic Verbal Fluency/ Category Fluency	Say as many words from a semantic category (e.g. animals) as possible in 1 (or 3) min.	1. Switches between subcategories 2. # of words	(Troyer, Moscovitch, & Winocur, 1997)
	Phonemic Verbal Fluency/ Controlled Oral Word Association (COWA)	Say as many items starting with a certain letter (usually F, A, S) as possible in 1 (or 3) min.	# of words	(Troyer et al., 1997)

Note. ¹Citations are for sources that provide a full description of the task and outcome measures of the most recent or recommended version of the task, not necessarily the first publication to report the task. ² Not recommended for future research because it has poor construct validity (e.g, effect sizes are equal for the Trail Making Test Part A measures, Snyder, 2013).

Table 2

Summary of Recent EF Meta-Analyses

	Meta-analysis	Clinical Group	Shifting	Inhibition	Updating	Verbal WM Manip.	Verbal WM Maint.	Visuospatial WM	Phonemic VF	Semantic VF	Planning
Depression	Snyder, 2013	MDD	0.47	0.58	0.57	0.52	0.39	0.45	0.46	0.70	0.38
BD	(Arts, Jabben, Krabbendam, & Van Os, 2008)	Euthymic BD (all)	0.94	0.73		1.02	0.37	–	0.59	0.87	–
	(Bora, Yucel, & Pantelis, 2009)	Euthymic BD (all)	0.78	0.76		0.75	0.37	–	0.60	–	–
	(Bora, Yücel, Pantelis, & Berk, 2011)	Euthymic BD II	0.51	0.72		–	0.39	–	0.47	0.46	0.29
	(Kurtz & Gerraty, 2009)	Euthymic BD (all)	0.67	0.75		0.65	0.41	–	0.51	0.75	–
	(Kurtz & Gerraty, 2009)	Manic/ mixed BD (all)	0.68	–	–	–	–	–	0.51	0.59	–
	(Kurtz & Gerraty, 2009)	Depressed BD (all)	0.64	–	–	–	–	–	0.93	–	–
	(Mann Wrobel, Carreno, & Dickinson, 2011)	Euthymic BD (all)	0.73	0.78		0.81	0.40	0.55	0.55	0.58	–
	Robinson et al., 2006	Euthymic BD (all)	0.77	0.63		0.98	0.47	–	0.34	1.09	–
	(Torres, Boudreau, & Yatham, 2007)	Euthymic BD (all)	0.62	0.71		0.54	–	–	–	–	–
	(Walshaw et al., 2010)	Pediatric BD (all)	0.73	0.46		–	–	0.80	0.34	0.38	0.96
Average			0.71	0.69	–	0.79	0.40	0.68	0.54	0.67	0.63
OCD	Snyder et al., under review	OCD	0.39	0.39	0.92	0.20	0.07	0.47	0.38	0.41	0.45
PTSD	(Polak et al., 2012)	PTSD vs. trauma exposed controls	0.70	0.10	–	0.45	–	–	–	–	–
Schizophrenia	(Bokat &	Schizophrenia	–	–	–	–	–	–	0.99	1.27	–

	Goldberg, 2003)	Schizophrenia	0.87	0.99	–	0.86	0.73	–	0.83	1.41	–
	(Dickinson, Ramsey, & Gold, 2007)	Schizophrenia	–	–	–	–	–	–	–	1.34	–
	(Doughty & Done, 2009)	Schizophrenia	–	–	–	1.08	0.82	0.87	–	–	–
	(Forbes, Carrick, McIntosh, & Lawrie, 2009)	Schizophrenia	0.98	0.98	–	–	–	–	0.95	1.12	–
	(J Henry & Crawford, 2005)	First episode psychosis	0.86	0.88	–	0.79	0.50	0.80	0.69	1.24	–
	(Mesholam-Gately, Giuliano, Goff, Faraone, & Seidman, 2009)	Schizophrenia	–	–	0.83	–	–	1.09	–	–	–
	(Piskulic, Olver, Norman, & Maruff, 2007)	Schizophrenia	0.99	–	–	–	–	–	1.03	–	–
	(Stefanopoulou et al., 2009)		0.92	0.95	0.83	0.91	0.68	0.92	0.90	1.28	–
Average ADHD											
	(Alderson, Rapport, & Kofler, 2007)	ADHD children & adolescents		Stroop	Motor	–	–	–	–	–	–
	(Bálint et al., 2009)	ADHD adults	0.72	–	0.63	–	–	–	–	–	–
	(Boonstra et al., 2005)	ADHD adults	0.65	0.30	–	–	–	–	–	–	–
	(Frazier, Demaree, & Youngstrom, 2004a)	ADHD all ages	0.50	0.13	–	–	0.44	0.29	–	0.62	–
	(Hervey, Epstein, & Curry, 2004)	ADHD adults	–	0.56	0.54	–	–	–	–	0.46	0.41
	(Lansbergen, Kenemans, & Van Engeland,	ADHD adults	–	0.15	–	–	–	–	–	–	–
		ADHD adults	–	0.24	–	–	–	–	–	–	–

	2007) (Lijffijt, Kenemans, Verbaten, & Van Engeland, 2005)	ADHD adults	–	–	0.58	–	–	–	–	–	–	–
	(Van Mourik, Oosterlaan, & Sergeant, 2005)	ADHD all ages	–	0.35	–	–	–	–	–	–	–	–
	(Walshaw et al., 2010)	ADHD children and adolescents	0.37	0.38	0.63	–	0.63	–	0.86	0.68	0.38	0.38
	(Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005)	ADHD children and adolescents	0.51	–	0.61	–	0.55	–	0.63	–	–	0.60
Average (all ages)			0.55	0.30	0.63	–	0.54	0.29	0.75	0.59	0.41	0.49
Children & Adolescents Only			0.44	0.23	0.61	–	0.59	–	0.75	0.68	0.38	0.49
Adults Only			0.69	0.38	0.79	–	0.44	0.29	–	0.62	–	–

Note. Summary of meta-analyses conducted in the last ten years. Weighted mean effect size (Cohen's *d*) comparing healthy control participants to the clinical group. All effect sizes have been recoded such that positive values represent worse task performance by the clinical group. When a meta-analysis reported effect sizes for multiple individual tasks within an EF component, the average of these effect sizes is reported.

Manip. = manipulation. Maint. = maintenance. VF = verbal fluency.

Attention

What is attention? Despite William James' famous assertion that "everyone knows what attention is (James, 1890, pp. 403–404)" attention has been conceptualized and defined in many ways, and is best thought of as an umbrella term for a set of processes that filter incoming information and allocate processing resources, including *sustained attention*, *selective attention*, and *divided attention*. Problems with attention can arise either because there are deficits in attentional processes themselves (e.g., difficulty maintaining any attentional focus), or because attention is allocated in a way that is not adaptive (*attentional bias*, e.g., focusing on negative information). There is evidence for both attentional deficits and attentional biases associated with psychopathology, with the strongest evidence for attentional deficits in schizophrenia, bipolar disorder and ADHD, and attentional biases in depression and anxiety disorders.

Sustained Attention. *Sustained attention* or *vigilance* maintains alertness continuously over time. For example, sustained attention is what enables you to stay focused on reading this chapter instead of drifting off into a daydream. Sustained attention is usually measured with continuous performance tests (CPTs), in which a stream of stimuli must be monitored for several minutes to detect infrequent and non-salient targets (e.g., a particular number sequence in a stream of numbers). Sustained attention involves subcortical brain systems that maintain basic arousal and alertness (e.g., reticular activating system), but also the fronto-parietal cognitive control network that also supports EF. This is probably because sustaining attention on tasks like the CPT, while seemingly simple, requires EF processes such as maintaining the task goal and monitoring for task-relevant stimuli. Sustained attention is particularly impaired in individuals with schizophrenia, BD, and ADHD. In contrast, there is limited evidence for sustained attention impairments in individuals with depression or anxiety disorders.

Recent meta-analyses have found impairments with large deficits on sustained attention tasks for individuals with schizophrenia and medium deficits for individuals with BD (Table 4). While most research has focused on adults, deficits in sustained attention have been reported in children and adolescents with schizophrenia (e.g., Groom et al., 2008; Thaden et al., 2006; but see Ueland, Øie, Inge Landrø, & Rund, 2004) and BD (Table 4). A recent meta-analysis also found that adolescents with psychosis risk syndrome have small but significant deficits in sustained attention, which are somewhat larger in those who go on to convert to schizophrenia (Giuliano et al., 2012). Sustained attention deficits appear to be relatively stable over the course of illness: they do not vary with age in individuals with schizophrenia through adulthood and older adulthood (Bozikas & Andreou, 2011; Irani et al., 2011), and persist during euthymia in individuals with BD (Table 4).

As for EF, there is some evidence that sustained attention impairments may be risk markers for psychosis and BD. Adolescents scoring in the high-risk range on a questionnaire for psychotic-like experiences had impaired sustained attention (S. J. Kim et al., 2012). Furthermore, unaffected first-degree relatives of individuals with BD also have significant, though milder, deficits in sustained attention (Bora et al., 2009). On the other hand, there is some evidence that sustained attention impairments develop over the course of psychotic illness: early-onset schizophrenia patients show a failure to improve performance on attention tasks during adolescence, while healthy adolescents do improve performance, suggesting that schizophrenia is associated with a failure in the developmental trajectory (Frangou, 2009).

Inattention symptoms obviously play a central role in ADHD. However, the diagnostic criteria for ADHD in the DSM-IV are not specifically defined in cognitive terms, and behaviors labeled as “inattentive” may not always arise from deficits in attentional processes per se (e.g., behavior labeled “inattentive” might be due to poor EF)(Huang-Pollock, Nigg, & Carr, 2005). Nonetheless, some

diagnostic criteria do appear to describe difficulty sustaining attention. Indeed, there is strong meta-analytic evidence that children, adolescents and adults with ADHD all have impaired performance on laboratory sustained attention tasks, with equal effect sizes across ages, including preschool, suggesting that attentional processes are affected from early in development (Table 4). Specifically, individuals with ADHD discriminate between targets and distracters more slowly and less accurately, and show an increasing number of omission errors over time during task performance, consistent with difficulty sustaining attention, but do not show a bias to make more target responses which would be consistent with impulsive responding (Huang-Pollock, Karalunas, Tam, & Moore, 2012).

There is much less evidence for sustained attention impairments associated with depression and anxiety disorders. There is mixed evidence of impairments in sustained attention associated with depression (e.g., R. Cohen, Lohr, Paul, & Boland, 2001; Godard, Grondin, Baruch, & Lafleur, 2011; but see e.g., Maalouf et al., 2010), PTSD (e.g., Jenkins, Langlais, Delis, & Cohen, 2000; Vasterling et al., 2002)(but see e.g., LaGarde, Doyon, & Brunet, 2010; Twamley, Hami, & Stein, 2004) and OCD (Morein-Zamir et al., 2010; Rajender et al., 2011)(but see e.g., Krishna et al., 2011; Shin et al., 2008), and no evidence of impairment in PD (e.g., Galderisi et al., 2008; Gladsjo et al., 1998).

Selective Attention

Selective attention filters incoming information to select relevant information for further processing. This selection can occur on the basis of sensory modality (e.g., pay attention to visual, not auditory, information), sensory attributes (e.g., look for something blue) or spatial locations (e.g., pay attention to the left side of space). (Selecting information on the basis of more abstract processes or goals is covered under executive function). For example, if you are reading this chapter in a busy coffee shop, selective attention enables you to attend to the words on the page while filtering out the conversations going on around you. Selective attention, especially in the visuospatial domain, is

largely supported by the dorsal attention network (also called the orienting network), which consists of the posterior parietal cortex and frontal eye fields, and is involved in voluntary orienting of attention based on cues (e.g., M. I. Posner & Rothbart, 2007). Somewhat surprisingly given impairments in seemingly more basic sustained attention, selective attention deficits do not appear to be strongly associated with psychopathology, with the possible exception of auditory selective attention in individuals with schizophrenia.

There is some evidence that auditory selective attention may be impaired in individuals with schizophrenia. Specifically, several studies have found that adults with schizophrenia or schizotypal personality disorder were worse at selective attention in dichotic listening tasks, in which two different verbal stimuli are presented simultaneously, one in each ear, with instructions to identify the stimuli in only one ear (Egeland et al., 2003; Hugdahl et al., 2003; Voglmaier et al., 2009). However, these deficits may not be present in younger and stabilized patients (Løberg, Jørgensen, & Hugdahl, 2002).

Unlike auditory selective attention, there is not strong evidence for impairments in visuospatial selective attention associated with schizophrenia. While individuals with schizophrenia are slower overall on measures of selective attention, they generally perform as well as healthy control participants at excluding distracters from attention (Gold, Hahn, Strauss, & Waltz, 2009). Specifically, in Posner spatial cueing tasks, in which a visual cue indicates the probable location of an upcoming stimulus, individuals with schizophrenia are slower to respond overall, but benefit as much from valid cues, and are slowed as much by invalid cues, as healthy control participants (Gold et al., 2009). Similar findings have been reported for visual search tasks, in which targets of a certain shape and color, rather than spatial location, must be selectively focused on (for review, see Gold et al., 2009). Event related potential (ERP) evidence further suggests that individuals with schizophrenia are

as fast as healthy participants to allocate attention to a target, even though their subsequent motor response is slowed (S. Luck et al., 2006).

Mirroring the findings in schizophrenia, there is not clear evidence for impaired visuospatial selective attention in individuals with ADHD despite impaired sustained attention. Specifically, while children with ADHD are slower to respond to cued targets, children with and without ADHD have a similar pattern of response slowing when distracters are present or cues are invalid (Friedman-Hill et al., 2010a; Huang-Pollock et al., 2005). Meta-analytic evidence suggests only small differences in cued visuospatial selective attention between children with ADHD and healthy children ($d = 0.22-0.27$, (Huang-Pollock et al., 2005). This suggests that children with ADHD are not specifically impaired at visually selecting target items or locations. While there is some evidence that children and adults with ADHD have difficulty selectively attending to auditory information (Gomes et al., 2012; Shafritz, Marchione, Gore, Shaywitz, & Shaywitz, 2004), the possibility that these findings could also be due to general slowing rather than selective attention deficits per se requires further investigation.

Interestingly, there is some evidence that children and adults with ADHD do as well as healthy controls at filtering out distracters when task demands are high, but show deficits when task demands are low (easy target discrimination and low distracter salience), suggesting that individuals with ADHD may have trouble endogenously sustaining attention in “easy” tasks, rather than selective attention per se (Friedman-Hill et al., 2010b). Thus, as in schizophrenia, it appears that individuals with ADHD may not have a core deficit in filtering irrelevant visual information, but rather problems with recruiting and sustaining attentional control. This view is consistent with the finding that individuals with ADHD are reliably impaired on sustained attention tasks, which are relatively undemanding and may therefore lead to more lapses in attention.

There has been little research on selective attention in other forms of psychopathology, with mixed reports of impaired and unimpaired selective attention in adults with depression (Egeland et al., 2003; Hammar, Kildal, & Schmid, 2012; Hugdahl et al., 2003) (but see e.g., Desseilles et al., 2009; Godard et al., 2011; Reppermund, Ising, Lucae, & Zihl, 2009), BD (Burdick et al., 2009), PTSD (Grethe E Johnsen, Kanagaratnam, & Asbjørnsen, 2011)(but see e.g., Leskin & White, 2007; Vasterling et al., 2002) and OCD (Clayton, Richards, & Edwards, 1999), but only reports of unimpaired performance in PD (Clayton et al., 1999) and SAD (Sachs, Anderer, Doby, Saletu, & Dantendorfer, 2003). Furthermore, it is not clear if those deficits that have been reported might result from general slowing, as in schizophrenia and ADHD.

Divided Attention. *Divided attention* allocates processing resources to more than one task at once. To return to our coffee shop example, divided attention enables you to continue reading the chapter while at the same time listening for your order to be called. In the laboratory, divided attention is generally assessed with tasks that require dividing attention between two simple tasks at once (e.g., identifying visual and auditory targets). Coordinating two tasks in this way activates the fronto-parietal cognitive control network, even when the individual tasks do not (Collette et al., 2005), suggesting that, like sustained attention, it places demands on EF.

There has been less research on divided attention than other forms of attention. However, those studies which have examined this domain found divided attention impairments in adults (e.g., Daban et al., 2005; Raffard & Bayard, 2012) and adolescents (Boutin, Gingras, & Rouleau, 2010) with schizophrenia, adults with BD (Godard et al., 2011), adults with depression (Godard et al., 2011; Reppermund et al., 2009), and both children/adolescents (e.g., Greimel et al., 2011; Kaufmann et al., 2010) (but see Koschack, Kunert, Derichs, Weniger, & Irle, 2003) and adults (Müller et al., 2007) with ADHD. In addition, non-psychotic relatives of individuals with schizophrenia have impaired

divided attention (Faraone et al., 1999), as do adolescents scoring in the high-risk range on a questionnaire for psychotic-like experiences (S. J. Kim et al., 2012), suggesting it may be an endophenotype or risk factor. However, there has been no research that could shed light on the causal links between divided attention impairments and other forms of psychopathology.

Biased Attention. *Biased attention* is the tendency to direct attention towards, and have difficulty disengaging attention from particular type of affective stimuli, often those that are mood-congruent. Attentional biases can arise both from increased bottom-up salience of the affective stimuli (e.g., because it is mood-congruent) and reduced top-down control of attention. In the laboratory, attentional bias is usually measured in dot-probe tasks, in which visual attention must be directed away from affective stimuli in order to detect a target, and emotional Stroop tasks in which the meaning of affective words must be ignored in order to respond based on the color of the ink the words are written in (see Table 3).

The tendency to inappropriately allocate attention to appropriate emotional cues is central to cognitive models of mood disorders (e.g., Beck, 2008). Meta-analytic evidence demonstrates that individuals with depression have an attentional bias towards negative information, with medium effect sizes on dot-probe tasks, but only small effect sizes on emotional Stroop tasks (Table 4), and may have a bias away from positive stimuli (Peckham, McHugh, & Otto, 2010). Interestingly, attentional biases on the emotional Stroop and dot probe tasks do not appear to be significantly correlated, suggesting that they may index different attentional processes (Daghighi et al., 2003). This could potentially be due to the need to shift visuospatial attention away from negative stimuli in the dot-probe tasks, in comparison to the emotional Stroop task, in which negative information is integral to the task stimuli. Depression is associated with extended maintenance of gaze to dysphoric stimuli in free viewing tasks, as measured with eye tracking (Armstrong & Olatunji, 2012),

suggesting difficulty shifting visuospatial attention away from negative stimuli could potentially account for the stronger effects seen in dot-probe tasks. In contrast to depression, there is limited and mixed evidence for attentional biases toward negative and manic stimuli individuals with BD on emotional Stroop (Besnier et al., 2011)(Lyon, Startup, & Bentall, 1999a) and dot-probe (Lyon, Startup, & Bentall, 1999b)(but see Whitney et al., 2012) tasks. Thus, more research is needed on attentional biases associated with BD.

Attentional biases associated with depression and BD do not seem to be driven by current mood state, and may be risk markers. Negative attentional bias does not occur after sad mood induction (Epp, Dobson, Dozois, & Frewen, 2012) but is present in adults (Peckham et al., 2010) and youth (Hankin, Gibb et al., 2010) with depression in remission, and unaffected relatives of individuals with BD (Besnier et al., 2009; Gotlib, Traill, Montoya, Joormann, & Chang, 2004). Further supporting the hypothesis that attentional biases may be a risk factor, training to modify attentional biases by directing attention towards positive stimuli has been shown to reduce the risk of depression recurrence (Browning, Holmes, Charles, Cowen, & Harmer, 2012b). However, one study did find that only depressed BD patients, and not euthymic BD patients or unaffected relatives of BD patients, demonstrated attentional bias away from positive stimuli (Jabben et al., 2012). Thus, more research is needed to determine if some attentional biases may be mood state dependent.

Like mood disorders, there is evidence for biased attention in anxiety disorders, specifically towards threatening stimuli. In emotional Stroop and dot-probe tasks, individuals with anxiety disorders have a bias towards threat information, with similar effect sizes across disorders, and for children and adults (Table 4). Likewise, individuals with PTSD have biased attention towards PTSD-specific threat words in the emotional Stroop task compared to non-trauma exposed individuals, though they do not differ significantly from trauma-exposed individuals without PTSD (Cisler et al.,

2011). These attentional biases may be driven largely by initial orienting towards threat, as evidenced by larger effects at shorter stimulus durations (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & Van IJzendoorn, 2007) and more frequently initial orienting towards threat during free viewing and visual search (Armstrong & Olatunji, 2012). In contrast, there is not consistent evidence for increased subsequent maintenance of gaze towards threat, although it may occur in some task contexts (Armstrong & Olatunji, 2012). Thus, the factors that influence initial orienting of attention towards threat may be distinct from those that influence later maintenance or shifts of attention (Shechner et al., 2012).

Attentional biases have been relatively infrequently studied in individuals with schizophrenia, but the extant research suggests that schizophrenia may be associated with attentional biases towards disorder-specific stimuli. There is some evidence that individuals with paranoid schizophrenia have increased interference from paranoia-related and threat words in the emotional Stroop task (Besnier et al., 2011; Kinderman, Prince, Waller, & Peters, 2003), but not negative or positive words more generally (Besnier et al., 2011; Demily et al., 2010; Waters, Badcock, & Maybery, 2006). However, it is possible there is a more general bias towards negative stimuli in schizotypy (Mohanty et al., 2005). There has been almost no research on attentional biases in individuals with ADHD, with one recent study finding that children with ADHD did not exhibit the attentional bias towards threat shown by those with anxiety disorders (A. S. Weissman, Chu, Reddy, & Mohlman, 2012). More research is needed in this area, especially with stimuli that may be more affectively relevant to those with ADHD, such as reward stimuli.

