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Exceptional Canadian Contributions to Research on Cognitive Vulnerability to Depression

David J. A. Dozois[1] and Elizabeth P. Hayden[1, 2]
[1] Department of Psychology, University of Western Ontario
[2] Brain and Mind Institute, University of Western Ontario

For more than four decades, Canadian psychologists have made significant contributions to the understanding of cognitive vulnerability to depression. This article highlights some of these exceptional contributions and the important roles Canadian scientists have played in enhancing our understanding of the cognitive products (e.g., dysfunctional attitudes), cognitive operations/processes (e.g., attention, encoding, and memory biases), and cognitive structures (i.e., cognitive organization) involved in depression. Following this review, we discuss research that has integrated cognitive vulnerability with other risk factors for depression, address some important measurement issues in cognitive vulnerability research, and highlight directions for future research.

Public Significance Statement
This article reviews some of the exceptional contributions that Canadian psychologists have made to understanding cognitive vulnerability to depression over the past four decades. We review research ranging from more surface-level negative thinking, to information processing biases (e.g., selective attention to, or enhanced recall of, negative content), to understanding deeper beliefs (e.g., the organization of information and core beliefs about self). We also provide several important suggestions for the next generation of cognitive vulnerability research, including developing more integrative models, refining the measurement of various constructs, testing causal mechanisms, and validating culturally sensitive models of cognitive vulnerability to depression.

Keywords: cognitive vulnerability, depression, information processing, schemas, schema structure

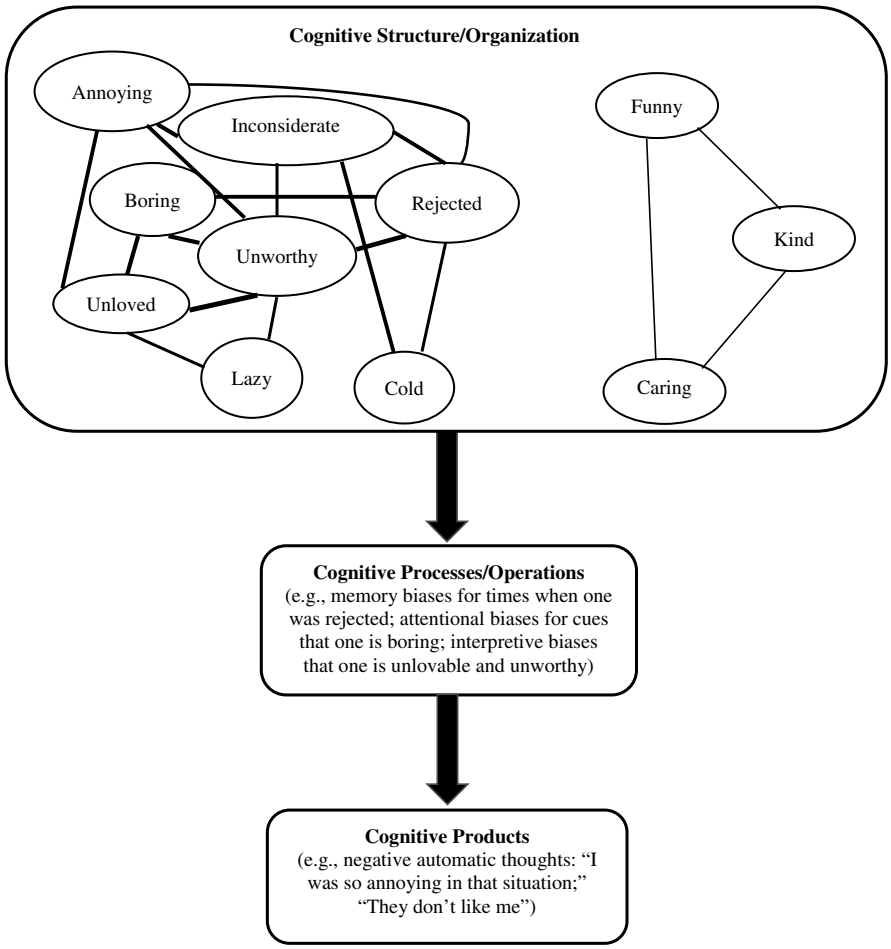
Cognitive models of vulnerability to depression share the premise that maladaptive thinking and negative appraisals of life circumstances play key explanatory roles in the development of this debilitating condition (Beck et al., 1979; Dozois & Beck, 2008) and that shifting cognition to be more evidence-based effectively disrupts the depressive process. Beck (1967), for instance, purported that depression is a result of maladaptive self-schemas, biased information processing, and negative automatic thoughts. These levels of cognition are important targets for intervention. For example, cognitive therapy works early in treatment to help individuals with depression test and modify negative automatic thoughts. In later sessions, core beliefs and schemas become an important focus. Behavioral activation been a been a critical component of cognitive behavioral therapy (CBT) for depression since its inception (Beck et al., 1979), and has garnered strong efficacy data as a stand-alone treatment for depression (see Leahy et al., in press). In CBT, these techniques are used to change reinforcement contingencies and modify a client's negative cognitions. In third-wave CBT interventions (e.g., acceptance and commitment therapy) the emphasis is on changing the relationship one has with their thoughts (e.g., by decentering, letting go, and accepting) which, in effect, also modifies cognition (see Dozois & Beck, 2012).