Table 3

Examples of commonly used attention measures

Construct	Task	Description	Outcome Measure(s)	Methods Citation¹
Sustained Attention	X-Type Continuous Performance Test	Stimuli (letters, numbers, or pictures) are presented continuously on the computer screen, participant presses a button whenever the target (e.g., X) appears.	Multiple measures including: 1. Commission errors (false alarms) 2. Omission errors 3. RT 4. Changes in RT and accuracy over time.	(Riccio, Reynolds, Lowe, & Moore, 2002)
	AX-Type and Identical Pairs Continuous Performance Test	Stimuli (letters, numbers, or pictures) are presented continuously on the computer screen, participant presses a button whenever the target (e.g., X) appears, preceded by a specific other stimulus (e.g., A), or when two identical stimuli appear in a row	Multiple measures including: 1. Commission errors (false alarms) 2. Omission errors 3. RT 4. Changes in RT and accuracy over time.	(Riccio et al., 2002)
	Conners' Continuous Performance Test	Letters presented continuously on the computer screen, participant presses a button whenever any letter other than X appears.	Multiple measures including: 1. Commission errors (false alarms) 2. Omission errors 3. RT 4. Changes in RT and accuracy over time.	(Strauss et al., 2006)
	Integrated Visual and Auditory Continuous Performance Test (IVA)	Two stimuli (e.g., 1, 2) are presented pseudorandomly, alternating between visual and auditory presentation, participants press a button for targets, regardless of modality	Standardized indexes calculated by the program, based on error and RT patterns.	(Strauss et al., 2006)
	Selective Attention	Ruff 2 & 7 Selective Attention Task	Draw lines through targets (2 & 7) while ignoring other numbers and letters.	1. Correct hits within time limit 2. Errors
	Visual Search (e.g., Test of Everyday Attention search tasks)	Many versions. Search for target stimuli (e.g., a number, letter, shape, or picture) in an array of distracter stimuli.	Vary between versions, generally search time or number of correct hits within a time limit.	(Strauss et al., 2006) for Test of Everyday Attention
	Dichotic Listening	Many versions. Two separate auditory stimulus streams (words or syllables) presented, one to each ear, via headphones. Participants asked to pay attention to one ear and repeat aloud the content.	1. Correct hits from attended ear. 2. Intrusions (false alarms) from unattended ear.	(Hugdahl, 2003)
	Posner Cueing task and similar	Participants must detect targets presented to the left or	Multiple measures including:	(M. I. Posner,

	spatial cueing tasks	right of a fixation point, with or without a preceding cue. Cues are endogenous (central arrows pointing to the left or right) or exogenous (highlighting the left or right target box). On valid trials (usually 80%), the cue indicates the correct target location, on invalid trials (usually 20%), the cue indicates the incorrect target location.	1. Target detection RT 2. Congruent RT – uncued RT (cueing benefit) 3. Incongruent RT – uncued RT (incongruent cueing cost)	Snyder, & Davidson, 1980)
Divided Attention	Dual Task paradigms (e.g., Test of Everyday Attention Telephone Switch While Counting; Test of Attentional Performance divided attention task)	Many versions. Require completing two simple tasks at once, usually one visual and one auditory (e.g. visual search while counting tones in TEA). Performance compared to each task performed separately.	RT or accuracy difference between dual task and single task conditions	(Strauss et al., 2006) for Test of Everyday Attention
Biased Attention	Dot-Probe	Two stimuli (usually faces or other pictures) of differing valence are briefly presented (usually 500 ms) simultaneously on either side of a fixation point, followed by an emotionally neutral probe (e.g., dot, arrow, or letter) at one location, which must be detected/identified.	RT difference to detect/identify the probe in the previous location of the valences vs. the neutral stimulus	(Yiend, 2010)
	Emotional Stroop	Report the color of emotional words and neutral words written in colored ink	RT difference between emotional and neutral words	(Yiend, 2010)

Note. ¹Citations are for sources that provide a full description of the task and outcome measures of the most recent or recommended

version of the task, not necessarily the first publication to report the task.

Table 4

Summary of Recent Attention Meta-Analyses

	Meta-analysis	Group	Sustained Attention	Biased Attention: Dot-probe	Biased Attention: Emotional Stroop
Depression	(Peckham et al., 2010)	MDD	–	0.70	0.16
	(Epp et al., 2012)	MDD			0.24
Average					0.20
BD	(Arts et al., 2008)	Euthymic BD (all)	0.58		
	(Bora et al., 2009)	Euthymic BD ((all)	0.83		
	(Kurtz & Gerraty, 2009)	Euthymic BD ((all)	0.69		
	(Kurtz & Gerraty, 2009)	Manic/mixed BD (all)	0.79		
	(L. J. Robinson et al., 2006)	Euthymic BD (all)	0.60		
	(Torres et al., 2007)	Euthymic BD (all)	0.74		
	(Walshaw et al., 2010)	Pediatric BD (all, clinical status not specified)	0.40		
Average			0.66		
Anxiety Disorders	(Bar-Haim et al., 2007)	OCD, GAD, PTSD, SAD, PD, and simple phobia	-	0.38	0.45
Schizophrenia	(Dickinson et al., 2007)	Schizophrenia	0.92		
	(Mesholam-Gately et al., 2009)	First episode psychosis	0.71		
	(Rajji, Ismail, & Mulsant, 2009)	First episode schizophrenia	0.83		
	Rajji et al., 2009	Youth-onset schizophrenia	0.73		
Average			0.80		
ADHD	(Bálint et al., 2009)	Adults	0.55		
	(Boonstra et al., 2005)	Adults	0.57		
	(Frazier, Demaree, & Youngstrom, 2004b)	All ages	0.61		
	(Hervey et al., 2004)	Adults	0.64		
	(Huang-Pollock et al., 2012)	Children and adolescents	0.59		
	(Pauli-Pott & Becker, 2011)	Preschool Children	0.54		
	(Schoechlin & Engel, 2005)	Adults	0.52		
	(Walshaw et al., 2010)	Children and adolescents	0.56		
	(Willcutt et al., 2005)	Children and adolescents	0.58		
	Children & Adolescents only			0.57	
Adults Only			0.57		
Average (all)			0.58		

Note. Summary of meta-analyses conducted in the last ten years. Weighted mean effect size (Cohen's *d*) comparing healthy control participants to the clinical group. All effect sizes have been recoded such that positive values represent worse task performance by the clinical group. When a meta-analysis reported effect sizes for multiple individual tasks within a EF component, the average of these effect sizes is reported.

Memory

Much of what we do in our daily lives, and indeed, who we are, depends on memory. There are multiple memory systems and memory processes, including *episodic memory* (memory for specific events), *semantic memory* (memory for facts and concepts), and *autobiographical memory* (memory for specific life events and facts about the self)¹. Memory problems are common in psychopathology, but the nature and magnitude of these impairments varies across disorders. While most forms of psychopathology are associated with impairments in episodic memory, only schizophrenia is associated with substantial impairments in semantic memory, and autobiographical memory may be particularly affected in schizophrenia, depression and PTSD.

Episodic Memory. *Episodic memory* refers to memories for specific events within a spatio-temporal context (e.g., yesterday, I sat in my office and read papers about episodic memory), and relies heavily on the hippocampus and surrounding areas of the medial temporal lobe (e.g., Eichenbaum, 2004). In the laboratory, episodic memory is assessed by asking participants to study stimuli (e.g., word lists or complex designs), then recall or recognize them after a delay. These tasks thus allow pure episodic memory to be separated from autobiographical memory for personally significant events. Many forms of psychopathology are associated with episodic memory impairments on these tasks, although the magnitude of these impairments varies both across disorders and across memory tests.

Adults with schizophrenia and bipolar disorders have similar profiles of episodic memory impairments, albeit with larger effect sizes for individuals with schizophrenia. Meta-analytic evidence demonstrates that individuals with schizophrenia and BD have large and medium impairments

¹ Here we focus on explicit (episodic and semantic) memory, which has been most often studied in relation to psychopathology. Skill learning and conditioning can also be considered forms of memory (implicit memory), but are beyond the scope of this chapter. Working memory is covered in the section on executive function.

respectively in episodic memory (Table 5). Impairments are similar for recall of verbal and non-verbal information, and for recall after short and long delays (Table 5). There is some evidence that deficits in recognition are somewhat smaller than those for recall, although recognition memory has been less studied (Table 5). While there have been relatively few studies of episodic memory in children and adolescents with BD or early-onset schizophrenia, they seem to have somewhat comparable levels of impairment as adults with these disorders (Frangou, 2009; Joseph, Frazier, Youngstrom, & Soares, 2008). A recent meta-analysis also found that adolescents with psychosis risk syndrome have small but significant deficits in both verbal and non-verbal episodic memory, which are somewhat larger in those who go on to convert to schizophrenia (Giuliano et al., 2012).

There is some evidence that episodic memory impairments may be a stable trait in individuals with schizophrenia and BD. Deficits in individuals with BD are not closely tied to current mood state, as they occur in euthymic BD (Table 5), although there is some evidence that they may be somewhat larger in manic/mixed and depressed BD (Kurtz & Gerraty, 2009). In addition, in individuals with schizophrenia, meta-analyses of longitudinal studies have found no increase in deficits over relatively brief periods (Irani et al., 2011; Szöke et al., 2008). However, declines may occur over a longer time period, or may have already occurred since many studies included individuals with chronic schizophrenia.

There has been less research on episodic memory in other forms of psychopathology, but the available evidence points to episodic memory impairments in most other disorders as well. Adults with depression appear to have impairments in both verbal and non-verbal episodic memory (Table 5; but see (R. S. C. Lee, Hermens, & Porter, 2012) for evidence of impaired non-verbal but not verbal episodic memory in first episode MDD). There has been no systematic examination as to whether recall and recognition are differentially affected. In addition, there has been little research on episodic memory in

children/adolescents with or at risk for depression, although a few studies have found deficits in verbal (Günther, Holtkamp, Jolles, Herpertz-Dahlmann, & Konrad, 2004; Horan, Pogge, Borgaro, Stokes, & Harvey, 1997) and non-verbal (Matthews & Coghill, 2008a) episodic memory.

At least some anxiety disorders are also associated with episodic memory impairments. Individuals with PTSD have impairments in verbal episodic memory recall (after both short and long delays), but only small impairments in non-verbal recall (Brewin, Kleiner, Vasterling, & Field, 2007). An important question is whether these deficits are due to PTSD per se, or the traumatic experiences themselves. Both appear to contribute: individuals with PTSD have significant verbal episodic memory impairments even compared to trauma-exposed individuals without PTSD, but have larger impairments compared to healthy, non-trauma-exposed control participants (Table 5)(G E Johnsen & Asbjørnsen, 2008). While most research included in these meta-analyses is on adults, studies in children with maltreatment-related PTSD have also reported episodic memory impairments (Carrion et al., 2012). However, there is some evidence that co-occurring depressive symptoms may account for episodic memory impairments in individuals with PTSD (Burriss, Ayers, Ginsberg, & Powell, 2008), and more research is needed to disentangle to what extent memory impairments are driven by factors specific to PTSD versus co-occurring depression or transdiagnostic processes.

A meta-analysis of compulsive checkers (most with a diagnosis of OCD) found significant, albeit moderate, impairments in verbal and non-verbal recall, and non-verbal recognition, although verbal recognition was not impaired, possibly due to ceiling effects (Table 5; (Woods, Vevea, Chambless, & Bayen, 2002). There has been no meta-analysis of memory in individuals with OCD more broadly, but recent reviews report mixed findings, with stronger evidence for impairments on non-verbal than verbal episodic memory tasks (Castaneda, Tuulio-Henriksson, & Marttunen, 2008; Cuttler & Graf, 2009; Olley, Malhi, & Sachdev, 2007). In addition, research has focused almost entirely on adults, and the few

studies in children and adolescents with OCD again show mixed findings of impaired (Andrés et al., 2007) and unimpaired (Ornstein, Arnold, Manassis, Mendlowitz, & Schachar, 2010; Shin et al., 2008) episodic memory. There has been very little research on memory function in individuals with other anxiety disorders. One study found significant verbal episodic memory impairments associated with PD and SAD, but not generalized anxiety disorder or specific phobias (Airaksinen et al., 2005; Galderisi et al., 2008). However, the lack of significant effects for panic disorder and generalized anxiety disorder must be interpreted with caution since the very small sample sizes limited power.

Finally, meta-analytic evidence demonstrates that adults with ADHD also have impairments in verbal episodic memory recall (after both short and long delays), and recognition (Table 5). While there has been no meta-analysis, individual studies have also found episodic verbal memory deficits in children and adolescents with ADHD (Cutting, Koth, Mahone, & Denckla, 2003; Udal, Øyegarden, Egeland, Malt, & Groholt, 2012)(but see Kibby & Cohen, 2008; Vakil, Blachstein, Wertman-Elad, & Greenstein, 2012). However, there is little evidence of deficits in non-verbal episodic memory (Table 5). This pattern suggests that factors other than episodic memory per se may be contributing to the verbal memory deficits in individuals with ADHD. Since reading and language disorders frequently co-occur with ADHD (e.g., Willcutt, Pennington, & DeFries, 2000), it is possible that difficulty processing verbal information may play a role, although even children without co-occurring reading disabilities have been reported to have verbal episodic memory impairments (Cutting et al., 2003; Kibby & Cohen, 2008).

Overall, the pattern of episodic memory impairments across disorders suggests a domain-general deficit in encoding or retrieval. First, deficits are not greater for recall after a longer delay compared to a shorter delay in any disorder where this has been investigated. This suggests that psychopathology is associated with difficulty in encoding and/or retrieval, rather than forgetting more information over time (storage). Second, in most disorders, memory for both verbal and non-verbal materials is impaired,

suggesting deficits are mostly domain-general, although the magnitude of deficits occasionally differs between domains, perhaps due to additional domain-specific deficits (e.g., visuospatial processing in OCD, (Rampacher et al., 2010)). There are two possible exceptions, PTSD and ADHD, in which there is little evidence for non-verbal episodic memory impairments; however, as there has been less research on non-verbal memory in these disorders, it is premature to conclude that it is not impaired. Third, in some cases recall is more impaired than recognition. While recall requires retrieval processes supported by the hippocampus and PFC, recognition can be accomplished based on familiarity, which is supported by areas surrounding the hippocampus (Yonelinas, 2002).

Thus, it has been proposed that impaired hippocampal binding and prefrontally mediated use of strategies for encoding and retrieval lead to particular impairments in episodic recall for individuals with psychopathology (e.g., Barch & Ceaser, 2012). Indeed, there is meta-analytic evidence for reduced hippocampal volume and/or alterations in hippocampal functional activation in individuals with multiple forms of psychopathology, including schizophrenia (Adriano, Caltagirone, & Spalletta, 2012), BD (Frey et al., 2007); depression (Arnone, McIntosh, Ebmeier, Munafò, & Anderson, 2012), and PTSD (compared to both trauma-exposed and unexposed controls; (Kühn & Gallinat, 2013; Woon, Sood, & Hedges, 2010); although effects have been much less consistently found in children, (Carrion et al., 2012).

There is some evidence suggesting that hippocampally-mediated memory problems may be risk factors or endophenotypes for psychopathology, rather than a neurodegenerative effect of illness. Hippocampal volume is reduced in at-risk individuals before psychosis onset (Fusar-Poli, Radua, McGuire, & Borgwardt, 2012), and is equally reduced in first episode and chronic schizophrenia (Adriano et al., 2012; Mesholam-Gately et al., 2009; Pelletier, Achim, Montoya, Lal, & Lepage, 2005) for increased recognition deficits with illness duration). Likewise, individuals who had lower episodic

memory recall performance pre-trauma subsequently developed more PTSD symptoms following a natural disaster (Parslow & Jorm, 2007) or deployment to a war zone (Marx, Doron-Lamarca, Proctor, & Vasterling, 2009). Moreover, unaffected relatives of individuals with schizophrenia, BD and PTSD also have smaller hippocampal volumes and moderate impairments in episodic memory (Arts et al., 2008; Barch, 2005; Bora et al., 2009; Gilbertson et al., 2006).

While this evidence suggests that hippocampal dysfunction may be an endophenotype, there is some evidence that neurodegenerative processes may also play a role. Specifically, stress has neurotoxic effects on the hippocampus, which has a particularly high concentration of glucocorticoid receptors (MacQueen & Frodl, 2010), suggesting that stress associated with psychopathology may contribute to hippocampal dysfunction. However, there is mixed and inconclusive evidence that hippocampal dysfunction increases with longer illness duration (MacQueen & Frodl, 2010). Rather, hippocampal dysfunction may arise from a combination of genetic factors and early life stress, and in turn increase risk for psychopathology (Pechtel & Pizzagalli, 2010).

Why might poor memory function serve as a risk factor? One possibility is that the ability to effectively solve problems depends in part on retrieval of information about similar past situations, and that poor problem solving increases stress and risk for psychopathology (e.g., Sutherland & Bryant, 2008). Moreover, under stress memories become more rigid and habitual, potentially contributing to a lack of flexibility in thinking and problem solving which could contribute to psychopathology (Schwabe, Wolf, & Oitzl, 2010).

Semantic Memory. *Semantic memory* is knowledge of facts and concepts, which are not tied to a particular time or place (e.g., knowing what semantic memory means, without remembering where and when you learned this). Semantic memory is generally assessed by asking people to define words, answer general factual questions, or sort items based on semantic categories or similarity. Semantic

memory depends on a distributed network of cortical areas that represent specific sensory properties (e.g., shapes and colors in visual association cortex, sounds in auditory association cortex) as well as anterior lateral temporal lobe and prefrontal areas involved in more abstract semantic processing (e.g., Patterson, Nestor, & Rogers, 2007).

Unique among the disorders reviewed here, meta-analytic evidence demonstrates that schizophrenia is associated with large impairments in semantic memory (Table 5). What is responsible for these semantic memory impairments? First, since many semantic memory tasks assess vocabulary and factual knowledge, performance could be affected by education; however, individuals with schizophrenia are impaired even compared to education-matched healthy control participants (Doughty & Done, 2009). Second, effect sizes are generally large for picture naming and vocabulary tests requiring language production, and moderate but still significant for picture matching and sorting tasks which do not require a verbal response (Doughty & Done, 2009). This suggests that language impairments may contribute to poor performance on verbal semantic memory tasks, but do not fully account for semantic memory impairments. Third, deficits are present even for naming and picture matching tasks that have few executive function demands and are generally preserved in individuals with prefrontal damage, suggesting that semantic memory deficits are also not solely due to EF impairments (Done, 2009).

Rather, schizophrenia may be associated with abnormalities in semantic associations themselves. Individuals with schizophrenia show bizarre associations, idiosyncratic categorization during sorting tasks, and increased semantic priming (especially indirect priming, e.g., tiger-stripe, (Pomarol-Clotet, Oh, Laws, & McKenna, 2008), consistent with a loosening of associations caused by excessive spreading activation through semantic networks (Doughty & Done, 2009). These abnormalities in semantic associations are more severe for those with formal thought disorder, and have been posited to

play a causal role in thought disorder (Pomarol-Clotet et al., 2008). However, while large deficits in semantic memory are already present in first episode psychosis (Mesholam-Gately et al., 2009), it is not known whether they precede illness onset.

In contrast to schizophrenia, semantic memory appears to only minimally impaired at most in individuals with other forms of psychopathology. Adults with BD show no deficit in vocabulary measures of semantic memory (Table 5). An older meta-analysis of adults with depression suggests only a small effect size on measures of semantic memory (Zakzanis, Leach, & Kaplan, 1998)(Table 5), and a number of more recent studies have found no impairment on measures of semantic memory (Castaneda, Suvisaari, et al., 2008; Herrera-Guzmán et al., 2010; Matthews & Coghill, 2008b)(but see Portella et al., 2003; Ravnkilde et al., 2002). ADHD is also associated with small deficits in semantic memory in both adults and children (Table 5), but as for verbal episodic memory, these deficits could potentially be driven by language difficulties, given that semantic memory is assessed with vocabulary and other verbal measures. There has been no meta-analysis of semantic memory in individuals with OCD, but individual studies suggest that performance on measures of vocabulary and semantic knowledge are not impaired (Andrés et al., 2007; Shin et al., 2008; Tekcan, Topçuoğlu, & Kaya, 2007) (but see Exner, Martin, & Rief, 2009). There has been virtually no research on semantic memory in individuals with other anxiety disorders.

Autobiographical Memory. *Autobiographical memory*, which refers to memory about the self and one's own life, is considered to be a mixture of episodic memory for specific life events, and semantic memory for facts about the self (e.g., Renault, Davidson, Palombo, Moscovitch, & Levine, 2012). Autobiographical memory is usually assessed using interview methods, in which people are asked to recall a series of autobiographical events, and responses are scored for specificity. In the Autobiographical Memory Test (AMT, J. M. Williams & Broadbent, 1986), participants are asked to

retrieve a memory specific to a time and place in response to a series of positive and negative cue words (e.g., safe, embarrassed), and responses are coded as specific (reflecting a specific event lasting no longer than a day), categorical (reflecting a repeated event), or extended (reflecting events extended over a long period of time). Autobiographical memory is considered *over general* when most memories retrieved are categorical or extended rather than specific. Importantly, memories scored as specific in the AMT can contain both episodic and semantic details (Levine, Svoboda, Hay, Winocur, & Moscovitch, 2002). In contrast, the Autobiographical Memory Interview (AMI, Kopelman, Wilson, & Baddeley, 1990) and Autobiographical Interview (Levine et al., 2002) provide indexes of both episodic and semantic autobiographical memory, by asking participants to recall specific life events and facts about the self, which are scored for the level of detail.

The main focus on autobiographical memory impairments has been on individuals with depression and PTSD. Over-general autobiographical memory in individuals with depression and PTSD is hypothesized to arise from (1) capture of attention by self-relevant information, activating ruminative processes that interfere with retrieval, (2) avoidance of retrieval of specific memories to regulate emotion, and (3) deficits in EF that limit the ability to successfully conduct retrieval searches of memory (for review, see Sumner, 2012). Meta-analytic evidence demonstrates that individuals with depression have large deficits in reporting specific autobiographical memories ($d = 1.12$, (J. M. G. Williams et al., 2007). It is unclear if these deficits are specific to episodic versus semantic autobiographical memory, as most studies have used the AMT, but one recent study using the AMI reported impairment only on episodic autobiographical memory (Semkovska, Noone, Carton, & McLoughlin, 2012). Deficits in autobiographical memory associated with depression are not valence specific, as they have been reported for positive, negative and neutral memory cues (see King et al., 2010 for review). In addition, autobiographical memory impairments persist during remission from depression, and thus are not

dependent on current mood state (King et al., 2010).

Over-general autobiographical memory has been found in individuals with depression from childhood through older adulthood, although no systematic analysis of age-related changes has been conducted (King et al., 2010). The presence of autobiographical memory impairments early in life and depression course is consistent with additional evidence that autobiographical memory deficits may be associated with risk for depression. Over-general autobiographical memory predicts the onset of depression (but not anxiety or externalizing disorders) in adolescents (Hipwell, Sapotichne, Klostermann, Battista, & Keenan, 2011; Rawal & Rice, 2012), and future levels of depressive symptoms in adults even controlling for baseline symptoms (see Sumner, Griffith, & Mineka, 2010 for meta-analysis), although these predictive effects are small.

Despite detailed and intrusive memories of trauma events, there is also strong evidence for over-general autobiographical memory of non-trauma events in individuals with PTSD. PTSD and acute stress disorder are associated with reduced specificity of autobiographical memories, and this deficit cannot be accounted for by co-occurring depression or trauma exposure alone (see Brewin, 2011; Moore & Zoellner, 2007 for review). Indeed, the majority of the literature is not consistent with the view that trauma exposure alone (independent of PTSD symptoms) either in childhood or adulthood, leads to over-general autobiographical memory (Moore & Zoellner, 2007). Rather, there is some evidence that poor autobiographical memory may be associated with risk for developing PTSD: trainee firefighters who had more over-general autobiographical memory retrieval went on to develop more PTSD symptoms after three years of firefighting experience, during which they encountered multiple traumatic events (Bryant, Sutherland, & Guthrie, 2007). As in the depression literature, studies of autobiographical memory in individuals with PTSD have relied almost entirely on the AMT, which does not provide a separate measure of semantic autobiographical memory. However, there are reports that PTSD

symptoms (Moradi et al., 2008), and traumatic experiences (Hunter & Andrews, 2002; Stokes, Dritschel, & Bekerian, 2008) are associated with worse semantic autobiographical memory.

Although autobiographical memory deficits associated with schizophrenia are less discussed, individual studies find evidence of impairment. Not surprisingly given impairments in both episodic and semantic memory, individuals with schizophrenia have difficulty recalling both autobiographical episodic and semantic memories, as indexed by both the number of memories recalled and their specificity and detail (e.g., McLeod, Wood, & Brewin, 2006; Neumann, Blairy, Lecompte, & Philippot, 2007). These deficits are not attributable to co-occurring depression or differences in premorbid IQ (McLeod et al., 2006). Autobiographical memories from all life stages, including early childhood, are affected, suggesting that the primary deficit arises during retrieval or that encoding is affected from early in development (McLeod et al., 2006). However, the largest impairment is for events and facts around the time of each individual's illness onset, suggesting that poor encoding during this time may contribute to autobiographical memory deficits (e.g., Riutort, Cuervo, Danion, Peretti, & Salamé, 2003). Interestingly, individuals with schizophrenia appear to also have difficulty generating detailed descriptions of possible future events (*prospective memory*; (D'Argembeau, Raffard, & Van der Linden, 2008), which involves the same brain network activated by retrospective memory processes (e.g., Schacter, Addis, & Buckner, 2008).

There is very little research on autobiographical memory in individuals with other disorders. One study found over-general autobiographical memory in adults with BD (Scott, Stanton, Garland, & Ferrier, 2000). One study found that individuals with OCD had over-general autobiographical memory, but that this was driven by co-occurring depression rather than OCD per se (Wilhelm, McNally, Baer, & Florin, 1997). In general, autobiographical memory does not appear to be impaired in individuals with other anxiety disorders, including SAD (Heidenreich, Junghanns-Royack,

& Stangier, 2010; Wenzel, Jackson, & Holt, 2002), or GAD/high worry (Finnbogadóttir & Berntsen, 2011; Wenzel & Jordan, 2005). Autobiographical memory has not been investigated in individuals with ADHD.

Thus, there is strong evidence for autobiographical memory impairments only in depression, PTSD and schizophrenia. However, given the limited amount of research in individuals with other disorders, it is premature to draw conclusions about the specificity of autobiographical memory deficits to these disorders.

Table 4

Examples of commonly used memory measures

Construct	Task	Description	Outcome Measure(s)	Methods Citation¹
Verbal Episodic Memory	California Verbal Learning Test (CVLT)	Semantically structured list of 16 items presented for five learning trials, each followed by recall. Then a second 16 word interference list is presented for one trial, followed by free and cued recall of the first list. After 20 min. of distracting tasks, first list free and cued recall or repeated, then recognition test.	Many outcome parameters, including: 1. Short delay free and cued recall. 2. Long delay free and cued recall. 3. Recognition (hits, false alarms, discrimination index).	Straus et al., 2006
	Rey Auditory Verbal Memory Test (RAVLT)	List of 15 words read aloud by experimenter for five learning trials, each followed by recall. Then a second 15word interference list is presented for one trial, followed by free recall of the first list. After a 20 min. delay, free recall of first list repeated, then recognition of first list words.	Many outcome parameters, including: 1. Short delay free and cued recall. 2. Long delay free and cued recall. 3. Recognition (hits, false alarms, discrimination index).	Straus et al., 2006
	Wechsler Memory Scale (WMS-III) Word List	Recall a list of 12 words after each of four learning trials, after presentation of an interference list, and again after a delay. Finally, a recognition trial is given.	1. Immediate recall 2. Delayed recall 3. Recognition	Straus et al., 2006
	Wechsler Memory Scale (WMS-III) Logical Memory	Recall two paragraphs read aloud by the experimenter immediately and after a delay, followed by a yes/no recognition test.	1. Immediate recall 2. Delayed recall 3. Recognition	Straus et al., 2006
	Wechsler Memory Scale (WMS-III) Verbal Paired Associates	Experimenter presents a list of word pairs, then reads the first word in each pair and participant must respond with the second word. Recall is re-tested after a delay, followed by a recognition test.	1. Immediate recall 2. Delayed recall 3. Recognition	Straus et al., 2006
Non-Verbal Episodic Memory	Benton Visual Retention Test (BVRT)	Geometric figure is displayed for 10 S. and then withdrawn. Participant must copy figure from memory.	1. Number of correct reproductions. 2. Error score.	Straus et al., 2006
	Brief Visuospatial Memory Test	Participant views six simple geometric patterns in a grid	Many outcome parameters,	Straus et al., 2006

	(BVMT)	for 10 S., which is then withdrawn. Participant must reproduce figures in correct locations from memory. Followed by two more learning and recall trials with same stimuli, then recall again after 25 min. of distracting tasks, followed by recognition trial.	including: 1. Recall on first three trials (short delay recall) 2. Delayed recall 3. Recognition (hits, false alarms, discrimination index).	
	Rey-Osterrieth Complex Figures Test (ROCF)	A complex geometric figure is presented. Administration procedures differ, but usually participants are asked to copy the figure, then it is withdrawn and participants must reproduce it from memory after a 3 min. delay and again after a 30 min. delay, which may be followed by a recognition trial.	Many outcome parameters, including: 1. Short and long delay recall accuracy (points for each part of figure correctly reproduced). 2. Recognition (points for each part of figure recognized correctly).	Straus et al., 2006
	Wechsler Memory Scale (WMS-III) Visual Reproduction	Participant must reproduce geometric figures both immediately and after a delay. Recognition can also be tested.	1. Immediate recall 2. Delayed recall 3. Recognition	Straus et al., 2006
	Wechsler Memory Scale (WMS-III) Faces	Participant must recognize faces both immediately after presentation and after a delay.	1. Immediate recognition 2. Delayed recognition	Straus et al., 2006
Semantic Memory	Expressive Vocabulary Test (EVT), Boston Naming Test, and similar picture naming tests	Label pictures with increasing item difficulty. Some tests (e.g., EVT) also require providing synonyms for some items.	Scores based on number of items correct.	Straus et al., 2006
	Peabody Picture Vocabulary Test (PPVT) and similar picture recognition tests	Point to pictures corresponding to a label from an array of four pictures.	Scores based on number of items correct.	Straus et al., 2006
	Wechsler Adult Intelligence Scale (WAIS) Vocabulary and similar vocabulary tests	Combination of naming pictures and defining words.	Standardized scores following scoring manual.	Straus et al., 2006
	Wechsler Adult Intelligence Scale (WAIS) Information and similar semantic information tests	Answering questions about a broad range of general knowledge, including scientific and geographical facts, history, and literature.	Standardized scores following scoring manual.	Straus et al., 2006
	Pyramid and Palm Trees/ Camel and Cactus	On each trial, point to one of four pictures that goes best with a target picture (e.g., target = bottle of wine, choices = apple, banana, grapes, pear).	Accuracy	(Bozeat, Lambon Ralph, Patterson, Garrard, & Hodges,

				2000; Howard & Patterson, 1992)
	Categorization/Sorting tasks	A variety of tasks, involving asking whether an item belongs to a certain category or not, or sorting items into categories, at different category levels (e.g., animals vs. plants; birds vs. mammals)	Accuracy	Doughty et al., 2009
	Semantic Priming	A simple task, usually lexical decision (word or non-word) is performed, with each word preceded by a semantically related word (prime) or unrelated word. Primes can be direct (e.g., tiger-lion) or indirect (e.g., stripes-lion).	Difference in RT between primed and unprimed words.	(Perea & Rosa, 2002)
Autobiographical Memory	Autobiographical Memory Interview	Semi-structured interview assessing semantic autobiographical memory (e.g., names of teachers) and episodic autobiographical memory (e.g., describing specific events) from childhood, early adulthood, and recent past.	1. Number of semantic facts recalled from each time period. 2. Points based on number and detail (time and place) of episodic events recalled from each time period.	Straus et al., 2006
	Autobiographical Memory Test	Participants are presented with words (e.g., safe, clumsy) and asked to retrieve a memory specific to time and place and describe it for 30-60 S.	Memories scored as categoric (repeated event), extended (lasting more than one day), or specific (reflect a unique occurrence lasting no longer than a day).	(J. M. Williams & Broadbent, 1986)
	TEMPau Task	Participants instructed to recall specific events (lasting less than one day) from different life periods, and classify them as remember (specific episodic details, sense of mental time travel), know (semantic details, sense of familiarity only), or guess (not certain about any details).	1. Overall and episodic specificity scores 2. What, where and when remember and know scores 3. Self-perspective (field vs. observer)	(Piolino, Desgranges, & Eustache, 2009)

Note. ¹Citations are for sources that provide a full description of the task and outcome measures of the most recent or recommended version of the task, not necessarily the first publication to report the task.

Table 5

Summary of memory meta-analyses

	Meta-analysis	Group	Short Delay Verbal Recall	Long Delay Verbal Recall	Short Delay Non-Verbal Recall	Long Delay Non-Verbal Recall	Verbal Recognition	Non-Verbal Recognition	Semantic Memory
Depression	(R. S. C. Lee et al., 2012)	First episode MDD		0.13		0.53			
	(Zakzanis et al., 1998) ¹	MDD	0.79	0.95	0.86	0.65			
	(Herrmann, Goodwin, & Ebmeier, 2007)	Early onset MDD			0.48 (episodic memory composite)				
	(Herrmann et al., 2007)	Late onset MDD			0.44 (episodic memory composite)				
BD	(Arts et al., 2008)	Euthymic BD (all)	0.82	0.85	–	–	–	–	–
	(Bora et al., 2009)	Euthymic BD (all)	0.33	0.27	–	–	0.44	0.59	–
	(Bora et al., 2011)	Euthymic BD II	–	–	–	–	0.22	–	–
	(Kurtz & Gerraty, 2009)	Euthymic BD (all)	0.74	0.80	0.78	–	–	0.86	–
	(Kurtz & Gerraty, 2009)	Manic/mixed BD (all)	–	1.05	–	–	–	–	–
	(Mann Wrobel et al., 2011)	Euthymic BD (all)	0.67	0.80	0.67	–	0.42	–	0.09
	(L. J. Robinson et al., 2006)	Euthymic BD (all)	0.73	0.71	–	–	–	–	–
	(Stefanopoulou et al., 2009)	BD (all)	–	0.99	–	–	0.71	–	–
	(Torres et al., 2007)	Euthymic BD (all)	0.74	0.72	0.43	–	–	–	0.08
Average			0.67	0.77	0.63	–	0.45	0.73	0.09
OCD	(Woods et al., 2002)	Compulsive checkers	0.51		0.66		-0.17	0.49	–
PTSD	(Brewin et al., 2007)	PTSD	0.56	0.46	0.23	0.23			
Schizophrenia	(Dickinson et al., 2007)	Schizophrenia	1.19	1.19	0.93	0.78	–	–	0.86
	(Doughty & Done, 2009)	Schizophrenia	–	–	–	–	–	–	0.78
	(Forbes et al., 2009)	Schizophrenia	1.11	–	0.91	–	0.63	–	–
	(Julie Henry & Crawford, 2005)	Schizophrenia	–	–	–	–	–	–	0.77
	(Mesholam-Gately et al., 2009)	First-episode psychosis	1.20	0.90	0.95	0.90	0.57	–	0.84
(Pelletier et al., 2005)	Schizophrenia	–	–	–	–	0.71	1.00	–	

	(Stefanopoulou et al., 2009)	Schizophrenia		1.26	–	–	0.45	–	–
Average			1.17	1.12	0.93	0.84	0.59	1.00	0.81
ADHD	(Frazier et al., 2004b)	ADHD all ages	–	–	–	0.26	–	–	0.46
	(Hervey et al., 2004)	ADHD adults	0.59	0.60	0.01	0.19	0.90	–	0.29
	(Schoechlin & Engel, 2005)	ADHD children and adolescents	–	–	–	–	–	–	0.27
Average (all ages)			0.59	0.60	0.01	0.23	0.90	–	0.34

Note. Summary of meta-analyses conducted in the last ten years. Weighted mean effect size (Cohen's *d*) comparing healthy control participants to the clinical group. All effect sizes have been recoded such that positive values represent worse task performance by the clinical group. When a meta-analysis reported effect sizes for multiple individual tasks within a EF component, the average of these effect sizes is reported.

¹ This older meta-analysis is included due to the lack of more recent meta-analyses covering multiple aspects of memory in individuals with MDD.

Gender, cultural, and ethnic differences and considerations. Basic cognitive processes are widely assumed to be human universals, and so not to vary greatly across cultures. Thus, while the research discussed above participants from many countries (including Asian, European, North American, and Latin American countries), there have been no systematic comparisons of cognitive process deficits across cultures. Socioeconomic status (SES) is perhaps a more likely moderator, as low SES is associated with worse performance on many cognitive tasks in both children and adults, with executive function perhaps most affected (see Hackman & Farah, 2009 for review). While many of these effects in adults are likely driven by education, which strongly influences performance on many tasks, SES effects are also found in infants and young children, thus education cannot be the only driving factor. Rather, there are multiple candidate causal pathways, including differences in parenting and chronic stress (Hackman & Farah, 2009). Since these factors are also known to affect risk for psychopathology, it seems plausible that SES could moderate the link between psychopathology and cognitive function. However, to our knowledge this has not been directly studied.

Gender has received somewhat more attention, given the differential rates of many forms of psychopathology between males and females. However, despite gender differences in psychopathology, there is not strong evidence that gender moderates the association between psychopathology and cognitive processes. Many meta-analyses have not examined gender as a potential moderator, and some that have done so have found that gender does not moderate cognitive impairments associated with psychopathology, including EF and attentional bias in depression (Peckham et al., 2010; Snyder, 2013), EF and episodic memory BD II disorder (Bora et al., 2011), and EF in ADHD (e.g., Lipszyc & Schachar, 2010; van Mourik et al., 2005). On the other hand, some meta-analyses have reported gender differences, including larger impairments in BD samples with more female participants on two measures of the shifting component of EF (Arts et al., 2008; Kurtz & Gerraty, 2009), but larger impairments for

samples with more male participants in first-episode psychosis on measures of verbal episodic memory (Mesholam-Gately et al., 2009), and in ADHD for a measure of the inhibition component of EF (Bálint et al., 2009). However, these effects are generally only found only for one task, are not consistent across meta-analyses, and may not remain significant if other confounding moderators are controlled for (e.g., see (Lipszyc & Schachar, 2010) for an apparent effect of gender on inhibition in ADHD which is no longer significant controlling for age and study quality). Thus, while gender effects merit further investigation, current evidence suggests that it is not a robust moderator of cognitive process deficits associated with psychopathology.

Cognitive Products

Cognitive Styles

The following section outlines well-established maladaptive patterns of explaining stressful events or situations. Some cognitive styles (e.g. negative inferential style, hostile attributional bias, looming maladaptive style) are event-specific, and although the style is consistent, the particular cognitions generated in response to each event are context dependent. Conversely, other cognitive styles (e.g. dysfunctional attitudes, early maladaptive schemas) are based upon stable mental representations, and are believed to be consistent across situations. Not only do these cognitive patterns involve assigning meaning to stressful situations in the environment, but they also involve the processing of self-related concepts.

Neuroimaging studies of cognitive styles have shown activation of the medial prefrontal cortex, which is associated with autobiographical content, as well as the anterior cingulate cortex, which is responsible for labeling incoming information as self-referential (for a review, see Disner, Beevers, Haigh, & Beck, 2011). Furthermore, these studies have found increased activation of the amygdala and hippocampus—limbic structures involved in emotional processing (see Disner et al., 2011). These

particular regions exhibit increased activity among participants with psychopathology, such as depression (Cooney, Joormann, Eugène, Dennis, & Gotlib, 2010) and social anxiety (Blair et al., 2008). Therefore, neuroimaging studies suggest that maladaptive patterns of self-referential processing in the context of stressful life events may be associated with emotional distress, and consequently, psychopathology.

Negative inferential style. Broadly defined, negative inferential style refers to an individual's characteristic pattern of explaining the meaning, causes, and consequences of stressful life events. One particular type of negative inferential style was outlined originally in the reformulated learned helplessness theory of depression (Abramson et al., 1978), which maintains that individuals who attribute negative events to internal (i.e., about the self), stable (i.e., enduring), and global (i.e., widespread) causes are said to possess a negative attributional style. An extension of the reformulated learned helplessness theory is the hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989). According to this model, individuals not only possess a negative attributional style for the *causes* of stressful life events, but they also make stable and global inferences regarding the *consequences* of an event and believe that a stressful life event has negative implications for the *self* (e.g., the self is deficit, flawed, or worthless).

Empirical research examining the role of negative inferential style as a cognitive vulnerability to psychopathology has found support in children, adolescents, and adults, although relatively less research with child samples suggests that more research needs to be conducted in this early cohort to fully understand the nature of the relationship between negative inferential style and psychopathology. More importantly, a substantial body of research has found that negative inferential style interacts with negative life events to predict changes in psychopathology, including symptoms and clinical diagnosis, over time. Although originally formulated as a theory to explain vulnerability to depression, it appears

as though a negative attributional style is present across other types of psychopathology, such as PTSD, ADHD, and schizophrenia.

Findings linking negative inferential style to depression, in particular, are the most robust compared to other forms of psychopathology. Numerous cross-sectional studies have demonstrated a relationship between negative inferential style and depression in children, adolescents, and adults (Alloy et al., 2012; Alloy, Abramson, Walshaw, & Neeren, 2006). Although informative, cross-sectional methodology makes it difficult to determine whether negative inferential style reflects a risk factor, causal risk factor, correlate, or consequence of depression. Studies that utilize more than one time point are best able to examine whether negative inferential style precedes and prospectively predicts changes in depression over time. Such studies have found that negative inferential style predicts changes in depression in children, adolescents, and adults (e.g., Abela & Hankin, 2008; Abramson et al., 2002; Alloy, Abramson, Walshaw, & Neeren, 2006). More specifically, negative inferential style prospectively predicts levels of depressive symptoms, first onsets of depression, as well as recurrence of depression in adults (e.g., Alloy et al., 2000), which is consistent with the risk factor model. Additionally, studies with remitted depressives can also be helpful in determining whether negative inferential style is mood-state dependent, or whether it represents a stable and enduring trait. Unfortunately, studies that have examined remitted depressives have produced mixed results. A majority of studies have found that remitted depressives do not exhibit negative inferential style, whereas a few studies have shown that these individuals do in fact demonstrate this type of cognitive vulnerability after recovering from depression (see Alloy et al., 2006). Therefore, there is insufficient evidence to conclude whether negative inferential styles represent a consequence of depression.

It is also important to examine the role of stressful life events as a moderator of the relationship between negative inferential style and depression. It should be emphasized that in the cognitive

vulnerability-stress model, negative inferential styles contribute to psychopathology only in the presence, not the absence, of stressful life events. Studies that examine the interaction of negative inferential style and stressful life events therefore provide a more stringent test of these models. Such studies have found consistent support for the vulnerability-stress model in predicting changes in depressive symptoms as well as the occurrence of a clinically significant depressive episode in both children and adolescents (e.g., Abela & Hankin, 2008; Jacobs, Reinecke, Gollan, & Kane, 2008; Lakdawalla, Hankin, & Mermelstein, 2007) and adults (e.g. Abramson & Alloy, 2006; Abramson et al., 2002; Ingram, Miranda, & Segal, 2006; Joormann, 2010).

A subset of research on negative cognitive style and depression has separately examined the specific role that each type of inference (e.g., cause, consequence, implication for the self) plays in the development of depression. Studies that have parsed out negative inferential style into three factors have yielded mixed findings. Some studies have found support for all three types of negative inferences as a predictor of increases in depressive symptoms when combined with hassles (e.g., (Brozina & Abela, 2006). In some youth samples, researchers have found that negative inferences about the *self*, but not about the causes or consequences regarding negative events, interacted with stress to predict changes in depressive symptoms over time (Abela, McGirr, & Skitch, 2007; Cohen, Young, & Abela, 2011) However, other studies with youth have found that only inferences about the *causes* of negative events interact with stress to predict changes in depression (e.g., Conley, Haines, Hilt, & Metalsky, 2001; Lau, Rijdsdijk, Gregory, McGuffin, & Eley, 2007). Still others have demonstrated that negative inferences about the *consequences* of negative events predict changes in depression (Abela, 2001; Cohen, Young, & Abela, 2011). It has been suggested that one specific type of inference may not be the strongest predictor of depression for all individuals, especially children and early adolescents. Rather, each individual is as vulnerable to depression as his or her most negative inferential style, a theory known as

the *weakest link approach* (Abela & Sarin, 2002). The weakest link approach should inform studies that choose to parse out negative inferential style, such that these studies should determine a participant's most negative inference as reflective of that participant's overall cognitive vulnerability. Studies using this approach to study the cognitive vulnerability-stress model have found support (Abela & Hankin, 2008).

The relationship between negative inferential style and BD is similar to that of depression. Cross-sectional studies with bipolar individuals currently experiencing a depressive episode found that negative inferential styles did not differ from those of unipolar individuals who were also currently depressed (Reilly-Harrington & Alloy, 1999; Reilly-Harrington & Miklowitz, 2010). As with studies of remitted individuals with depression, studies with remitted individuals with BD have been mixed. A majority of studies report that remitted individuals do not exhibit more negative inferential styles than healthy individuals, whereas other studies do find a significant difference between these two groups (Alloy, Abramson, Walshaw, Keyser, & Gerstein, 2006; Lex, Hautzinger, & Meyer, 2011). In a similar vein, some studies have found that negative cognitive style remained stable across different mood states in bipolar individuals, including hypomania, depression, and euthymia (Reilly-Harrington & Miklowitz, 2010). Cross-sectional studies with bipolar individuals in hypomanic and manic states also found evidence for negative inferential styles (Lex et al., 2011; Reilly-Harrington & Alloy, 1999; Reilly-Harrington & Miklowitz, 2010; Scott & Pope, 2003), but these were only found in those who had a past history of depression. Those without depression did not differ from healthy controls. Longitudinal studies have found that negative cognitive styles interact with stressful life events to predict changes in depressive symptoms, as well as manic symptoms, in individuals diagnosed with BD (Reilly-Harrington & Alloy, 1999) as well as individuals with subsyndromal levels of bipolar symptoms (Reilly-Harrington & Alloy, 1999).

Research with bipolar individuals has also found a link between positive inferential style and mania. Individuals with a positive inferential style make stable and global attributions for the causes of positive events, anticipate positive consequences as a result of these positive events, and identify positive implications for the self as a result of these events (Alloy, Abramson, Walshaw, & Neeren, 2006). Cross-sectional findings show that bipolar individuals in a manic state exhibit a positive inferential style (Lex et al., 2011), which when combined with positive life events, prospectively predicts increases in hypomanic symptoms (Alloy, Abramson, Walshaw, Keyser, et al., 2006). Taken together, findings for both negative and positive inferential style in BD are consistent with the risk factor model.

Most studies with both youth and adults have shown that a negative inferential style is not associated with certain forms of anxiety symptoms or anxiety disorder either as a main effect predictor or in the context of stressful life events (e.g. Alloy, Abramson, Whitehouse, Hogan, et al., 2006; Hankin, Abramson, Miller, & Haefffel, 2004; Hankin, 2008; Joiner, 2000). However, a recent study by Alloy et al., (2012) found that negative inferential styles were concurrently associated with anxiety symptoms and diagnoses (e.g. social phobia). This discrepancy may be due to the fact that the sample in Alloy et al., (2012) was largely composed of early adolescents, which is a critical developmental period for first onset of anxiety disorders. Depression, on the other hand, typically emerges in mid- to late-adolescence or early adulthood. In addition, many studies linking negative inferential style and depression found that negative inferential style was higher among individuals diagnosed with *comorbid* anxiety and depression compared to those diagnosed with only depression or only anxiety (Alloy, Abramson, Whitehouse, Hogan, et al., 2006; Fresco, Alloy, & Reilly–Harrington, 2006). Prospective longitudinal studies have demonstrated that individuals with high levels of negative attributional style were more likely to experience an onset of an anxiety disorder that was comorbid with depression compared to low-risk

individuals (Alloy, Abramson, Walshaw, & Neeren, 2006).

Despite the lack of support for negative inferential style as a cognitive vulnerability for anxiety broadly defined, a small line of research has found associations between attributional style and PTSD, specifically (Elwood, Hahn, Olatunji, & Williams, 2009). Individuals who attribute interpersonal and non-interpersonal traumatic events to stable and global causes experience more severe PTSD as well as prospective increases in PTSD symptoms over time (Elwood, Hahn, et al., 2009; Palker-Corell & Marcus, 2004; Runyon & Kenny, 2002; Williams, Evans, Needham, & Wilson, 2002). In addition, some other studies have reported that individuals who attribute these events to external causes also report higher levels of PTSD (Elwood, Hahn, et al., 2009). In sum, there is little support for the role of negative inferential styles in anxiety generally, however, a line of research examining PTSD in particular has found support for an external, stable, and global style among individuals with PTSD.

There is a small body of research linking negative inferential style and ADHD (see (see Rucklidge, Brown, Crawford, & Kaplan, 2007) for a review). It is hypothesized that individuals with ADHD most likely experienced a higher rate of repeated experiences of failure, and if undiagnosed, teachers and parents might have attributed these difficulties to characteristics of the individual (e.g., lazy, stupid, unmotivated). As in depression, individuals with ADHD tend to make stable and global attributions for negative events as well as unstable and specific attributions for positive events (Collett & Gimpel, 2004; Rucklidge & Kaplan, 2000). In addition, another type of negative inferential style that is most commonly associated with ADHD involves a hypothesized fourth dimension, known as controllability (see Rucklidge & Kaplan, 2000 for a discussion). This dimension reflects the degree to which the causes and consequences of negative events are viewed by individuals as within their control. Researchers have found that individuals with ADHD possess an internal-uncontrollable inferential style, such that they believe that their cognitive and behavior problems are the result of variables that are

internal, but also out of their control (Rucklidge et al., 2007). In line with this theory, studies have found that adults with ADHD tended to make internal and uncontrollable attributions for negative events in their childhood (Rucklidge et al., 2007) as well as for current negative events (Rucklidge & Kaplan, 2000). This pattern is also found in children and adolescents with ADHD (Rucklidge et al., 2007; Rucklidge & Kaplan, 2000). Very few studies have examined associations between negative attributional style and conduct disorder/oppositional defiant disorder (CD/ODD). These studies have found that the interaction of attributional style and stressful life events has exhibited specificity with depression, and did not predict externalizing symptoms over time (Hankin, 2008b; N. S. Robinson, Garber, & Hilsman, 1995).

Studies of negative inferential style in schizophrenia have mostly examined the internal/external domain for attributions regarding the causes of negative events. Healthy individuals typically attribute negative events to external causes, but these causes are more likely to be universal (i.e., due to chance or circumstance), as opposed to personal (i.e., due to other individuals) (Aakre, Seghers, St-Hilaire, & Docherty, 2009). Individuals with schizophrenia, especially those currently experiencing persecutory delusions, tend to attribute negative events to external personal causes (Aakre et al., 2009). Individuals with schizophrenia who are not currently paranoid, however, do not attribute negative events to external-personal causes (Aakre et al., 2009). Therefore, the attributional style of individuals with schizophrenia appears to be a state dependent correlate of schizophrenia, and not a stable and enduring cognitive pattern.

Dysfunctional Attitudes. Beck's cognitive theory of psychopathology posits that self-schemas are associated with the onset, maintenance, and recurrence of emotional disorders, such as depression (Beck, 1976). Broadly speaking, schemas are well-organized cognitive representations of the past experiences that represent an individual's knowledge of the world, relationships with others, and the

self. Depressive self-schemas, in particular, represent a set of rigid core beliefs or dysfunctional attitudes related to performance, perfectionism, and self-worth, and guide the selective processing of negative information in the environment over and above positive or neutral information (D. J. Dozois & Beck, 2008). In the presence of stressful life events, these depressive self-schemata act as filters for stimuli in the environment, affecting the way an individual perceives, encodes, interprets, and remembers emotionally salient information (Beck, 1976; D. J. Dozois & Beck, 2008; Joormann & Gotlib, 2010). This selective processing of negative information contributes to negative automatic thoughts, and consequently, negative perception about oneself, the world, and the future—the negative cognitive triad (D. J. Dozois & Beck, 2008). This negatively biased information processing ultimately leads to the onset of emotional distress symptoms or disorders. Although there is conceptual and empirical overlap between dysfunctional attitudes and negative inferential style, dysfunctional attitudes represent universal beliefs and rules, as opposed to event-specific attributions inherent in negative inferential style. Indeed, studies have shown that measures of dysfunctional attitudes and negative inferential style load onto distinct latent factors (e.g., Haeffel et al., 2003; see Taxonomy and Structure of Cognitive Risks section in Discussion).

In addition to an overall cognitive vulnerability-stress model for dysfunctional attitudes, Beck (1987) has hypothesized that the experience of stressful events that are congruent with one's own particular cognitive schema should confer particular vulnerability to the development of depression (Zuroff, Mongrain, & Santor, 2004). These particular schemas are divided into two categories: sociotropy and autonomy. Individuals with a sociotropic cognitive style value intimacy, relationships, and acceptance from others, and consequently, are most vulnerable to emotional distress when they experience rejection, loss, and disappointment in the interpersonal domain (Beck, 1987). Those with an autonomic cognitive style place great importance on achievement, independence, and control, and so are

at a greater risk for distress when they experience failure or are precluded from making their own choices (Beck, 1987).

Young, Klosko, and Weishaar (2003) have extended the work of Beck and identified a variety of early maladaptive schemas (EMS) that underlie several forms of psychopathology. These maladaptive schemas are pervasive patterns of memories, emotions, cognitions, and bodily sensations that represent an individual's knowledge about the world, relationships with others, and the self. These schemas guide the appraisal of situations, such that the individual focuses on elements that confirm the schema and minimize or deny information that contradicts the schema, thus perpetuating the schema. Young's early maladaptive schemas fall into five domains: *disconnection/rejection*, *impaired autonomy*, *other-directedness*, *impaired limits*, *over-vigilance/inhibition*. Individuals who are high on the disconnection/rejection domain believe that others will not meet their needs for security, acceptance, and respect. Such individuals fear being rejected by others and anticipate that certain negative events will lead to negative evaluation and loss of status. Those who are high on the impaired autonomy domain anticipate that aspects of the self and the environment will interfere with their ability to function independently and successfully. Other-directedness is related to an extreme focus on the desires of others, even at the expense of the individual's needs. Impaired limits encompasses characteristics such as difficulties exerting self-control. Finally, over-vigilance and inhibition includes particular schemas, such as negativity/pessimism, emotional inhibition, and punitiveness.

Overall, dysfunctional attitudes and early maladaptive schemas have shown a robust relationship with depression, anxiety, BD, and schizophrenia, with fewer studies examining the relationship with externalizing disorders. As with negative inferential style, both dysfunctional attitudes and early maladaptive schemas are linked to psychopathology as main effect predictors, as well as in the context of negative life events. Dysfunctional attitudes demonstrate the strongest associations with depression in

children, adolescents, and adults both concurrently and prospectively over time (Abela & Hankin, 2008; Abramson et al., 2002; Alloy, Abramson, Walshaw, & Neeren, 2006; Jacobs et al., 2008). In addition, the interaction of dysfunctional attitudes and negative life events is linked to increases in depressive symptoms in adults (Abramson et al., 2002; Joormann, 2010a) as well as adolescents (Abela et al., 2011; Hankin et al., 2009). Similar findings have been demonstrated for clinically significant depressive episodes with adolescents (Lewinsohn, Joiner, & Rohde, 2001) and adults (Hankin et al., 2004; Otto et al., 2007). This constitutes solid evidence for dysfunctional attitudes as a risk factor for the development of depression.

Studies of early maladaptive schemas in non-clinical adult samples have found that specific schemas falling under the domains of disconnection and rejection, impaired autonomy and other-directedness interacted with stressful life events to predict increases in depressive symptoms over time (Cámara & Calvete, 2011; Eberhart, Auerbach, Bigda-Peyton, & Abela, 2011). Cross-sectional studies (Lumley & Harkness, 2007; Muris, 2006; Vlierberghe, Braet, Bosmans, Rosseel, & Bögels, 2009) and one longitudinal study (Calvete, Orue, & Hankin, 2012) of adolescents have found that these three domains are associated with depression in adolescents.

Dysfunctional attitudes have been shown to relate specifically to depression, and not anxiety generally, both concurrently and prospectively (e.g. Alloy, Abramson, Walshaw, & Neeren, 2006; Hankin, Abramson, Miller, & Haeffel, 2004). However, studies of early maladaptive schemas, in particular, have found that the domain of impaired autonomy, which includes vulnerability to harm and dependence, predicted increase in anxiety symptoms in the context of stressful life events (Cámara & Calvete, 2011; Glaser, Campbell, Calhoun, Bates, & Petrocelli, 2002; Lumley & Harkness, 2007; Schmidt, Joiner, Young, & Telch, 1995; Welburn, Coristine, Dagg, Pontefract, & Jordan, 2002). In terms of SAD, specifically, one study has identified the other-directedness schema domain as a

significant predictor of social anxiety symptoms over time (Calvete, Orue, & Hankin, 2012). Studies in the PTSD literature have identified mistrust/abuse, defectiveness/shame, emotional deprivation, dependency, and vulnerability to harm as particularly related to early traumatic and victimization experiences (Harding, Burns, & Jackson, 2011; Price & Mohlman, 2007b).

Few studies have explored the relationship between early maladaptive schemas and BD. Nilsson, Jørgensen, Straarup, and Licht (2010) found that individuals diagnosed with BD were higher on the domain of impaired limits, especially insufficient self-control, compared to healthy controls. Hawke, Provencher, and Arntz (2011) also found that impaired limits, specifically entitlement/grandiosity and insufficient self-control/self-discipline, was associated with BD. The authors also identified a negative relationship between emotional inhibition and BD.

Although early maladaptive schemas have not been studied in individuals with externalizing disorders, a smaller number of studies have examined the link between early maladaptive schemas and aggression. Calvete (2008) found that entitlement/grandiosity was the strongest predictor of aggressive behavior. Similarly, Rijkeboer & De Boo (2010) identified defectiveness/shame, mistrust/abuse, entitlement/grandiosity, and insufficient self-control schemas in relation to aggressive mood in children.

Finally, a small body of research in dysfunctional attitudes in schizophrenia has found that these individuals demonstrate a great deal of concern with their performance and other's evaluation of their performance (Rector, 2004). Patients with schizophrenia possess maladaptive beliefs about their ability to succeed in various activities, and as a result, have low levels of interest and motivation in engaging in these activities (i.e. negative symptoms) (Rector, 2004). In addition, individuals with schizophrenia also are concerned about how they are perceived by others (Grant & Beck, 2009; Horan et al., 2010; Rector, 2004).

Hostile Attribution Bias. Hostile attribution bias is a type of attributional style that is based

upon social information processing theory (Crick & Dodge, 1994). Broadly speaking, social information processing involves five stages: encoding of social cues, interpretation of cues, response access, response evaluation, and response enactment (Mathieson, Murray-Close, Crick, & Woods, 2011). Hostile attribution bias is grounded in the second stage of processing: interpretation of cues. In this stage, an individual assigns meaning to social cues that have been perceived, attended to, and stored in short-term memory during the encoding stage. The interpretation of social cues is contingent upon two factors: the nature of encoded social cues (e.g. quantity, sensory modality, emotional valence), as well as the ability of the individual to utilize those social cues in order to infer others' intentions and beliefs. In hostile attribution bias, individuals interpret the intentions of others as hostile in ambiguous social situations (Andrade et al., 2012; Crick & Dodge, 1994; Dodge, 1980; Mathieson & Murray-Close, 2011; Orobio de Castro, Veerman, Koops, Bosch, & Monshouwer, 2002). Therefore, a hostile attribution bias is the result of a maladaptive pattern of inferring others' intentions and beliefs.

A preponderance of evidence has demonstrated an association between hostile attribution bias and subsequent aggressive behavior. In addition, there is evidence that individuals with schizophrenia, especially those with paranoia or persecutory delusions, may exhibit hostile attribution bias. Finally, very few studies have found evidence of hostile attributional style among children who are high in depression and anxiety. To our knowledge, there are no studies of hostile attribution bias in BD.

The link between hostile attribution bias and aggressive behavior is robust and has been found among community populations of elementary and junior high school age youth (Andrade et al., 2012; Crick, Grotpeter, & Bigbee, 2002), clinical populations of youth (MacBrayer, Milich, & Hundley, 2003), incarcerated offenders (Dodge, Price, Bachorowski, & Newman, 1990), and adults (DeWall, Twenge, Gitter, & Baumeister, 2009; Dodge, 2006). Prospective longitudinal studies have also found that hostile attribution bias predicts changes in aggression over time (Dodge, 2006), which suggests that

this type of bias may serve as a risk factor. Some studies that examine the link between social rejection and subsequent aggression have found that hostile attribution bias mediates this link (DeWall et al., 2009; Reijntjes et al., 2011; Yeung & Leadbeater, 2007).

There is some discussion in the hostile attribution literature of specificity between the type of provocation situation, the hostile attribution bias, and the type of retaliatory aggression. Aggressive behavior is typically classified along two specific dimensions (Crick & Grotpeter, 1995). Physical aggression involves the use or threat of physical force in order to influence perceptions of safety and power. Relational aggression, on the other hand, involves damage to relationships through mechanisms such as friendship withdrawal, manipulation of social status among peers, or gossiping. Researchers have found some support for this hypothesized specificity. For instance, individuals who were relationally aggressive exhibited hostile attribution biases for ambiguous provocation scenarios that were relational in nature (Bailey & Ostrov, 2007; Crick & Grotpeter, 1995; Crick et al., 2002; Yeung & Leadbeater, 2007). In addition, physically aggressive individuals demonstrated hostile attribution biases for ambiguous provocation scenarios that were instrumental or physical in nature (Bailey & Ostrov, 2007; Crain, Finch, & Foster, 2005; Crick et al., 2002). However, other researchers have not found these exact relationships. For instance, Godleski & Ostrov (2010) found that hostile attributions for instrumental provocations predicted both physical and relational aggression.

In light of evidence that children with ADHD are also particularly prone to act aggressively in social situations, a small body of literature has also explored hostile attribution bias in individuals with this disorder. Attentional difficulties in ADHD cause individuals to encode fewer social cues in experimental vignettes, and as a consequence these children do not have the same amount of relevant social cue information with which to make response decisions (Andrade et al., 2012). In negatively valenced vignettes, children with ADHD make significantly more negative intent attributions than

controls (Andrade et al., 2012).

In addition to disorders characterized by externalizing behaviors, some research has explored hostile attribution bias in schizophrenia. Findings show that individuals with paranoia or persecutory delusions make hostile attributions by consistently blaming other people for negative events (e.g. personalizing bias, Penn, Sanna, & Roberts, 2008). Finally, a few studies have examined the role of hostile attributional style in depression and anxiety. Quiggle, Garber, Panak, and Dodge (1992) found that children with high levels of depressive symptoms showed a hostile attributional bias. Bell-Dolan (1995) found that anxious children identified hostile intent within non-hostile vignettes, whereas the control children did not. Because these were cross-sectional studies, no conclusions can yet be made as to whether hostile attribution bias may also serve as a risk factor for these disorders.

Looming maladaptive style. The looming maladaptive style (Riskind, Williams, Gessner, Chrosniak, & Cortina, 2000) is based on a danger or threat schema and is traditionally associated with anxiety, and not depression. These danger or threat schemas are hypothesized to lead to the formulation of biased expectations about the temporal and spatial progression of potential threats, such that these threats are perceived as rapidly mounting, escalating, or approaching the individual (Riskind et al., 2000). As a result of this active and dynamic perception of threat, individuals interpret even mundane situations as being potentially dangerous, and therefore initiate cognitive processes to assess these situations.

Findings have shown that looming maladaptive style is concurrently associated with both clinical and subclinical anxiety disorders (Riskind et al., 2000). In addition, looming maladaptive style is a prospective predictor of changes in anxiety symptoms overall (Williams, Shahar, Riskind, & Joiner, 2005) as well as changes in specific types of anxiety symptoms, such as OCD (Elwood, Riskind, & Olatunji, 2009; Riskind, Tzur, Williams, Mann, & Shahar, 2007), SAD (Brown & Stopa, 2008),

generalized anxiety (Williams et al., 2005) and PTSD (Elwood, Williams, Olatunji, & Lohr, 2007).

Because looming maladaptive style is conceptualized to relate specifically to anxiety, no study has examined their role in other internalizing disorders (e.g. depression and BD), externalizing disorders (e.g. ADHD, aggression), or schizophrenia. However, the fact that looming maladaptive style is a way of identifying, interpreting, and categorizing information that is grounded in schemas suggests some conceptual relation to previously discussed schema theories (Beck, 1967; Jeffrey E. Young et al., 2003)

Repetitive Negative Thought

This section describes various forms of repetitive negative thought. The response style theory (Nolen-Hoeksema, 1991) defines repetitive negative thought, or rumination, as a type of self-focused attention that involves repetitively and passively focusing on negative events and symptoms of distress, as well as the causes and consequences of these events and symptoms (Susan Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). It is important to note that the defining feature of rumination is the *process* of perseverative thinking, and not necessarily the *content* of the thinking. More recently, theorists have determined two distinct components within the greater construct of rumination (Treyner, Gonzalez, & Nolen-Hoeksema, 2003). The first component, called *brooding*, involves negative and self-critical thinking about the self (Watkins, 2008). Brooding is considered to be a maladaptive component of rumination and is positively associated with negative mood (Rood, Roelofs, Bögels, Nolen-Hoeksema, & Schouten, 2009). *Reflective pondering*, on the other hand, represents contemplation of symptoms of distress and negative events in order to better engage in problem solving behaviors (Watkins, 2008). This is the adaptive component of rumination and is negatively associated with negative mood (Roelofs et al., 2009).

Rumination is associated with various form of psychopathology, especially depression, anxiety, and aggression. There is a lack of studies that have explored rumination among individuals with ADHD

and schizophrenia, however. It is important to note that the process of rumination is nearly identical across these disorders, however the *content* of ruminative thought demonstrates content-specificity (e.g. sadness in depression, anger in aggression). Similar to cognitive styles, repetitive negative thought activates regions of the brain associated with self-referential processing, such as the medial prefrontal cortex, anterior cingulate cortex, and amygdala. For instance, the medial prefrontal cortex showed greater activity for depressed participants compared to control participants during a rumination task (Cooney et al., 2010).

Depressive rumination. There is a substantial body of literature linking rumination to depression. Cross-sectionally, rumination is elevated in both currently and formerly depressed inpatients, which suggests that rumination is a stable trait factor (Watkins, 2008). Rumination is also concurrently associated with elevations in depressive symptoms among children, adolescents, college students, and adults (Abela & Hankin, 2008; Nolen-Hoeksema et al., 2008; Watkins, 2008). Furthermore, children, adolescents, and adults who engage in rumination when distressed demonstrate more severe and prolonged periods of depression and are more likely to develop depressive disorders (Abela & Hankin, 2011; Nolen-Hoeksema et al., 2008). For example, a recent meta-analysis (Aldao, Nolen-Hoeksema, & Schweitzer, 2010) found large effect sizes for the relationship between rumination and depression. Longitudinally, rumination predicts increases in depressive symptoms (Hankin, 2008b; Rood, Roelofs, Bögels, & Alloy, 2010; Watkins, 2008) as well as the onset of a clinically significant depressive episode among never-depressed individuals (Abela & Hankin, 2011; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007; Watkins, 2008) consistent with the risk factor model. In parsing out the specific effects of brooding and reflective pondering, researchers have found that brooding alone significantly predicts increases in depressive symptoms over time (Burwell & Shirk, 2007; Rood et al., 2010 for youth; Treynor et al., 2003 for adults). Reflective pondering, on the other hand, did not significantly

predict changes in depression (Rood et al., 2010).

Findings in BD indicate that rumination is comparable to that seen in unipolar depression (Johnson, McKenzie, & McMurrich, 2008), or at a level that is intermediate between those with unipolar depression and healthy controls (Johnson et al., 2008; Thomas, Knowles, Tai, & Bentall, 2007). Rumination in BD is associated with greater depression, but not mania levels (Alloy et al., 2005). In addition, studies have found higher rumination levels among individuals at risk for BD (Johnson et al., 2008). Studies of individuals with remitted BD have also found higher levels of rumination compared to healthy controls (Thomas et al., 2007), which provides additional support for the idea that rumination is a stable and enduring trait-like characteristic. Unfortunately, there is a relative dearth of longitudinal research on rumination in BD (Johnson et al., 2008), so it is unclear whether rumination represents a risk factor for BD.

Anxious rumination and worry. Anxious rumination has found support as a form of rumination that is distinct from depressive rumination (Rector, Antony, Laposa, Kocovski, & Swinson, 2008). A recent meta-analysis demonstrated medium to large effect sizes for the relationship between anxious rumination and anxiety among clinical and community samples of adults, as well as children, with different strengths applying to different anxiety disorders (Aldao et al., 2010). For anxiety disorders that feature repetitive thoughts related to personal concerns, such as SAD, GAD, OCD, and PTSD, these effect sizes were particularly large. In particular, GAD and OCD, which are characterized by not only persistent and repetitive thought, but also lack of cognitive control, demonstrated rumination scores that were comparable to those of mood disorders (Aldao et al., 2010). Conversely, panic disorder exhibited the smallest association with rumination. This is not surprising, given that some of the more defining features of panic disorder include physical arousal, rather than repetitive thoughts.

A line of research has explored a specific type of repetitive thought, known as post-event

processing, in social anxiety. Individuals with social anxiety maintain a negative mental representation of themselves in social situations based upon how they believe they appear to others (Rapee & Heimberg, 1997). Following a social interaction, socially anxious individuals immediately conduct a “post-mortem” of the event, recalling not only the mental representation of the self in that particular social situation, but also memories of the self in other social situations. Because socially anxious individuals perceive the social situation as more negative than it actually was, they believe that the interaction reflected another social failure, thus preserving their beliefs of their own social inadequacy and thus perpetuating their social phobia (Clark & Wells, 1995). Both clinical and non-clinical socially anxious individuals reported higher levels of post-event processing compared to non-socially anxious individuals (Brozovich & Heimberg, 2008). Findings indicate that socially anxious individuals engage in more post-event processing following a social interaction or speech task in the lab (Abbott & Rapee, 2004; Mellings & Alden, 2000; Perini, Abbott, & Rapee, 2006; Rachman, Grüter-Andrew, & Shafran, 2000). Finally, non-anxious college students who were instructed to ruminate following a similar lab-based socially evaluative task maintained their levels of state anxiety (Wong & Moulds, 2009).

Another line of research has examined the role of rumination in PTSD, as trauma-related rumination features prominently in this type of anxiety disorder (Elwood, Hahn, et al., 2009). More specifically, trauma-related rumination may involve thinking about the causes and consequences of the trauma, which prevents focusing on the events of the trauma itself (Elwood, Hahn, et al., 2009). Cross-sectional studies have found associations between rumination and PTSD for several types of trauma, including physical and sexual assault, motor vehicle accidents, and disasters (Elwood, Hahn, et al., 2009). Prospective longitudinal analysis has shown that rumination shortly following a traumatic event predicts changes in PTSD symptoms over time (Elwood, Hahn, et al., 2009). It is important to note that the relation between rumination and PTSD is also present in children and adolescents (Ehlers, Mayou, &

Bryant, 2003).

Similar to rumination, worry is another form of self-focused perseverative thought for negative events that provoke fear and anxiety due to perceived lack of control and uncertainty (Susan Nolen-Hoeksema et al., 2008). Worry is an attempt at problem solving, such that individuals who worry try to anticipate all potential future outcomes, especially those that are negative and also highly unlikely. When these negative outcomes do not occur, fear is reduced and the worry is reinforced. Despite the substantial conceptual overlap with rumination, worry can be differentiated from rumination according to several factors. First, worry is focused on future threats, or on past events that have implications for future threats (Susan Nolen-Hoeksema et al., 2008). Rumination, on the other hand, is related solely to past events. In addition, individuals who worry anticipate future outcomes in order to avoid present negative affect and cognitions. In contrast, rumination is actively focused on current negative affect and cognitions (Susan Nolen-Hoeksema et al., 2008).

Anger rumination. Anger rumination, which involves perseverative thought on angry affect and planning future aggressive acts, is associated with anger (Pedersen et al., 2011), as well as verbal (Anestis, Anestis, Selby, & Joiner, 2009), relational (Peled & Moretti, 2009) and physical aggression (Bushman, Bonacci, Pedersen, Vasquez, & Miller, 2005; Bushman, 2002). Findings have indicated that both state and trait rumination are linked to aggression (Borders & Giancola, 2011). Whereas anger rumination is positively associated with anger and aggression, sadness rumination has been found to be a negative predictor of aggression (Peled & Moretti, 2009). It is hypothesized that sadness rumination involves a passive form of repetitive thought that is focused on the self, which leads to withdrawal behaviors. In contrast, anger rumination is a more action-oriented type of thought that is focused on others, which facilitates aggressive behavior and retaliation towards the offender (Peled & Moretti, 2009).

Cognitive Emotion Regulation Strategies

Cognitive emotion regulation strategies are cognitive responses to stressors that are used to mitigate emotional distress or modify the type of emotional experience (Aldao et al., 2010).

Additionally, cognitive emotion regulation strategies can also be used to target the stressor itself. Unlike the maladaptive cognitive products reviewed to this point, distraction and reappraisal are two *adaptive* cognitive strategies that are shown to decrease negative emotional experience.

The medial prefrontal cortex has also been implicated in reappraisal and distraction, as this brain region is involved in appraising emotional stimuli in relation to the self and the environmental context (McRae et al., 2010). Lateral regions of the prefrontal cortex, which are closely related to cognitive control, are also recruited during these regulation strategies. Researchers have posited that activation of these regions may reflect the use of verbal working memory to generate and maintain the necessary cognitions to mitigate emotional distress (McRae et al., 2010). This hypothesis provides one potential connection between cognitive processing (e.g. working memory, EF) and products (e.g. reappraisal).

Distraction. Distraction is a form of emotion regulation that involves engaging in thoughts or behaviors to divert one's attention away from negative mood and instead towards more positive stimuli (Nolen-Hoeksema et al., 2008). Distraction can involve a change in internal focus, such as invoking pleasant thoughts or memories, or it can involve a change in external focus, such as engaging in a favorite hobby (Gross & Thompson, 2007). Distraction can be further divided according to two domains of strategies: active and passive. Active strategies require effort to attend to and engage in positive or neutral information that is unrelated to the negative stimulus (Webb, Miles, & Sheeran, 2012). For example, in a laboratory experiment, participants might be given explicit instructions to distract themselves by thinking about something that is unrelated to the negative emotional stimulus. Passive strategies, on the other hand, do not involve explicit instructions to distract, but still involve engagement

in emotionally neutral or positive materials or tasks that are unrelated to the negative emotional stimulus (Webb, Miles, & Sheeran, 2012). In utilizing distraction as an emotion regulation strategy to replace negative mood with a more neutral or positive mood, an individual avoids the potential for the negative mood to bias self-reflection or cognitive problem solving strategies (Nolen-Hoeksema, 1991)

Overall, findings indicate that distraction may serve as an important strategy for mitigating negative mood, however, studies examining the relationship between distraction and specific disorders are lacking. Meta-analytic findings suggest that distraction contributes to relatively large shifts in affect ($d=0.46$; Augustine & Hemenover, 2009). A more recent review by Webb and colleagues (2012) examined studies with non-clinical samples that employed experimental manipulation of emotion regulations strategies, such as distraction, and found variable effect sizes depending on the type of instructions that were provided. For instance, active distraction strategies demonstrated larger effect sizes ($d = 0.47$ when participants were asked to focus on positive material; $d = 0.38$ when asked to focus on neutral material) than passive distraction strategies ($d = 0.18$ when focusing on positive material; $d = 0.23$ when focusing on neutral material). The fact that these studies utilized experimental manipulation provides evidence that distraction may act as a causal protective factor that buffers against negative emotional experience.

A small body of research has explored the relationships between distraction and specific emotional disorders. A number of cross-sectional studies with adults have found that distraction confers benefits for dysphoric individuals (e.g., Chang, 2004; Lam, Smith, Checkley, Rijdsdijk, & Sham, 2003; Lyubomirsky & Nolen-Hoeksema, 1993), however other studies have not found this relationship (e.g., Just & Alloy, 1997; Nolen-Hoeksema & Morrow, 1993). Longitudinal studies with youth (e.g., Abela, Aydin, & Auerbach, 2006; Ziegert & Kistner, 2002) and adults (e.g. Huffziger & Kuehner, 2009) have found some support for the finding that distraction predicts decreases in depressive symptoms over time,

but others have not been able to support these findings (e.g., Abela, Brozina, & Haigh, 2002; Abela, Vanderbilt, & Rochon, 2004).

Some studies have also examined remitted depressed patients (e.g., Huffziger & Kuehner, 2009; Joormann, Siemer, & Gotlib, 2007; Singer & Dobson, 2007). Overall, these studies have found that experimentally induced distraction strategies were as effective as other strategies, such as mindfulness, at improving mood. In addition, Huffziger and Kuehner (2009) found that a greater tendency to engage in distraction was associated with less negative mood and more positive mood across all experimental groups. Moreover, Huffziger, Reinhard, and Kuehner (2009) also found that distraction in formerly depressed inpatients predicted fewer depressive symptoms over time.

There is a lack of research on the association between distraction and other types of disorders. One study by Ehring, Fuchs, and Kläsener (2009) conducted an experimental manipulation of distraction among individuals with PTSD and found that distraction led to decreases in negative mood and also a decrease in the frequency and distress of intrusive memories. A few studies have examined the relationship between distraction and anger. (Ray, Wilhelm, and Gross (2008) and Denson, Moulds, and Grisham (2012)) examined a nonclinical undergraduate sample and found that distraction significantly reduced anger. Moreover, Bushman et al. (2005) and Rusting and Nolen-Hoeksema (1998) found that distraction was not only associated with reports of decreased anger, but also better problem solving abilities. These experimental manipulation strategies lend further support to the causal role of distraction in reducing the impact of negative affect in various forms of psychopathology.

Cognitive reappraisal. Cognitive reappraisal refers to the ability to change how one thinks about a situation or about one's capacity to manage the demands of a situation in order to alter its emotional significance (Gross & Thompson, 2007). Reappraisal can act upon different aspects of the situation (Webb et al., 2012). One could reinterpret the cause or context of the situation that is evoking

the emotion. For instance, Hajcak and Nieuwenhuis (2006) instructed participants to “Come up with a less negative interpretation of the picture content (e.g. a bloody crime scene could be seen as the place where a murder investigation was finally solved)”. One could adopt a more objective or third person perspective.

As with distraction, reappraisal may also serve as an important strategy for mitigating negative mood, however, studies examining the relationship between reappraisal and specific disorders is still in its infancy. Meta-analytic findings for experimental manipulation of emotion regulation strategies, such as reappraisal, have found a small-to-medium sized effect of reappraisal on emotional responding ($d = 0.36$) (Webb et al., 2012). These researchers further examined this effect by considering the target of the reappraisal strategy. Reappraising the cause or context of the situation had a small-to-medium effect on emotional response ($d = 0.45$), distancing oneself or taking a third person perspective had a small-to-medium size effect ($d = 0.36$), and reappraising the resulting emotional response had a small effect ($d = 0.23$).

Although a substantial body of literature has examined the relationship between reappraisal and various types of affect (e.g., general negative affect, sadness, anger) fewer studies have considered how reappraisal may relate to the more extreme and severe forms of affect that are implicated in psychopathology. A meta-analysis of reappraisal found that self-reported reappraisal was negatively associated with psychopathological symptoms overall ($d = -0.28$; Aldao et al., 2010). In the case of depression, specifically, trait reappraisal is associated with lower symptom levels (Gross & John, 2003) and fewer depressive episodes (Garnefski, Kraaij, & Spinhoven, 2001; Garnefski & Kraaij, 2006; Kraaij, Pruymboom, & Garnefski, 2002).

Reappraisal manipulations in a laboratory setting have also been demonstrated as an effective strategy for reducing anxiety in college undergraduates (Hofmann, Heering, Sawyer, & Asnaani, 2009)

and for decreasing negative emotion in children and adolescents diagnosed with GAD, separation anxiety, and SAD (Carthy, Horesh, Apter, Edge, & Gross, 2010). Reappraisal was also associated with lesser total PTSD severity among military service veterans diagnosed with PTSD (Boden, Bonn-Miller, Kashdan, Alvarez, & Gross, 2012). Conversely, less frequent use of cognitive reappraisal was associated with higher levels of PTSD symptom severity (Eftekhari, Zoellner, & Vigil, 2009; Ehring & Quack, 2010).

A moderate reduction in anger was shown for treatment programs that utilized cognitive restructuring, a correlate of reappraisal (DiGiuseppe & Tafrate, 2003). Ray et al., (2008) and Denson et al., (2012) examined a nonclinical undergraduate sample and found that reappraisal of an anger-eliciting event significantly reduced anger. Among outpatient participants with schizophrenia and schizoaffective disorder, those who were instructed to reappraise reported experiencing less sadness and general negative affect (Perry & Henry, 2012). However, there may be no trait differences in tendency to reappraise, as individuals with schizophrenia did not differ from control participants in their use of reappraisal strategies (Badcock, 2010; Henry, Rendell, Green, McDonald, & O'Donnell, 2008). As with distraction, experimental manipulation of reappraisal, as well as treatment interventions that employ reappraisal, this type of cognitive emotion regulation strategy may be causally related to emotional distress and psychopathology.

Table 6

Commonly used cognitive product measures

Construct	Measure	Description	Methods Citation
Attributional Style: Attributions for the <i>cause</i> of life events along three dimensions: 1) Internal vs. external 2) Stable vs. unstable 3) Global vs. specific	Attributional Style Questionnaire (ASQ)	Presents 12 hypothetical events (i.e. 6 are positive, 6 are negative; in addition, 6 are interpersonal, 6 are achievement-related). Participants indicate a cause of the event and answer questions about the cause of the event on a 7-point Likert scale.	(Peterson & Semmel, 1982)
	Children's Attributional Style Questionnaire – Revised (CASQ-R)	Presents 24 hypothetical negative events. Participants choose between two possible explanations for this event. Each item holds two dimensions of attributional style constant while the third is varied between the explanations.	(Thompson, Kaslow, Weiss, & Nolen-Hoeksema, 1998)
Negative Inferential Style: Attributions for the <i>cause</i> , <i>consequences</i> , and <i>self-worth implications</i> of life events along three dimensions.	Cognitive Style Questionnaire (CSQ)	Presents 24 hypothetical events (i.e. 12 are positive, 12 are negative; in addition, 12 are interpersonal, 12 are achievement-related). Participants indicate a cause of the event and answer questions about the cause, consequences, and self-worth implications of the event on a 7-point Likert scale.	(Haefffel et al., 2008)
	Adolescent Cognitive Style Questionnaire (ACSQ)	Presents 12 hypothetical negative events (i.e. 6 are interpersonal, 6 are achievement-related). Participants indicate a cause of the event and answer questions about the cause, consequences, and self-worth implications of the event on a 7-point Likert scale.	(Hankin & Abramson, 2002)
Dysfunctional Attitudes: Core beliefs about the world, relationships with others, and self that are related to performance, perfectionism, and approval by others.	Dysfunctional Attitudes Scale (DAS)	Presents 40 dysfunctional attitude statements. Participants indicate agreement on a 7-point Likert scale (e.g. "Totally Agree" to "Totally Disagree")	(A. Weissman & Beck, 1978)
	Children's Dysfunctional Attitudes Scale (CDAS)	Presents 40 dysfunctional attitude statements. Participants indicate agreement on a 4-point Likert scale (e.g. "never True" to "Always True").	(Abela & Sullivan, 2003)

<p>Early Maladaptive Schemas: Core beliefs about the world, relationships with others, and self that are related to childhood adverse relational experiences:</p> <p>1) Disconnection: expectation that need for security, acceptance, respect will not be met.</p> <p>2) Impaired Autonomy: concerns about ability to function competently, capably, or independently.</p> <p>3) Other Directedness: excessive focus on desires, feelings, & response of others.</p> <p>4) Over-vigilance & Inhibition: emphasis on suppressing feelings, impulses, & choices.</p> <p>5) Impaired Limits: deficiency in internal limits, responsibility to others, & long-term goal orientation.</p>	<p>Young Schema Questionnaire (YSQ)</p>	<p>Presents 205 schemas (75 schemas in the Short Form). Participants indicate agreement on a 6-point Likert scale (e.g. “Describes me perfectly” to “Completely untrue of me”).</p>	<p>(J. Young & Brown, 1994)</p>
<p>Looming Maladaptive Style: Mental representation of future threat as rapidly rising in risk, progressively worsening, and accelerating.</p>	<p>Looming Maladaptive Style Questionnaire</p>	<p>Presents 6 hypothetical events. Participants answer questions about the event on a 5-point Likert scale.</p>	<p>(J H Riskind et al., 2000)</p>
<p>Hostile Attribution Bias: In ambiguous provocation situations, the tendency to interpret the intentions of another as hostile.</p>	<p>Hypothetical situation instruments (multiple versions)</p>	<p>Presents hypothetical provocation situations (i.e. some are instrumental, some are relational) in which the intent is ambiguous. Participants answer questions about the reasons for the provocation and intent of the provocateur.</p>	<p>(e.g. Crick, 1995 for questionnaire-based vignettes; (Dodge, Pettit, McClaskey, & Brown, 1986) for videotaped vignettes).</p>
<p>Repetitive Negative Thought: Self-focused attention that involves repetitively and passively focusing on negative events and symptoms of distress, as well as the causes and consequences of these events and symptoms.</p>	<p>Response Style Questionnaire – Ruminative Responses Scale (RSQ - R)</p>	<p>Presents 22 statements regarding tendency to ruminate. Participants indicate agreement on a 4-point Likert scale (e.g. “Almost Never” to “Almost Always”)</p>	<p>(Susan Nolen-Hoeksema, 1990)</p>
	<p>Children’s Response Style Questionnaire (CRSQ)</p>	<p>Presents 10 statements regarding tendency to ruminate. Participants indicate agreement on a 5-point Likert scale (e.g. “Never” to “Always”)</p>	<p>(Abela, Rochon, & Vanderbilt, 2000)</p>
	<p>Penn State Worry Questionnaire (PSWQ)</p>	<p>Presents 16 statements regarding tendency to worry. Participants indicate agreement on a 5-point Likert scale.</p>	<p>(Meyer, Miller, Metzger, & Borkovec, 1990)</p>

<p>Reappraisal: Change how one thinks about a situation or about one's capacity to manage the demands of a situation in order to alter its emotional significance.</p>	<p>Emotion Regulation Questionnaire (ERQ)</p>	<p>Presents 10 items that assess trait use of reappraisal & suppression. Participants indicate agreement on a 7-point Likert scale.</p>	<p>(James J. Gross & John, 2003)</p>
<p>Distraction: Engaging in thoughts or behaviors to divert one's attention away from negative mood and instead towards more positive stimuli.</p>	<p>Response Style Questionnaire – Distractive Responses Scale (RSQ - D)</p>	<p>Presents 11 statements regarding tendency to distract. Participants indicate agreement on a 4-point Likert scale (e.g. "Almost Never" to "Almost Always").</p>	<p>(Susan Nolen-Hoeksema, 1990)</p>
	<p>Children's Response Style Scale (CRSQ)</p>	<p>Presents 10 statements regarding tendency to distract. Participants indicate agreement on a 5-point Likert scale (e.g. "Never" to "Always")</p>	<p>(Abela, Rochon, & Vanderbilt, 2000)</p>

Gender, cultural, and ethnic differences and considerations

A preponderance of studies examining demographic differences in cognitive products have focused largely on gender, as rates of internalizing psychopathology are markedly higher in females, whereas externalizing psychopathology is more prevalent in males (Zahn-Waxler, Crick, Shirtcliff, & Woods, 2006). Studies of gender differences in negative inferential styles, for instance, have found that females report more negative inferential styles than males, which might explain the fact that females are more likely to develop depression in adolescence than males (Hankin & Abramson, 2002; Mezulis, Abramson, Hyde, & Hankin, 2004). In addition, studies have found that negative attributional style was only associated with elevations in depressive symptoms at high levels of negative life events for males (e.g. Hankin, Abramson, & Siler, 2001; Stone, Gibb, & Coles, 2009). Among females, however, negative attributional style was associated with depressive symptom elevation even at low levels of negative events. Therefore, a negative inferential style may both mediate and moderate gender differences in depression. There is a relative lack of studies examining gender differences for other types of cognitive styles. A few studies have found that males exhibit more dysfunctional attitudes compared to females (Haefffel et al., 2003; Hankin, 2009). In addition, some studies have identified gender differences in hostile attribution biases, such that boys demonstrate hostile attribution bias for instrumental provocation situations and girls exhibiting this bias for relational provocation (Crick & Dodge, 1994; Crick et al., 2002).

There are also notable gender differences in repetitive negative thought, such that females report higher levels of depressive rumination compared to men (Hankin, 2008b; Susan Nolen-Hoeksema, 2012; Rood et al., 2010), whereas males may engage in more anger rumination compared to women (e.g. Rusting & Nolen-Hoeksema, 1998). As with negative inferential style, it has been suggested that the gender difference in depressive rumination, in particular, partially accounts for the gender difference in

depression rates among females and males (Hankin, Wetter, & Cheely, 2008; Hankin, 2009; Watkins, 2008). However, some studies, especially with children and early adolescents, have not found sex differences in rumination (Abela, Brozina, & Haigh, 2002; Abela, McGirr, & Skitch, 2007; Abela & Hankin, 2011; Abela, Vanderbilt, & Rochon, 2004). This underscores the point that consistent sex differences in response styles, such as rumination, may not develop until early to middle adolescence (Hankin et al., 2008; Watkins, 2008).

There is also some evidence for gender differences in cognitive emotion regulation strategies. According to a review by Nolen-Hoeksema (2012), women were more likely than men to report using reappraisal and distraction. Some researchers (e.g. Barrett, Lane, Sechrest, & Schwartz, 2000) have posited that men may engage in more automatic, subconscious emotion regulation, and as a result may not report the use of conscious, language-based strategies, such as reappraisal or distraction. In line with this finding, a neuroimaging study by McRae and colleagues (2008) suggests that men may indeed engage in reappraisal, however results indicate that they do so more automatically and with less mental effort compared to women.

Compared to research on gender differences in cognitive products, relatively few studies have explored cultural and ethnic differences in these constructs. There is evidence showing that predictive associations are maintained across different cultural and ethnic groups. For example, interactions between negative cognitive styles (e.g. inferential style and dysfunctional attitudes) and stressful life events predict depressive symptoms in youth samples of rural and urban regions of China (Abela et al., 2011) as well as youth in Spain (Calvete, Villardón, & Estévez, 2008), the same as found in the United States. Furthermore, early maladaptive schemas are risk factors for the development of depression (Calvete, Orue, & Hankin, 2012), anxiety (Cámara & Calvete, 2011), and aggression (Calvete, 2008) in Spanish youth. While predictive associations seem to be similar across cultural groups, there is also

evidence for mean level differences in cognitive influences. For example, the positivity bias (i.e., stronger positive relative to negative attributional style) is higher among individuals from Western, individualistic cultures relative to East-Asian, collectivistic cultures (Mezulis et al., 2004). Additional research that directly compares these constructs across cultures will help to elucidate potential cultural and ethnic differences.

Discussion

Summary: Empirical status of cognitive products and processes in the development of psychopathology

There are several main points to emphasize based upon our review of the literature. First, it is clear that the preponderance of evidence shows that cognitive products and processes are associated, at least concurrently, with many prevalent psychopathologies in children, adolescents, and adults. At this point and with the current state of knowledge, the field generally does not need more cross-sectional, case control designs comparing a group with one specific disorder to healthy controls (especially adults, as much of the literature, particularly with cognitive processes, has used almost exclusive adult samples). The cross-sectional case-control design only addresses the question of whether there is a difference in cognitive risk between groups, and that question has been satisfactorily answered in the affirmative, at least for disorders, cognition, and age groups reviewed herein. There is value for cross-sectional work in new areas (e.g., preschool psychopathology). At a minimum, future research examining the cognitive products and processes reviewed here should utilize longitudinal designs to better disambiguate which of several logical conceptual models (e.g., cause, consequence, correlate; see earlier for expanded discussion) best characterizes how these cognitive influences relate to developmental trajectories of psychopathology (onset, maintenance, desistance) over the life-course.

Second, there is need for research using carefully selected age-relevant samples that are

thoughtfully chosen to investigate developmentally oriented questions. As just noted, most of the process studies have used adult samples, yet such research cannot easily address developmentally sensitive questions relevant for understanding onset, course, and trajectory of disorders because many disorders have child or adolescent onsets and then exhibit a chronic pattern, especially in adulthood. As such, exclusive use of adult samples, which likely comprises an unknown admixture of some new onsets alongside mostly recurrences (and it would be important to assess and investigate whether these are first recurrences or multiple), does not significantly advance knowledge on the role of cognitive influences in developmental trajectories of psychopathology. On the other hand, very few studies have examined cognitive influences with preschool-aged children; developmentally sensitive and age-appropriate measures of cognitive products and processes can be used to examine associations with prevalent preschool psychopathologies (Egger & Angold, 2006). While our review shows that cognitive products, in particular, and likely cognitive processes, are associated with common psychopathologies in children and adolescents to an equal magnitude as found in adults, often the ages of samples in these studies employed relatively wide age ranges (e.g., 9-18 year olds) that may not carefully match developmental questions. Future research would benefit from using tighter age ranges that are thoughtfully connected to developmentally sensitive questions.

Last, there likely exist meaningful developmental patterns in how cognitive risks operate over time to influence the development of psychopathology, yet it is not entirely clear how various developmental factors (e.g., biological, cognitive, and emotional development; changes in social contexts—peers, parents) affect this process. The extant data, as reviewed earlier, suggest that cognitive products and processes may broadly apply as risks to multiple common psychopathologies across the lifespan, including children, early adolescents, adolescents, and adults. Yet at the same time, it is likely that much change occurs with respect to these cognitive risks throughout childhood and adolescence. In

particular, it is likely that accumulating environmental experience (e.g., stressful life events) and known changes in cognitive, emotional, and biological influences across development contribute to: (1) the emergence, and developmental origins, of these cognitive influences and when they function to confer risk to development of psychopathologies; (2) greater stabilization of cognitive influences into more enduring, trait-like risks over time; and (3) enhanced inter-relatedness and consolidation among the different cognitive products and processes. In sum, there is still a need for research examining cognitive products and processes in the development of psychopathology across the lifespan. In the sections that follow, we discuss developmentally relevant areas for advancing knowledge of cognitive risks to psychopathology.

Table 8

Summary of Findings

	Executive Function	Attention	Memory	Cognitive Styles	Repetitive Negative Thought	Cognitive Emotion Regulation Strategies
Depression	<ul style="list-style-type: none"> • Broad impairment across all aspects of EF. 	<ul style="list-style-type: none"> • Mixed evidence for impaired sustained attention. * Little research on selective attention, but appears intact. • Little research on divided attention, but may be impaired. • Biased attention towards negative stimuli. 	<ul style="list-style-type: none"> • Impaired episodic memory. • Impaired autobiographical memory. • Little research on semantic memory, but appears to be intact. 	<ul style="list-style-type: none"> - Negative inferential style, dysfunctional attitudes, & early maladaptive schemas (e.g. disconnection/rejection, impaired autonomy, other-directedness) linked to symptoms and episodes of depression; mixed evidence for remitted depression - Very little evidence for link to hostile attribution bias - No evidence for looming maladaptive style 	<ul style="list-style-type: none"> - Depressive rumination, especially brooding linked to symptoms and episodes of depression 	<ul style="list-style-type: none"> - Distraction and reappraisal linked to lower levels of negative affect - Mixed findings linking distraction and reappraisal to dysphoria and depressive symptoms
Bipolar Disorder	<ul style="list-style-type: none"> • Broad impairment across all aspects of EF. 	<ul style="list-style-type: none"> • Impaired sustained attention. • Little research on selective attention. • Little research on divided attention, but may be impaired. • Little research on biased attention, but may be bias towards negative and manic stimuli. 	<ul style="list-style-type: none"> • Impaired episodic memory. • Intact semantic memory. • Little research on autobiographical memory, but may be impaired. 	<ul style="list-style-type: none"> - Negative inferential style linked to depression and mania - Positive inferential style linked to mania - Early maladaptive schema (e.g. impaired limits) linked to BD - No evidence for hostile attribution bias or looming maladaptive style 	<ul style="list-style-type: none"> - Small body of evidence linking depressive rumination to depression in BD 	
Anxiety	<ul style="list-style-type: none"> • Impairments 	<ul style="list-style-type: none"> • Little research on sustained or 	<ul style="list-style-type: none"> • Impaired episodic memory 	<ul style="list-style-type: none"> - Some evidence for 	<ul style="list-style-type: none"> - Anxious 	<ul style="list-style-type: none"> - Little evidence

	<p>across most aspects of EF for OCD and PTSD.</p> <p>• Little research on other anxiety disorders.</p>	<p>selective attention.</p> <ul style="list-style-type: none"> • Little research on divided attention, but may be impaired. • Biased attention towards negative stimuli. 	<p>in OCD and PTSD.</p> <ul style="list-style-type: none"> • Impaired autobiographical memory in PTSD. • Little research on other anxiety disorders. 	<p>link between negative inferential style and comorbid depression and anxiety, but not anxiety alone (PTSD is an exception)</p> <ul style="list-style-type: none"> - Early maladaptive schemas (e.g. impaired autonomy) linked to anxiety - Strong link to looming maladaptive style 	<p>rumination linked primarily to SAD, GAD, OCD, PTSD</p> <ul style="list-style-type: none"> - Support for post-event processing, a type of anxious rumination, in SAD - Little evidence for link to PD 	<p>linking distraction to decreases in negative mood in PTSD, only</p> <ul style="list-style-type: none"> - Reappraisal linked to decreases in anxiety
Schizophrenia	<ul style="list-style-type: none"> • Broad impairment across all aspects of EF. 	<ul style="list-style-type: none"> • Impaired sustained attention. • Visual selective attention appears largely intact (overall response slowing only). Auditory selective attention may be impaired. • Little research on divided attention, but may be impaired. • Little research on biased attention, but may be bias towards disorder-specific stimuli (e.g., paranoia words). 	<ul style="list-style-type: none"> • Impaired episodic memory. • Impaired semantic memory. • Impaired autobiographical memory. 	<ul style="list-style-type: none"> - Persecutory delusions associated with attributions of negative events to external personal causes as well as hostile attribution bias - Some evidence showing link to concern about self-efficacy and performance - No evidence for looming maladaptive style 	<ul style="list-style-type: none"> - No evidence for rumination in schizophrenia 	<ul style="list-style-type: none"> - No evidence for distraction or reappraisal in schizophrenia
ADHD/Externalizing	<ul style="list-style-type: none"> • Broad impairment across all aspects of EF. 	<ul style="list-style-type: none"> • Impaired sustained attention. • Selective attention appears largely intact (overall response slowing only). • Little research on divided attention, but may be impaired. • Little research on biased attention. 	<ul style="list-style-type: none"> • Impaired verbal episodic memory, but non-verbal episodic memory appears to be intact. • May be small impairments in semantic memory. • Little research on autobiographical memory. 	<ul style="list-style-type: none"> - Some evidence for negative inferential style in ADHD, includes a “controllability” dimension - Early maladaptive schemas (e.g. entitlement/grandiosity) linked to aggression - Hostile attribution 	<ul style="list-style-type: none"> - Anger rumination linked to aggression 	<ul style="list-style-type: none"> - Distraction and reappraisal linked to decreases in angry mood

				bias linked to aggression and ADHD - No evidence for looming maladaptive style		
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Developmental Considerations

Cognitive risks to the development of psychopathology, by definition and theory, are believed to reflect stable individual differences, certainly by adulthood. However, the age when cognitive products and processes emerge as stable risks for psychopathology, and the extent to which they represent trait-like risk processes over the lifespan, especially in childhood and adolescence, remains largely unknown. Addressing these questions is essential for both basic developmental clinical scientific knowledge and for informing translational applications. Understanding when and how cognitive products and processes emerge as stable risks to various psychopathologies can potentially inform when to implement optimal, developmentally sensitive interventions and provide etiologically valuable clues to better understand the established developmental epidemiological patterns (e.g., first onset, maintenance, recurrence, desistance) in various prevalent psychopathologies.

Moreover, and as we discuss in greater detail later, it may be that different underlying *mechanisms* lead to various psychopathologies at different developmental periods. In particular, two logical hypotheses are important to consider. First, it may be that the mechanisms fundamentally implicated in products and processes only emerge and work to contribute to development of psychopathologies shortly before these disorders typically onset. Alternatively, the various cognitive influences may exist and function as risks earlier, but remain latent until activated (e.g., by stressors, for those cognitive influences postulated to operate per a vulnerability-stress model). In other words, a critical, but relatively uninvestigated question, is *why* various cognitive risks predict developmental patterns of psychopathology (e.g., first onsets) and *when* these phenomena are most likely to occur. The ensuing discussion focuses on theoretical possibilities and some key empirical evidence regarding developmental influences, specifically emergence and origins, then stabilization, and finally the structure and eventual consolidation, of cognitive influences.

Emergence of cognitive products. Historically, since the original theorizing on cognitive theories of psychopathology, there has been considerable speculation, and some empirical research, regarding which developmental stage and age period best characterize when cognitive products emerge as potent risks for the development of psychopathology (e.g., Gibb & Coles, 2005; Hankin et al., 2009; M Rutter, 1987). For example, from the perspective of Beck's cognitive theory, it is hypothesized that schema are not consolidated until adolescence or even early adulthood because it has been postulated that repeated learning experiences need to reinforce the schemas to be actively operating and lead to symptoms (e.g., Hammen & Zupan, 1984). Also earlier theorizing for the hopelessness theory (Abramson et al., 1989) postulated that a depressogenic attributional style only emerges during the transition from childhood to adolescence when children acquire the ability to engage in abstract reasoning and formal operational thought (e.g., Susan Nolen-Hoeksema, Girgus, & Seligman, 1992).

In seeking to explain mechanisms that may underlie potential developmental changes in cognitive products based on these early conceptual speculations, researchers have drawn from a wide variety of findings in the cognitive development literature – particularly those pertaining to middle childhood. For example, during middle childhood, children start to develop a more stable and less concrete sense of self (Rholes, Blackwell, Jordan, & Walters, 1980). Their self-views become increasingly differentiated as they shift their focus from concrete, behavioral characteristics in early childhood to relatively more personality trait-like characteristics in middle childhood to more abstract psychological constructs during adolescence (e.g., Harter, 1990). During this developmental period, children also become less here-and-now oriented (Shirk, 1988) and more likely to integrate past experience into working knowledge in a manner that informs interpretations and predictions (Rholes et al., 1980). Whereas very young children have a rudimentary understanding of causality (e.g., Oakes,

1994), middle childhood is when children use stable personality traits increasingly to explain their behavior (e.g., Corrigan, 1995).

Some early empirical research examined the hypothesis that cognitive products (mostly a negative attributional style, as the most theorized and researched) emerge during the transition from middle childhood to early adolescence, based on the view that it is necessary that concrete operations be present for negative attributions to be made about explaining the cause of negative events. Results from two studies, in particular, provide the most compelling support for this early hypothesis based on necessary cognitive developmental maturity requirements in late childhood (Susan Nolen-Hoeksema et al., 1992; Turner & Cole, 1994); these studies suggest that a depressogenic attributional style interacted with negative events to predict higher levels of depressive symptoms in later childhood/early adolescence, but not earlier in childhood. However, these early studies tended to focus relatively narrowly on only one particular cognitive risk (e.g., negative attributional style) and tended to investigate age of emergence with psychometrically poor measures, which obviously affect ensuing conclusions. Since these initial investigations, more recent studies using more reliable and developmentally sensitive measures have shown that cognitive products, such as a negative attributional style, predict increases in depressive symptoms much earlier (e.g., by age 6, (Conley et al., 2001); age 8, (John R Z Abela, 2001) than previously thought and than what was originally theoretically expected based on cognitive maturity hypotheses.

Presently, the developmental period and age by which cognitive products emerge is not clearly established. Our review indicates that a negative cognitive style predicts depressive symptoms as early as age 6 (Conley et al., 2001), dysfunctional attitudes by age 6 (John R Z Abela & Skitch, 2007) in offspring of depressed mothers), and rumination by age 6 (John R Z Abela, Hankin, Sheshko, Fishman, & Stolow, 2012) in offspring of depressed mothers; age 8 in normative sample,

(Lopez, Felton, Driscoll, & Kistner, 2012). However, these findings should be interpreted tentatively because there have been insufficient studies that have focused on the earliest ages by which these cognitive products predict various psychopathological outcomes, and the earliest prediction may vary depending on the form of psychopathology examined (e.g, separation anxiety symptoms with earlier modal age of onset versus depression or PTSD). Also, developmentally sensitive and reliable measures are needed to rigorously examine emergence of these risks in earlier ages (e.g., preschoolers). Last, there is a paucity of theory to suggest when and why particular cognitive products would emerge as contributing factors conferring risk to different psychopathologies. (Note, there is some theory and evidence regarding developmental origins and precursors of these cognitive products, and such work, reviewed later, has relevance for understanding the emergence of these risks.)

Developmental theory and research on origins and emergence for hostile attributional bias has been less controversial. According to developmental literature, hostile attribution bias serves as the default attributional style in prevocational situations and is present very early in life (Dodge, 2006). Hostile attributions follow from a simple assumption that an act that brings about a negative outcome for the self must logically originate from an actor with a negative intent (Dodge, 2006). This rigid association between negative outcome and negative intent becomes more flexible as a result of cognitive development that begins at about age three or four. It is during this time that young children acquire the capacity to understand that others' mental states are different from our own, and they can make correct inferences about the content of the mental states (e.g. intentions and beliefs) of others (Couture, Penn, & Roberts, 2006).

These inferences serve an adaptive purpose by allowing individuals to predict and explain others' behavior in a social context (K.-H. Lee, Farrow, Spence, & Woodruff, 2004). In the specific

case of negative outcomes, identification and interpretation of social cues that signal that the actor may have acted without intent or with a benign intent, instead of malevolently, is a particularly important (Dodge, 2006), albeit more complicated, formal operation. Early adolescence is another key developmental period that facilitates the ability to infer lack of intent or benign intent, as this stage of cognitive development is characterized by the ability to generate alternatives and consider hypothetical possibilities (Dodge, 2006). Over time, the ability to interpret ambiguous social cues as nonintentional facilitates the development of a stable pattern of inferring benign intent, especially in ambiguous situations. Therefore, a hostile attribution bias evolves from an individual not acquiring a benign attributional style and instead continuing to match intent with outcome (Dodge, 2006).

Emergence of cognitive processes. While much is known about typical (and to some extent abnormal) development of cognitive processes, little is known about when cognitive processes emerge as risks for the development of psychopathology. Unlike cognitive products, which as discussed above are hypothesized to emerge only after particular stages of development, in typically developing children cognitive processes develop continuously throughout childhood, and in some cases into early adulthood. EF has a particularly protracted developmental course, following the protracted development of prefrontal cortex. Some early developmental milestones are reached during the preschool years (e.g., the ability to switch between two simple tasks rather than perseverating on the first task (Munakata, Chatham, & Snyder, 2012a). Throughout early and middle childhood, there is continued quantitative (e.g., ability to switch between tasks more efficiently), and qualitative (e.g., shift from reactive to proactive control strategies) development (Munakata et al., 2012a). Unlike some other cognitive abilities, which are relatively mature by the end of middle childhood, EF continues to develop through adolescence and even into early adulthood (e.g., Huizinga, Dolan, & van der Molen, 2006), and then declines in older adulthood (e.g., Buckner, 2004).

In contrast to EF, attention and memory processes develop relatively early, though they continue to develop throughout childhood. Infants already have the ability to selectively attend to particular stimuli in some circumstances (Atkinson & Braddick, 2012). Selective, divided, and sustained attention abilities continue to develop through middle childhood when they generally reach adult levels (e.g., Waszak, Li, & Hommel, 2010), although some development may continue in adolescence (e.g., McAvinue et al., 2012). As with attention, even infants have impressive memory abilities (Bauer, 2007), although the ability to retrieve specific episodic memories continues to improve through middle childhood (e.g., see Ghetti & Bunge, 2012 for review), and semantic memories continue to accrue throughout the lifespan. Like EF, age-related declines in memory are common (Buckner, 2004).

Given these normal patterns of development, there are several logical possibilities as to when these cognitive processes might emerge as psychopathology risks. First, deficits in these processes might be present from early in development and serve as risks throughout the lifespan. This pattern is consistent with the evidence in ADHD, which is associated with poor EF from preschool to adulthood. There has been little research with young children in other areas, but many cognitive deficits are similarly associated with multiple forms of psychopathology in childhood, adolescence and adulthood, suggesting that cognitive process risks may be relatively stable across the lifespan. Second, cognitive deficits could be present from early in development, but only become associated with psychopathology in the presence of other risk factors or developmental vulnerability periods for psychopathology. Detecting this pattern requires long-term prospective longitudinal studies beginning in early childhood. Finally, cognitive deficits could emerge later in development, either because of neurodevelopmental abnormalities or neurodegenerative processes, as discussed later.

Developmental origins of cognitive risks. In addition to knowing when cognitive influences

emerge as salient predictors of psychopathology, an important and related question concerns the developmental origins of these cognitive risks. While many precursors have been postulated and examined, here we consider some of the more consistently conceptualized and examined factors, including negative parenting and social modeling, negative life events including abuse and trauma, and genetics.

Parenting. A prominent hypothesis is that cognitive products and processes may arise from maladaptive parenting practices. Based on a cognitive-developmental framework, parenting behaviors convey information to the child which may be internalized and subsequently contribute to the construction of beliefs and related processes (Bruce et al., 2006). Positive aspects of parenting (e.g., high levels of warmth, acceptance, autonomy promotion, consistency, and positive reinforcement) would be expected to contribute to the development of positive self-beliefs, whereas negative forms of parenting (e.g., high levels of criticism, rejection, control, and inconsistency) likely contribute to the formation of negative self-views and related processes (e.g., Beck, 1967; J E Young, 1999). Several cross-sectional studies support the association between maladaptive parenting practices and cognitive products. For example, regarding Beck's cognitive theory, high levels of parental criticism, indifference, and control and low levels of parental acceptance and care are associated with dysfunctional attitudes and cognitive distortions in youth (e.g., Bruce et al., 2006; Garber & Flynn, 2001; Liu, 2003) as well as a negative self-concept (e.g., Bruce et al., 2006; Liu, 2003). Similarly, high levels of parental criticism and control and low levels of parental care and acceptance correlate with a depressogenic attributional style in youth (e.g., Bruce et al., 2006; Garber & Flynn, 2001). While comparatively less research has extended this line of inquiry into developmental origins of cognitive processes, one study demonstrated that coded observations of negative parenting styles (authoritarian) and behaviors (criticism, negative affect) were associated with youths' attentional

biases to negative emotion, especially angry faces, in two independent samples (Gulley, Oppenheimer, & Hankin, 2013).

The few prospective studies to examine parental influences as precursors to development of cognitive products have been supportive. Maternal reports of high rejection and restrictiveness, when children were age five, prospectively predicted youths' self-criticism at age twelve (Koestner, Zuroff, & Powers, 1991). Also, observations of maternal feedback to child failure as well as temperament (from infancy to age 11, (Mezulis, Hyde, & Abramson, 2006); and from age 11 to 15, (Mezulis, Funasaki, & Hyde, 2011) along with maternal cognitive style (from age 11 to 15; (Mezulis et al., 2011) predicted development of youths' cognitive style. Last, maternal encouragement of emotion expression in daughters predicted higher rumination levels from age 11 to 15 (Cox, Mezulis, & Hyde, 2010).

What mechanisms might explain this relation between negative parenting and youths' cognitive risks? Mostly, social modeling processes have been evaluated. Evidence for this proposed process is mixed. More consistent evidence has accumulated for one variation of the modeling hypothesis, in which children model feedback communicated to them by their parents about events in the children's own lives rather than modeling their parents' own negative cognitions (e.g., Garber & Flynn, 2001; Mezulis et al., 2011, 2006). Less evidence exists for the modeling variant that children learn and adopt negative cognitions by observing their parents' own negative styles concerning their parents' own behavior. One study showed that children's attributional styles correlated with those of their mothers, but not their fathers (Sehman et al., 1984), whereas others have failed to replicate these findings (e.g., Garber & Flynn, 2001).

Negative life events. Second, certain types of negative life events, including (1) repeated exposure to stressors in multiple and interacting domains (e.g., family conflict, divorce, poverty) and

(2) traumatic events and maltreatment, may contribute as salient precursors in the development of cognitive products and processes. Accumulation of chronic exposure to negative events can lead to formation of negative cognitions that become deeply ingrained in self-structures (e.g., Rose & Abramson, 1992). Prospective research has supported this hypothesis with children's experience of negative events (Garber & Flynn, 1998) as well as various facets of family disturbance (i.e., divorce, abandonment, parental death, interparental conflict; (Rudolph, Kurlakowsky, & Conley, 2001). With respect to trauma and maltreatment, experience of these more adverse negative environments has been shown to associate with cognitive products in adults (e.g., Gibb et al., 2001; Hankin, 2005) and children (e.g., Dodge, Pettit, Bates, & Valente, 1995; Gibb et al., 2006) as well as various attentional (e.g., Pollak & Tolley-Schell, 2003) and memory (Parsons & Ressler, 2013) processes.

These robust associations between traumatic events and cognitive risks may occur for at least two reasons. First, after trauma exposure, youth attempt to understand the causes, meaning, and consequences of such events; especially when such events are chronic (recurrent across time) and pervasive (recurrent across situations), it is more likely that the child will develop stable, enduring cognitive beliefs to explain the maltreatment and to better predict and control future adverse events (Rose & Abramson, 1992). Formation of negative cognitive products may be particularly likely when the child is emotionally maltreated, as the abuser is directly providing the child with negative information (e.g., "you're stupid, lazy") that can directly become cognitive products (e.g., attributions, dysfunctional attitudes, early maladaptive schemas). Second, Pollak's theory of experience-dependent affective learning (2003) proposes that children are biologically prepared for emotion, and the formation of such systems depends, at least to a certain extent, upon salient environment inputs. Abusive experiences are associated with threatening, inconsistent, or excessive emotional signals, which in turn contribute to enhanced threat detection and attentional allocation to

predictive environmental and emotion cues (e.g., parents' angry face can probabilistically portend greater likelihood of physical abuse). What started as an initially adaptive learning process to detect threat to keep the child safe can extend into the formation of biased processing of social and emotional information across many contexts that are no longer objectively threatening (e.g., Pollak, Vardi, Bechner, & Curtin, 2005), and in turn, contribute to psychopathologies (Gulley et al., 2013; Shackman, Shackman, & Pollak, 2007).

Genetic and Biological Influences. Genetic, as well as other neural and biological influences, may serve as developmental precursors to the emergence of cognitive risks. First, significant associations have been observed between molecular genetics and cognitive products (e.g., rumination, Beevers, Wells, & McGeary, 2009; Hilt, Sander, Nolen-Hoeksema, & Simen, 2007) and processes (e.g., attention bias to negative emotion, (Gibb et al., 2013). Similarly, behavioral genetic research has demonstrated that cognitive risks, such as a negative attributional style (Lau, Rijdsdijk, & Eley, 2006), are moderately heritable. Furthermore, there is also evidence that some deficits in EF, memory and attention may be a particular form of genetic risk factor: endophenotypes (Gottesman & Gould, 2003). To be considered an endophenotype, a characteristic must be (1) associated with illness in the population, (2) heritable, (3) primarily state-independent (manifest even when illness symptoms are not present), (4) co-segregate with illness within families, and (5) be present in unaffected family members at a greater rate than in the general population. Criteria 1 and 2 are met for most cognitive processes across most disorders, since cognitive deficits are associated with mental illness as discussed in the literature review, and cognitive processes are largely heritable (Friedman et al., 2008). Moreover, there is evidence for criterion 3 in many cases— for example, cognitive deficits are present in individuals with MDD in remission and euthymic BD. Finally, criteria 4 and 5 are also met in many cases, with evidence that unaffected family members of individuals with schizophrenia, BD, OCD, and PTSD have cognitive

function which is more impaired than the general population but less impaired than their affected family members. While these data showing associations between genetic factors and cognitive risks are consistent with a developmental origins hypothesis, the cascade of mechanisms connecting initial genetic risk (e.g., polymorphic variation in particular candidate genes) to later individual differences in cognitive products and processes are unknown and in need of theoretical and empirical investigation.

Second, regardless of whether cognitive factors are a cause, correlate or consequence of psychopathology, **neurodevelopmental and neurodegenerative mechanisms** may play a role.

Neurodevelopmental processes involve an abnormal unfolding of brain development, while neurodegenerative processes involve loss of neural function over time. While neurodevelopmental processes are posited to play a role in many disorders, the lack of large prospective studies starting early in life has hampered efforts to test these hypotheses and determine the neural processes involved. Neurodevelopmental processes involved in psychopathology are hypothesized to be driven by both genetic and early environmental influences (e.g., prenatal exposure to toxins or infection, postnatal stress) and to take many forms, including alterations in neurogenesis and apoptosis, the formation and pruning of synaptic connections, and receptor expression (e.g., Rappaport, Giedd, & Gogtay, 2012a).

Perturbations in neurodevelopment could take the form of delays in normal developmental processes (e.g., children with ADHD undergo the same sequence of cortical maturation as healthy children, but the timing is delayed, Shaw et al., 2012), or abnormal developmental processes (e.g., atypical patterns of white matter connectivity in neonates at genetic risk for schizophrenia, Shi et al., 2012). While cognitive impairments are presumed to be consequences of neurodevelopmental abnormalities in neurodevelopmental models, links between neurodevelopment and psychopathology are potentially more complex. Early neurodevelopmental abnormalities could be either causes or

correlates of psychopathology, while perturbations in neurodevelopment after disorder onset could also conceivably be a consequence of psychopathology, via either direct alterations in neurobiology or indirectly by altering the individual's environment and experiences in a way that changes the course of development (e.g., transactional model).

The most common neurodegenerative model applied to cognitive deficits associated with psychopathology centers around disordered stress and immune responses (e.g., Conrad, 2008). Since chronic stress and elevated cortisol have been linked to many forms of psychopathology (e.g., adult depression, (Stetler & Miller, 2011); schizophrenia, (Walker, Mittal, & Tessner, 2008), this is a plausible pathway for neurodegeneration and subsequent cognitive deficits associated with psychopathology. Interestingly, abnormally low levels of cortisol (e.g., in PTSD, (Morris, Compas, & Garber, 2012) may also lead to neurodegeneration, although the mechanisms need specification (Conrad, 2008).

Neurodegenerative processes could be a consequence of psychopathology (e.g., psychopathology leads to disordered stress response, which leads to neurodegeneration), but could also be a cause (e.g., chronic stress leads to neurodegeneration which leads to psychopathology) or a correlate (e.g., chronic stress leads independently to both psychopathology and neurodegenerative processes), or the result of a positive feedback loop between these processes (Conrad, 2008).

However, in all of these cases cognitive deficits are presumed to be a consequence of the neurodegenerative processes. Since glucocorticoid receptors are highly expressed in the hippocampus and prefrontal cortex, neurodegenerative processes may have the greatest impact on memory and EF (e.g., Hinkelmann et al., 2009), perhaps accounting for their widespread impairment across disorders.

Cognitive consequences. While not exactly a factor that is contributing as a precursor, per se, to cognitive risk, researchers have hypothesized that experiencing sustained psychopathology may

further enhance the development of cognitive products and processes that persist after the peak of symptoms decline (i.e., cognitive scar). From this perspective, a youth's initial psychiatric episode may be caused by factors other than cognitive risks (e.g., a youth's coping abilities are overwhelmed following the experience of a severe stressor). As a consequence of and following the onset of the episode, the individual develops cognitive risks to the recurrence of psychopathology.

This exacerbation of cognitive risk into a more enduring cognitive vulnerability can occur through several (not necessarily exhaustive) pathways. First, youth who develop psychopathology frequently exhibit other non-cognitive risk factors, such as poor academic performance and impaired peer relations, and these failures and risks may lead youth to the development of particular cognitive products (e.g., beliefs about low abilities; lack of control over important outcomes) and processes (e.g., greater attention to, encoding of, and memory for such negative experiences). Second and related, experiencing psychopathology symptoms may facilitate access to negative memories and thoughts due to the priming effects of mood on cognition (Bower, 1981). These negative cognitions can be encapsulated and coalesce into enduring products and processes, especially if they persist over an extended period of time and while essential self-concepts and thinking styles are under construction and developing (e.g., middle childhood; early adolescence). Finally, there can be dynamic, transactional relations between the stressful life events that may have triggered the onset of the disorder, and in turn, youth may behave in ways that reinforce the development of the cognitive risks. For example, a child with poor social skills, who consequently encounters rejection, may learn to expect such rejection. Subsequent behavior (e.g., withdrawing from social activity) may contribute to further social rejection, and consequently reinforce and strengthen the developing cognitive products and processes.

A first, necessary step to demonstrate support for these and other potential scar processes is

that cognitive products and processes worsen after experience of psychopathology. Research that examined the cognitive scarring after various forms of psychopathology has been mixed. Consistent with a cognitive scar model, deficits in EF, memory and attention remain in individuals in remission (e.g., euthymic BD, remitted depression, see Cognitive Processes). Findings for cognitive products, such as negative attributional style or rumination, have found mixed evidence for the cognitive scar hypothesis (e.g. remitted depression and BD; see Cognitive Products). However, since this evidence largely comes from cross-sectional studies, it is possible that cognitive deficits preceded illness onset, or that there is a transactional positive feedback loop between cognitive deficits and psychopathology. Answering these questions will require more prospective longitudinal studies, starting before disorder onset and following individuals over the course of their illness and into remission.

Longitudinal measurement invariance; Stability and change. Investigating the degree of stability and change in cognitive influences to psychopathology is a fundamental area of inquiry in developmental psychopathology. Yet, surprisingly sparse research has examined this core issue: “the degree of stability of cognitive vulnerability from childhood to adulthood is unknown” (Michael Rutter, Kim-Cohen, & Maughan, 2006, p. 283). Examining the degree of stability and change in cognitive risks, and the likely variance in stability estimates across key developmental periods, can vitally inform mechanisms that may help to explain the strongly chronic, recurrent nature of most forms of psychopathology after initial onset and symptom increases occurring with most child- and adolescent-onset disorders.

Examining **measurement invariance** across development is a first and necessary step when considering both stability and change in cognitive influences to psychopathology, but this is a little researched and frequently ignored topic. In essence, the core question is whether any particular

cognitive product or process, at the latent construct level, is the same when assessed with available measures across development. For example, multiple measures exist to assess individuals' negative cognitive style for different ages: young children (Conley et al., 2001), older children (Mezulis et al., 2006), adolescents (Hankin & Abramson, 2002), and adults (Haefffel et al., 2008). It is generally assumed that each of these manifest measures is assessing the latent construct of a negative cognitive style, and the evidence that each of these measures predicts later depressive symptoms, as the theory postulates, is presumed and taken as *prima facie* evidence to demonstrate that the manifest measures are, indeed, assessing the same core latent construct. However, virtually no research has been conducted to rigorously and properly examine whether the extant manifest measures used presently by investigators are, in fact, assessing the same latent construct over different developmental periods (i.e., measurement invariance; (Widaman, Ferrer, & Conger, 2010).

Equivalently, using the same manifest measure across different developmental periods (e.g., childhood through adolescence and adulthood) still necessitates examination of measurement invariance; utilization of the same measure cannot be assumed to assess the same latent construct across development. Establishing that a particular measure, at the manifest level, is assessing the same theoretical construct and process at the latent level, is a fundamental step. Yet this critically essential work has not been conducted, so research investigating vital developmental psychopathology questions of cognitive influences, including stability, change, and developmental origins, rest on the untested assumption that presently used manifest measures of cognitive products and processes demonstrate a reasonable degree of measurement equivalence over time and across development.

Measurement invariance issues also connect to and raise questions concerning homotypic and heterotypic continuity. For example, is rumination exactly equivalent (homotypic continuity), at both

the observed measurement and at the conceptually defined process level, across development (e.g., ages 5, 8, 13, 16, 25, 40, 68), or is rumination similar at the latent process level (e.g., perseverating on negative mood), albeit with some change in its manifestation and form (i.e., heterotypic continuity)? Related, trying to establish the degree of continuity, especially whether the particular cognitive product and process conforms best to homotypic or heterotypic continuity, is further complicated by a lack of established theory and empirical knowledge on the overall hierarchical structure and taxonomy that organizes and connects the different cognitive products and processes together. How does this architecture and structure change across development (e.g., consolidation of cognitive influences) and as a function of psychopathology (e.g., consequence effects)? Issues of structure and consolidation are discussed in greater detail later in the next section, but it's important to highlight here that homotypic and heterotypic continuity concerns directly relate to issues of structure and consolidation across development.

Assuming that observable methods to assess cognitive influences exhibit reasonable measurement invariance, then processes underlying the **degree of stability and change** in these cognitive risks can be examined. A useful and rigorous approach to understanding processes in stability and change derives from conceptual and empirical advances in research on basic personality development (Caspi, Roberts, & Shiner, 2005; Fraley & Roberts, 2005), including examination of rank-order stability and mean-level changes over time. Different processes, including (1) a trait-like model, (2) a contextual/autoregressive model, and (3) a combined trait/contextual model, can explain how cognitive risks maintain rank-order, or test-retest, stability over time. Trait models predict that the empirically obtained test-retest correlations will be invariant as the period between test-retest interval increases because there is a stable, enduring force (i.e., the trait cognitive risk) that organizes the manifestation of measured cognitive risks over time. In contrast, contextual models predict that

the magnitude of the test-retest correlations for cognitive risk will decrease monotonically as the length of the test-retest interval increases (i.e., an autoregressive simplex pattern; (Kenny & Zautra, 2001) because there is no enduring influence that drives the stability over time.

Testing which process (i.e., trait, autoregressive, combination) may best account for the degree of stability in cognitive risk over time requires that multiple waves of data be used. The typical two-time point study most typically used to demonstrate test-retest reliability is inadequate for formally and rigorously investigating the processes underlying the degree of stability observed over those two time points, regardless of the length of time (Fraley & Roberts, 2005), because it is the pattern of test-retest correlations over time, not the strength of the test-retest correlation, that indicates whether the particular cognitive influence is understood to be organized best from a trait, contextual, or a combined process. With multiple assessments of longitudinal data, the pattern of test-retest correlations can be examined to evaluate whether a trait-like or autoregressive contextual model best explains the rank-order stability of data over time. Moreover, longitudinal analyses can determine the mean levels of stability over time for the participants on average.

To date, little research has examined the degree of stability and change, and the underlying processes that give rise to that stability, in cognitive risks to psychopathology. The first examination of the processes underlying stability of cognitive vulnerability, in any age group, used a diary design with late adolescents who completed daily ratings of the cognitive inferences they made for the most negative event experienced every day for a month (Hankin, Fraley, & Abela, 2005). Findings showed mean-level stability: negative inference scores did not change on average over 35 days. Moreover, moderately stable rank-order stability was observed, and importantly, an enduring, trait-like model best explained the pattern of test-retest stability. Building on this approach and investigating additional cognitive products in younger adolescents (ages 11-16;

6th-10th grades), Hankin (2008b) examined the degree of stability and change in negative cognitive style, dysfunctional attitudes, and rumination across four time points. A negative cognitive style demonstrated mean-level stability, whereas rumination and dysfunctional attitudes showed some mean-level change. The magnitudes of test–retest reliabilities were moderately high for a negative cognitive style (mean $r = .52$), and more modest for rumination (mean $r = .28$) and dysfunctional attitudes (mean $r = .26$). The pattern of these test–retest reliabilities over time was best explained by relatively trait-like enduring processes for a negative cognitive style and dysfunctional attitudes, whereas both enduring and contextual dynamics contributed to the moderate stability for rumination. Future research is needed to continue this line of work to younger ages to better establish when these cognitive products begin to coalesce into the enduring, relatively trait-like risks to psychopathology and to expand this approach to understanding the dynamics underlying stability and change to other cognitive products and processes across various developmentally salient age groups.

Taken together, these findings showing that a negative cognitive style, in particular, already demonstrates modestly strong stability, and that enduring dynamic forces underlie this stability, have interesting applications when synthesized with established developmental epidemiological facts. It is intriguing that a negative cognitive style is already relatively trait-like by age 11 when the modal increase in depression rates occurs later in adolescence (especially ages 15-18; (Hankin et al., 1998). Moreover, these findings open new questions that convey interesting translational implications for the timing of delivering evidence-based efforts to reduce depression rates. Would it be more effective to implement cognitive-behavioral (CB) depression preventions (Brunwasser, Gillham, & Kim, 2009) before certain cognitive risks, such as a negative cognitive style, have crystallized into relatively trait-like vulnerabilities to depression (i.e., before age 11)? How long do the effects of CB-based preventions last in changing these cognitive vulnerabilities that are believed to be important

mechanisms of change in the prevention of depression? Meta-analytic reviews of depression prevention trials indicate that effects are generally short-lived (Horowitz & Garber, 2006; Stice, Shaw, Bohon, Marti, & Rohde, 2009), and similarly, research on CB treatments of depression and anxiety reveals these psychotherapies exhibit enduring effects (e.g., relatively improved effects lasting up to 5 years), although the psychoactive mediating mechanisms appear to be mainly palliative and do not endure (Hollon, Stewart, & Strunk, 2006). As such, would reductions in core cognitive risks, if changed in late childhood, persist over several years to meaningfully decrease the surge in depression rates that are most likely to occur with modal age of onset in later adolescence?

Escaping the Silos: Building Integrative Models Across Boundaries

Taxonomy and structure of cognitive risks: Informing consolidation in the interrelation among cognitive risks. As alluded to earlier, there exists no theoretically specified nor empirically determined structure of either cognitive products or processes, separately, nor both cognitive influences together in a hierarchical taxonomy model. The main issue is that numerous cognitive products, in particular, have been postulated and investigated as putative risks to psychopathology. Yet, little is known about how these different cognitive products (each of which carries a different name and derives from supposedly relatively unique theoretical underpinnings), relate to each other, and how much they overlap. In other words, do cognitive products demonstrate factorial independence—with other cognitive products as well as possibly conceptually similar individual differences (e.g., neuroticism, as a personality trait, that shares some conceptual and empirical overlap with several cognitive products)?

In contrast and as a shining example of what is needed, considerable research, both theoretical and empirical, has reasonably well established the structure of temperament (e.g., Rothbart & Bates, 2006) and personality traits (e.g., Caspi et al., 2005). A consequence of a well-established personality

taxonomy is that existing traits, as well as potentially new ones, can be placed systematically, organized, and understood conceptually within the larger trait taxonomy and structure. Moreover, a fair amount of work has investigated questions of personality development, including stability and change in traits (e.g., Roberts & DelVecchio, 2000) as well as developmental changes in structure and underlying processes contributing to the degree of stability (e.g., Fraley & Roberts, 2005) of these traits over time.

However, the essential theoretical and empirical work has not been conducted on the hierarchical structure of many of the cognitive influences that are the focus of this chapter. This is another concrete example of the general silo approach in much of the scientific inquiry that has taken place with cognitive influences to psychopathology. The factor structure of many cognitive processes has been well defined, at least in healthy individuals (e.g., see Executive Function for a review of the factor structure of EF component processes). However, only a handful of factor analytic studies have examined the factor structure of cognitive products. Those done with young adult samples (e.g., Hankin, Lakdawalla, Carter, Abela, & Adams, 2007) show that each of the cognitive risks (e.g., negative cognitive style, dysfunctional attitudes, rumination) are moderately correlated with each other but factorially independent (i.e., not completely overlapping at the latent level) and are mostly separate from related, but non-cognitive, constructs (e.g., low self-esteem, neuroticism, depressed mood). Factor analytic studies of youth are more mixed but tend to suggest that some of these cognitive products overlap and are less distinct at younger ages (e.g., Adams, Abela, & Hankin, 2007). To our knowledge, no other research has examined the factorial structure and latent taxonomy of other cognitive products.

While it is a clear priority to establish a hierarchical structure of both cognitive products and processes to psychopathology across developmental periods, another important, yet under investigated,

area is evaluating how the various cognitive risks interrelate with one another and potentially become more consolidated over time. Examining the interrelation among different risks is important because the various forms of cognitive products, for example, may all emerge and stabilize along a similar time-course, but each of these cognitive risks may be relatively independent of each other earlier in development and then begin to become increasingly more interrelated and coalesce. Indeed, this pattern of increasing interrelatedness would be expected based on research in the temperament and personality development literatures (e.g., Caspi et al., 2005), in which many more narrow, lower-order factors are observed early in development, but by middle adolescence and throughout adulthood, the standard personality taxonomy (e.g., Big-5 factors) is routinely obtained. The results from the few factor analytic studies of depressogenic cognitive products suggest that these cognitive risks are more distinct in children than in adolescents and young adults (Hankin et al., 2007). It appears that the various cognitive risks may initially be relatively independent of one another but then become increasingly more inter-related during the transition from childhood to adolescence. As multiple cognitive products potentially coalesce into a consolidated *set* of moderately inter-correlated cognitive risk, youths' degree of vulnerability to psychopathology may potentiate. An interesting hypothesis in need of empirical examination is that this contamination and consolidation process may occur around the same time that prior researchers (Susan Nolen-Hoeksema et al., 1992; Turner & Cole, 1994) earlier hypothesized that some cognitive risks first emerge.

Given that cognitive risks appear to become more inter-related with one another with progressing age, different approaches towards conceptualizing the relationship among multiple cognitive risks may be optimal for youth at different developmental stages. When youth's cognitive risks are still relatively distinct, knowledge of any particular factor may convey minimal information about overall degree of vulnerability to psychopathology, so a weakest link approach may be the most appropriate

conceptualization at this stage: The child's strongest cognitive risk factor may likely be the best indicator of his/her propensity to engage in negative thinking leading to psychopathology. As cognitive risks become more inter-related with one another over time, however, knowledge of a child's level with respect to any given factor provides information about his/her levels with respect to other cognitive risks. At this point in development, an additive or multiplicative model may become more appropriate than a weakest link approach as the presence of multiple vulnerabilities may become an equally, if not more, important indicator of the likelihood the individual engages in negative thought processes contributing to psychopathology (see John R Z Abela & Hankin, 2008 for expanded discussion of the strengths and weaknesses of each of these models).

It is axiomatic in developmental psychopathology that there exist multiple developmental pathways (equifinality) and that the various influences leading to these pathways cut across multiple levels of analysis (e.g., from genes to neurocircuits to behaviors and cognitions in relevant social contexts in an ecological framework). Given that multiple factors and processes are needed to predict known heterogeneity in causes for prevalent psychopathologies, it is essential to formally organize and consider how the various risk and resilience factors and mechanisms are structured together (i.e., a weakest link, additive, or multiplicative approach), when across development each of these model approaches provides the best fit for organizing cognitive risks, and how these best predict psychopathologies and for whom.

Links between cognitive processes and products: Still a bridge too far? In addition to determining the structure within cognitive products and processes, a further challenge is to determine how cognitive products are related to cognitive processes. Cognitive products, such as attributions, biases, and ruminative thoughts must necessarily arise from basic cognitive processes. However, efforts to understand how cognitive processes and products are linked are still in their infancy, akin to

the early years of cognitive neuroscience, when scientists knew that neural events must give rise to cognitive processes but had very little idea of how they did so. Just as cognitive neuroscience has made great strides in discovering links between brain and cognitive processes over the past twenty years, the field seems poised to make significant progress in linking cognitive processes and products in coming years.

Indeed, in a few areas progress is already being made towards this goal. For example, two closely related models have recently been proposed, linking EF deficits to cognitive products in depression (Joormann, Levens, & Gotlib, 2011; Joormann, 2010b; Koster, De Lissnyder, Derakshan, & De Raedt, 2011), and aspects of these models have been empirically supported (e.g., Berman et al., 2010; Joormann & Gotlib, 2008; Zetsche, Avanzato, & Joormann, 2012). Specifically, according to these models, the experience of a negative event triggers negative mood and cognitions, which are normally transient, with mood-congruent cognitions quickly being replaced by more positive thoughts that regulate and repair the negative mood. However, depression is associated with deficits in EF, which lead to difficulty shifting attention and updating the contents of working memory to remove negative material and replace it with the more positive material that would normally aid in mood repair. As a result, negative material remains in working memory, leading to increased rumination, decreased reappraisal and impaired problem solving, which in turn reinforce and maintain the negative mood and activation of negative cognitions (Joormann et al., 2011; Joormann, 2010b; Koster et al., 2011).

In other areas, there has been speculation regarding how cognitive products and processes may be linked. For example, there are potential parallels between hostile attributional bias and attentional bias towards negative information. However, research on these topics is still nascent, and efforts to understand the mechanisms giving rise to cognitive products are likely to face some significant

challenges distinct from those faced by efforts to map cognitive processes onto neural mechanisms. In particular, many cognitive processes rely on brain networks that, while complex, are fairly well defined (e.g., the medial temporal lobe memory system and the frontal-parietal-striatal EF network). Identifying the brain networks involved then provides traction for understanding the component mechanisms (e.g., unique properties of the hippocampus that support memory). However, cognitive products seem highly unlikely to arise from dedicated brain networks (e.g., there is not likely to be an “attribution network”). Rather, cognitive products are most likely emergent phenomena of multiple interacting cognitive processes, supported by widely distributed neural activity, making it difficult to gain traction in understanding the mechanisms involved.

How do we get there from here? Bridging multiple levels of analysis. The research reviewed in this chapter spans many levels of analysis, from probing people’s accessible thoughts and behaviors through self-report, to assessing their behavioral performance on laboratory tasks, to measuring specific aspects of brain structure and function, and finally connecting these to psychopathological phenotypes. Furthermore, within each of these levels of analysis, there are myriad methodological approaches. Each level of analysis provides valuable information, and each methodological approach has its own strengths and weaknesses. Certainly, there is no one right approach. Rather, like the parable of the blind men and the elephant, each approach provides only partial information, which when taken alone can be misleading.

To give just one example, fMRI provides valuable information about which areas of the brain may be functioning differently in individuals with psychopathology. However, there are limitations to what fMRI can tell us about *how* those brain areas are functioning differently. For example, fMRI signal reflects a mixture of excitatory (e.g., glutamatergic) and inhibitory (e.g., GABAergic) neural activity, with the contributions of each depending on the task, the brain region, and many unknown

variables (e.g., Logothetis, 2008). Thus, reduced fMRI signal (hypoactivation) can be the result of reduced activity of excitatory neurons, inhibitory interneurons, or both— the specific neural mechanism underlying the observed change in brain activation cannot be determined by fMRI alone. Thus, while correlational mapping of fMRI brain activation and psychopathology has made important contributions, it must be combined with other methods to move beyond the *where* and *how much activation* questions it can address to specific mechanistic *how* questions it can not (e.g., Munakata, Chatham, & Snyder, 2012b).

Likewise, deficits on behavioral tasks or differences on self-report measures may arise from a mixture of sources (e.g., deficits in different cognitive processes or use of different strategies may contribute to poor performance on a task, and responses to questions about cognitive products may arise from different streams of biased information processing). Thus, in order to build more complete cognitive models of developmental psychopathology that can address *how* questions, to construct a picture of the whole elephant as it were, it will be necessary to combine evidence across multiple methods and levels of analysis.

Areas in which progress in generating such integrated models has been made include linking EF deficits in schizophrenia to specific abnormalities in the dopamine system (Barch & Ceaser, 2012), attention biases in depression to specific abnormalities in the serotonin system (Gibb et al., 2013), and connecting cognitive risks to multiple levels of analysis spanning genetics, neurobiology, physiology (e.g., Hankin, 2012).

An example of an area in which multiple levels of analysis are just beginning to be linked is executive function deficits associated with depression. Here, a more detailed model is slowly emerging that has combined findings across disparate disciplines and methods. People with depression report difficulties with concentration and completing goals (symptom level); behavioral testing indicates

deficits on a wide range of EF tasks, suggesting a problem with processes common to all EF tasks, such as maintaining goals in working memory (behavioral level, (Snyder, 2013); neuroimaging research finds that individuals with depression have less activation in prefrontal brain areas involved in working memory (neural networks level, (e.g., Fitzgerald, Laird, Maller, & Daskalakis, 2008); spectroscopy studies suggest that these lower levels of activation may be due to reduced glutamatergic function (neurotransmitter level, (e.g., Yüksel & Ongur, 2010); and finally, computational computer simulations and neural recordings in animals suggest that working memory in prefrontal cortex depends on sustained neural firing triggered by glutamatergic signaling (cellular level, (e.g., O'Reilly, Hazy, & Herd, 2013).

Fitting these pieces of evidence together across levels of analysis can generate a hypothesized model— concentration difficulties associated with depression are due to reduced prefrontal glutamatergic functioning leading to impaired ability to maintain goals in working memory through sustained neural firing. This model may prove to be wrong, but it has the virtue of generating specific, testable predictions (e.g., the largest impairments should be found on tasks that most heavily tap active goal maintenance) and implications for treatment (e.g., glutamatergic drugs should improve EF and concentration in individuals with depression; (e.g., see Hashimoto, 2009) for promising research on novel glutamatergic antidepressants). However, such multi-level integrative models are still relatively rare, and to our knowledge no model has thus far spanned from cognitive products to specific neural mechanisms. Building such models will undoubtedly be challenging, and will require a major interdisciplinary effort, but we believe they have tremendous potential for understanding the mechanisms involved in the cognitive aspects of psychopathology at a level that will enable the generation of new developmental clinical scientific knowledge and translation to new treatment and prevention approaches.

Transdiagnostic approaches: Bridging multiple forms of psychopathology. One theoretical approach that has received increasing attention is the study of common processes across seemingly separable constructs. This is known as a *transdiagnostic* approach to examining risk for psychopathology (e.g., McLaughlin & Nolen-Hoeksema, 2011). Our review of cognitive products and processes in relation to multiple prevalent psychopathologies suggests that certain cognitive risks may broadly predict several forms of emotional and behavioral disorders. Here we comment on two issues to consider that require additional future research with respect to a transdiagnostic approach to cognition and development of psychopathology.

The transdiagnostic approach to studying *cognitive* constructs, in particular, can be illustrated by an examination of rumination, worry, and post-event processing, which are three cognitive constructs hypothesized to fall under the common process of repetitive negative thinking (RNT). It is thought that these three cognitive constructs are similar in that they all involve a repetitive thought *process*, however they are distinguishable across different forms of psychopathology by their *content*. Rumination is commonly linked to depression, worry to generalized anxiety disorder, and post-event processing to social phobia (see McEvoy, Mahoney, & Moulds, 2010 for review). For example, in an effort to disentangle the common and unique components of these three cognitive constructs, McEvoy and colleagues (2010) factor analyzed common measures of RNT, including worry, rumination, and post-event processing, and they identified a common RNT thinking factor. This common RNT scale was associated with symptoms of depression, anxiety, general distress, anger, and shame. This study demonstrates that putatively different cognitive products may be more appropriately and parsimoniously conceptualized as a single common process of repetitive negative thinking across various types of psychopathology and emotional states.

On the other hand, cognitive factors that appear transdiagnostic at one level of analysis may not be

when more detailed measures at multiple levels of analysis are considered. Just as many problems with a car (e.g., a dead battery, broken starter, or being out of gas) could all lead to the same outcome (the car won't start), the same cognitive endpoint might be reached by many different underlying mechanisms (*equifinality*). For example, at the level of performance on neuropsychological tasks, EF deficits appear to be a transdiagnostic feature of psychopathology. However, as we reviewed, in some cases these shared behavioral deficits may arise from distinct neural mechanisms (e.g., perturbations in different neurotransmitter systems). Thus, determining whether a product or process is truly transdiagnostic requires escaping both diagnostic and methodological silos to consider underlying mechanisms at multiple levels of analysis.

One area where progress has been made in doing so is EF. Here, we discuss in some detail efforts to understand why transdiagnostic impairments in EF occur, as an example of how interdisciplinary research can shed light on transdiagnostic processes more broadly. What gives rise to broad patterns of impairment in core EF processes across most disorders? First, these deficits cannot be easily explained by non-specific factors such as psychomotor slowing, differences in IQ or education, or medication use (Barch, 2005; Forbes et al., 2009; Snyder et al., 2013; Snyder, 2013). Second, in most cases, effect sizes are similar across the core EF domains, suggesting that psychopathology may be associated with impairment in what is common across EF tasks (common EF, posited to be monitoring for and maintenance of task goals and contextual information; (Miyake & Friedman, 2012), rather than a specific aspect of EF. Future research is needed to refine understanding of how such broad EF deficits arise. One possibility is that impaired function in brain networks involved in EF, including PFC, may lead to broad impairment in EF.

Indeed, extensive neuroimaging evidence indicates that individuals with many forms of psychopathology have structural and functional abnormalities in brain networks involved in EF. Meta-analyses have demonstrated reduced prefrontal grey matter volume in individuals with

schizophrenia (e.g., Yu et al., 2010), BD (Yu et al., 2010), depression (Bora, Fornito, Pantelis, & Yucel, 2012), and OCD (Menzies et al., 2008a). Alterations in prefrontal activation during EF tasks are also widespread across disorders. While both decreased (hypoactivation) and increased (hyperactivation) relative to healthy controls has been reported, in general reduced activation is found when task performance is impaired, and increased activation is found when task performance is unimpaired, consistent with inefficient processing (Cortese et al., 2012; Menzies et al., 2008b; Minzenberg, Laird, Thelen, Carter, & Glahn, 2009; Patel, Spreng, Shin, & Girard, 2012; Snyder, 2013).

Thus, multiple forms of psychopathology all appear to be associated with prefrontal volume reductions and functional inefficiency. However, the cellular level abnormalities underlying these broad changes in prefrontal function may vary across disorders. For example, while depression is associated with *reduced* glutamate function (e.g., Luykx et al., 2012), individuals with BD show *increased* glutamate levels in PFC (Gigante et al., 2012). The role of cellular level mechanisms in EF impairments has been most extensively investigated in individuals with schizophrenia. Phasic dopamine bursts, which normally serve to regulate updating of goals and information in working memory, are dysregulated in schizophrenia, leading to a failure to appropriately update relevant information into working memory and lack of stability of representations in the face of interference (Barch, 2005; Eisenberg & Berman, 2010). Thus, altered dopamine function is one key candidate mechanism that may underlie impaired common EF in schizophrenia, leading to impairments across all EF tasks.

In addition, PFC dysfunction may differentially impact different posterior and subcortical areas in different disorders. For example, in the case of PTSD, hypoactivity of the prefrontal-parietal EF network is hypothesized to lead to a loss of top-down control over emotion and memory systems (e.g.,

the amygdala and hippocampus, (Patel et al., 2012), as well as EF impairments. In addition, the largest deficit for individuals with OCD was for updating, which is believed to depend critically on striatal gating of information into prefrontal cortex (Hazy, Frank, & Reilly, 2007). This suggests that updating may be particularly impaired in people with OCD, since both striatal and prefrontal dysfunction may contribute to deficits on updating tasks. However, there have been few studies of updating in individuals with OCD, so additional research is needed to explore this possibility. Thus, while multiple disorders may be associated with prefrontal dysfunction leading to broad impairment in EF, the underlying neurobiological abnormalities, at the cellular and network level, may be quite distinct.

A further important question is how these prefrontal abnormalities, and thus EF deficits, arise. There is some evidence that PFC abnormalities and impaired EF may be endophenotypes for psychopathology. Significant, though more subtle, impairments in EF and prefrontal abnormalities have been reported in currently healthy individuals with greater genetic risk for schizophrenia (Barch, 2005), BD (Bora et al., 2009), OCD (Menziés et al., 2007) and PTSD (Gilbertson et al., 2006).

These findings suggest that EF deficits may precede, and potentially be a risk factor for, psychopathology. Although there have been few longitudinal prospective studies which could speak to this issue, there is some evidence that EF deficits may precede illness onset. Specifically, early adolescent EF predicts adult psychosis (Cannon et al., 2006b), preschool EF predicts middle-childhood ADHD (even controlling for preschool ADHD symptoms; (Campbell & Stauffenberg, 2008), and pre-trauma EF predicts post-trauma PTSD (Parslow & Jorm, 2007). On the resilience side, individuals who have experienced trauma but do not have PTSD have greater PFC activity not only compared to individuals with PTSD, but also compared to healthy controls who have not experienced trauma (Patel et al., 2012). It is thus possible that individual differences in prefrontal function and resulting differences in EF predispose some people to develop psychopathology, while buffering others (Aupperle et al., 2012). These

patterns are consistent with neurodevelopmental models of psychopathology, in which disorders are posited to be the behavioral outcome of aberrant neurodevelopment that begins long before the onset of clinical symptoms and is caused by a combination of genetic and environmental factors (Rappaport, Giedd, & Gogtay, 2012b; Seidman, 2006).

Translational Implications

This section applies what is known about cognitive products and then processes in relation to the treatment and prevention of common psychopathologies. We consider some future directions that can be taken to further reduce mental health burden of individuals across the lifespan based on theoretical and empirical knowledge covered in this chapter.

Since the initial, ground-breaking clinical work of Beck (1967) and Ellis (1957), cognitive (and cognitive-behavioral, CB) therapies have been developed and rigorously evaluated for many prevalent psychopathologies. While there are important, subtle differences in these CB interventions for different disorders, all of these evidence-based interventions share an emphasis on cognitive products and processes as essential mechanisms that are hypothesized to produce meaningful symptom, emotion, and behavior change. Indeed, perusal of empirically supported treatments (e.g., see Barlow, 2008) for adults (e.g., Division 12 of APA) and youth (e.g., Division 53 of APA) reveals that CB-based interventions are established for depression, general child anxiety, Panic Disorder, PTSD, GAD, SAD, substance abuse and dependence, binge eating disorder, bulimia nervosa, and disruptive behavioral problems. Other CB-based treatments have shown promise for other disorders, including schizophrenia (Tarrier, 2005) and BD (Miklowitz, 2008). In addition to treatments for diagnosed psychopathologies, several CB-based preventions have been demonstrated to reduce likelihood of future increases in symptoms of and episodes for depression (Horowitz & Garber, 2006; Stice, Rohde, Gau, & Wade, 2010), anxiety (Fisak, Richard, & Mann, 2011), disruptive behavioral problems (Powell

et al., 2011), and comorbid internalizing and externalizing problems (e.g., Weiss, Harris, Catron, & Han, 2003).

Cognitive products as mediating mechanisms and moderators in CB-based interventions.

In addition to CB-approaches as evidence-based interventions to treat and prevent psychopathology, research has also examined various cognitive risks as mediating mechanisms of intervention effects. Despite the central importance of cognitive products and processes as putative mediators of change in CB-approaches, it is surprising that relatively few studies have empirically examined cognitive influences as mechanisms (Hundt et al., 2013 with adults; C. A. Webb, Auerbach, & Derubeis, 2012 with youth). It is worth noting that this relative paucity of cognitive mediation research is not unique to investigation in CB-based interventions, but rather applies broadly to general lack of understanding of how and why psychotherapy is efficacious and leads to change (e.g., Kazdin, 2009). Such research on mediating mechanisms is important also because it can provide evidence regarding cognitive risks as a possible causal risk factor in developmental psychopathology (Kazdin et al., 1997; Kraemer et al., 2001).

Much of the research on cognitive mechanisms of change in psychotherapy has focused on depression (e.g., Kazdin, 2007), and most of that has used adult samples (see Garratt, Ingram, Rand, & Sawalani, 2007 for review). As such, the following studies reflect this knowledge base to provide the most evidence-based examples of how cognition may explain how psychotherapies work. Generally a number of studies have shown that CBT for depression in adults (Garratt et al., 2007) and adolescents (Weersing, Rozenman, & Gonzalez, 2009) is related to reductions in cognitive products, but not all have formally examined mediation to link change in negative cognitions to subsequent change in symptoms. Among youth, CBT for anxiety produced moderate to large effects in various theoretically specified mechanisms, whereas CBT for depression yielded small effects for change (Chu & Harrison,

2007).

Some individual studies suggest that particular cognitive risks may mediate CB-based intervention effects. A negative attributional style has been shown to mediate the efficacy of CBT for depression (e.g., Barber & DeRubeis, 2001). Dysfunctional attitudes (or automatic thoughts, depending on the study) mediated effects of CBT for adult depression (e.g., Beevers, Keitner, Ryan, & Miller, 2003; Warmerdam, van Straten, Jongma, Twisk, & Cuijpers, 2010) and adolescent depression (e.g., Jacobs et al., 2009). Moreover, mediation through dysfunctional attitudes has been shown in CBT for adult alcoholism comorbid with depressive symptoms (Ramsey, Brown, Stuart, Burgess, & Miller, 2002), and preliminary mediation of effects for CBT were found in an open trial for schizophrenia (Morrison et al., 2012). There have been demonstrations of specific mediation for dysfunctional attitudes in CBT versus interpersonal psychotherapy for adult depression (Quilty, McBride, & Bagby, 2008) and versus pharmacotherapy (D. J. A. Dozois et al., 2009; Garratt et al., 2007). Indeed, the preponderance of data suggest that antidepressants modify many of the cognitive biases in depression (e.g., Roiser, Elliott, & Sahakian, 2012). Moreover, several brain areas and cognitively mediated neurocircuits implicated in depression etiology show change resulting from CBT as well as pharmacotherapy (Roiser et al., 2012). Anxiety schema change (Teachman, Marker, & Smith-Janik, 2008) and panic appraisals (Cho, Smits, Powers, & Telch, 2006) in Panic Disorder mediated effects of CBT for panic. Dysfunctional trauma-related appraisals demonstrated temporal precedence in mediation for Trauma-Focused CBT in treatment for PTSD (Kleim et al., 2012). Self-efficacy beliefs mediated efficacy in CBT, specifically in contrast to IPT, for bulimia among adults (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002).

Regarding rumination as a putative mechanism, research has shown that reductions in rumination mediated effects in mindfulness based CBT for adult depression (Van Aalderen et al., 2012). It has

been suggested that addressing repetitive negative thinking, broadly defined, represents an efficient way to target the cognitive risk and maintenance factors that are transdiagnostically present across multiple psychopathologies (McLaughlin & Nolen-Hoeksema, 2011). Mindfulness-based interventions, in which clients are taught to emotionally disengage from recurrent negative thoughts (e.g., rumination) instead of letting them adversely affect one's mood, may be another therapeutic approach to reducing repetitive negative thinking and psychopathology (Rood et al., 2010).

While this review is consistent with the view that cognitive products mediate effects of CB-based interventions, it is noteworthy that few studies have rigorously examined essential mechanism questions and have properly examined mediation (Kazdin, 2009). Most study designs do not go beyond showing that CBT is associated with reductions in cognitive products and symptoms and that these two variables are correlated, so the temporal precedence in the vast majority of cognitive mechanism studies of therapy is unknown. Indeed, results from a CB-based prevention trial of adolescents at-risk for depression highlight that cognitive risks may be changed by the intervention, but the temporal precedence of change in cognition prior to change in symptoms is not guaranteed (Stice et al., 2010) and still needs to be established.

In addition to examination of mediation, a few studies have investigated whether cognitive products moderate treatment outcome in depression. In a review of adult CBT for depression studies, dysfunctional attitudes were reported to moderate CBT response (Hamilton & Dobson, 2002). The available studies with depressed adolescents suggest likewise. Cognitive distortion predicted depression in treatment (Brent et al., 1998). High cognitive distortions helped depressed adolescents receiving combined pharmacotherapy and CBT (Curry et al., 2006), although dysfunctional attitudes did not predict adolescent treatment response across intervention modalities in contrast to the adult research (Jacobs et al., 2010).

Cognitive products as mediators and moderators in prevention work. With respect to preventive interventions, selective and indicated programs, such as those focused on youth with enhanced cognitive risk (e.g., negative cognitive style), have been demonstrated to be more effective than universal preventions for youth depression (Horowitz & Garber, 2006), although definitive tests of moderation have not been examined (Garber, Korelitz, & Samanez-Larkin, 2012). A negative cognitive style partially mediated the effects of a CB-based prevention, the Penn Resiliency Program, on depressive symptoms (Brunwasser et al., 2009).

Cognitive processes as mediators in interventions. Regarding translation implications for the cognitive processes that comprised the focus of this chapter, pre-treatment cognition has been shown to predict treatment response. In particular, EF and episodic memory task performance predict pharmacotherapy response in individuals with depression (McLennan & Mathias, 2010), schizophrenia (Y.-K. Kim et al., 2008), OCD (e.g., D'Alcanta et al., 2012), and BD (e.g., Gruber, Rosso, & Yurgelun-Todd, 2008). Although the precise reasons are unclear (e.g., there could be neurobiological explanations), poor medication compliance is the most likely and parsimonious explanation because these findings hold across several different types of psychiatric medications. Moreover, over-general autobiographical memory predicts poor clinical outcomes after treatment, over and above what is predicted by initial symptoms (Sumner et al., 2010). Finally, Cognitive Bias Modification training, which redirects attentional biases from negative emotional stimuli (e.g., threat) to more neutral stimuli, has been shown to reduce symptoms of anxiety and depression (e.g., see MacLeod & Mathews, 2012 for review). Redirecting subtle perceptual cognitive biases in emotion recognition toward happy faces, and away from angry faces, can reduce anger and aggression, consistent with Dodge's model via mediating hostile attribution bias (Penton-Voak et al., 2013). It is important to note that despite these individual studies finding positive, preliminary evidence, to date none of these interventions focused on changing cognitive processes has

been established as fully evidence-based according to rigorous EST criteria (e.g., Chambless & Hollon, 1998).

In addition to cognitive processes predicting later treatment response, the opposite direction, in which interventions affect cognitive processes, has also been investigated. The majority, but not all, studies find that stimulant medications improve cognitive performance in individuals with ADHD, and more studies have found positive effects on EF and attention than memory (see Pietrzak, Mollica, Maruff, & Snyder, 2006 for review). Cognitive rehabilitation interventions, which are aimed at teaching compensatory strategies (e.g., use of lists and memory cues, dividing tasks into smaller steps, etc.), can improve functional outcomes (e.g., occupational/academic functioning) in individuals with schizophrenia (e.g., see Kluwe-Schiavon, Sanvicente-Vieira, Kristensen, & Grassi-Oliveira, 2013 for review), BD (e.g., Deckersbach et al., 2010), and ADHD (e.g., Hahn-Markowitz, Manor, & Maeir, 2011). There is less research on cognitive remediation in individuals with depression or anxiety disorders, although it is interesting that some therapies (e.g., behavioral activation; (e.g., Dimidjian, Barrera, Martell, Muñoz, & Lewinsohn, 2011) incorporate compensatory strategies (e.g., memory cues to engage in an activity, like putting walking shoes by the door). There are interesting suggestions that certain medications (e.g., Modafinil, a cognitive enhancer) have potential for improving EF in individuals with depression (e.g., improved response to antidepressant treatment with Modafinil, (Abolfazli et al., 2011), but these effects are not yet well established with cognitive control (Murrough, Iacoviello, Neumeister, Charney, & Iosifescu, 2011).

Finally, it is notable that there is less evidence in support of direct training of cognitive processes (i.e., targeting the weakness rather than compensatory strategies). In general, while task performance improves, there is little evidence that training effects generalize to real-world function or improve clinical symptoms (e.g., Rabipour & Raz, 2012). Two possible exceptions are EF training in children with ADHD,

which may decrease symptoms and increase academic function (Rabipour & Raz, 2012), and Cognitive Bias Modification, as reviewed earlier. Intriguing findings suggest that certain types of EF training may change the underlying neural mechanisms to be more efficient rather than changing strategy use only (Owens, Koster, & Derakshan, 2013), suggesting that such training might transfer more broadly to processes involving the same neural mechanisms, although this has not yet been tested.

Future translational directions for cognitive risk research in developmental psychopathology. We focus on five future directions for translation of the basic theoretical and empirical work on cognitive influence into enhancement of current therapies as well as development and testing of novel intervention approaches. First, there is considerable promise and gains to be made by applying developmentally sensitive knowledge about cognitive products and processes to the personalization of treatments and preventions (Insel, 2009), especially to answer the questions of what works for whom (i.e., moderation) and why (i.e., mediation), and for which forms of psychopathology (i.e., transdiagnostically across several disorders or specifically to one). Second, novel areas for investigation include the optimal timing of when it may be best to implement evidence-based preventions to forestall the development of psychopathology, based on accumulating knowledge of the emergence, stabilization, and consolidation of cognitive influences (as discussed earlier) and for how long interventions can reduce cognitive risks in an enduring manner (whether for relapse prevention in treatments or for onset reduction via preventions). Third, a continued emphasis on investigating mediating mechanisms that underlie demonstrated efficacy of treatments is a must. While CB-based approaches are all grounded in the underlying theory of change that underscores identification and reduction of negative cognitions, which, in turn, are hypothesized to ameliorate symptoms and problematic behaviors, this does not have to be the case (Stice et al., 2010).

Also, the fundamental processes at play may change across development. In particular,

incorporating capitalization vs. compensation approaches to therapy change (Cheavens, Strunk, Lazarus, & Goldstein, 2012) can be an important, but relatively under-investigated, individualized approach to understanding how changes in cognition may lead to improved mental health. For example, it is not clear whether various therapies are reducing cognitive risks (e.g., dysfunctional attitudes), enhancing cognitive strengths (e.g., improved EF), or both. Fourth, this review revealed a paucity of research focusing on cognitive influences as potential moderators of treatments, especially, and preventions more formally. There likely are individual differences in cognitive products and processes that moderate intervention efficacy, and this knowledge would be very helpful for adapting and enhancing personalized treatment approaches. For example, the efficacy of CBT is believed to partly depend on individuals' EF ability, yet only one pilot study has examined this theory-based moderating hypothesis: older adults with poor EF did not respond as well to CBT for anxiety (Mohlman & Gorman, 2005). Last, tailoring interventions to make them more developmentally appropriate, based on the cognitive skills, strengths and weaknesses, as informed by the basic knowledge reviewed in this chapter, may improve efficacy when CB-interventions are designed, tested, and delivered at a level commensurate with the individual's cognitive abilities (both products and processes).

Conclusions

In closing, this chapter is the first to have reviewed several of the most prominent cognitive products and processes in relation to the development of multiple common, prevalent psychopathologies. There is a considerable corpus of evidence using cross-sectional designs, predominantly with adult samples, demonstrating that both products and processes are associated with several psychopathologies, although there are gaps in coverage. Studies that have spanned different developmental periods, from childhood into adolescence and adulthood, suggest that cognitive

influences are related to psychopathologies across the lifespan, at least in the ages studied to date. In many cases, longitudinal studies indicate that cognitive factors, especially products, predict prospective elevations of psychopathological symptoms and disorder. Still, the evidence basis at this point is not sufficiently advanced to make definitive conclusions regarding which of several logical models may best account for these associations. Last, this review generally revealed a silo approach in the many studies that have examined isolated cognitive influences separately from other cognitive risks, and usually in relation to a single psychopathological outcome. We urge future research efforts to take a more interdisciplinary, integrative approach that seeks to synthesize conceptual and empirical knowledge across cognitive products and processes, with assessment and analysis of multiple forms of psychopathologies, across multiple levels of analysis using prospective longitudinal designs with carefully considered, developmentally appropriate samples. In summary, this chapter demonstrates that the field investigating cognitive risks to developmental psychopathology is a vibrant, active area that has produced numerous, impressive, and important findings that are critical for a comprehensive understanding of the development of psychopathology across the lifespan and possess clear translational import for enhancing many evidence-based, CB interventions. We are excited about the future of inquiry in cognitive influences in developmental psychopathology that can build upon the currently strong theoretical and empirical foundation and then rapidly, significantly propel knowledge forward.

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