According to Beck's (1967) model, negative self-schemas—stable internal structures of stored information, including core beliefs about self—develop early in life, often as a result of insecure attachment experiences, childhood maltreatment, or other adverse events (e.g., Lumley & Harkness, 2009). Once activated, schemas are believed to influence how incoming information is processed and interpreted. Individuals vulnerable to depression, for example, may have underlying beliefs that they are fundamentally unlovable, incompetent, or worthless. Depression may not develop in these individuals as long as their core belief system remains latent. When life stressors (e.g., interpersonal rejection or a failure experience) activate the self-schema, however, information processing biases and negative thoughts ensue, leading to an onset of symptomatology (Beck et al., 1979; Dozois & Beck, 2008).

Many approaches have been used to assess cognitive vulnerability across levels of the cognitive taxonomy (Beck & Dozois, 2014; Ingram et al., 1998). By "taxonomy," we mean that the cognitive system related to vulnerability to depression is comprised of a range of cognitive components spanning surface-level thoughts to deeper structures (see Figure 1): cognitive products (accessible thoughts and beliefs), cognitive operations/processes (that include such

David J. A. Dozois https://orcid.org/0000-0002-0857-0679
Correspondence concerning this article should be addressed to David J. A. Dozois, Department of Psychology, University of Western Ontario, Westminster Hall, Rm. 313E, London, ON N6A 3K7, Canada. Email: ddozois@uwo.ca

Figure 1
The Cognitive Taxonomy



Note. Thicker lines represent stronger associations among core beliefs.

variables as attention, encoding, retrieval, and interpretation), and cognitive structure (the internal representation and organization of information in memory which, together with core beliefs, comprise the schema; see Dozois & Beck, 2008; Ingram et al., 1998). The cognitive taxonomy has long been recognized as an important model for understanding the various levels of cognition associated with depression (Ingram et al., 1998).

For more than 40 years, Canadian psychologists have made significant contributions to the understanding of cognitive vulnerability to depression at each level of the cognitive taxonomy. This article highlights some of these exceptional contributions and the important roles they have played in the research literature. Space restrictions limit our ability to provide a comprehensive review; we have chosen instead to highlight some key examples in the areas of cognitive products, operations/processes, and structures. Following this review, we discuss research that has integrated cognitive vulnerability with other risk factors for depression, address important issues related to the measurement of cognitive vulnerability, and highlight directions for future research.

Cognitive Products

Self-report measures have been the primary means by which researchers have evaluated the products of cognitive vulnerability to depression. Throughout the last four decades, Canadian psychologists have contributed importantly to this research literature, demonstrating that depression is associated with an increase in negative or maladaptive thinking and deficiencies in positive cognition (see Clark et al., 1999 and Ingram et al., 1998, for comprehensive reviews).

The Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978)—a widely used index of negative thinking in depression—has been studied extensively. The DAS was designed to measure the "silent assumptions," dysfunctional cognitions, and maladaptive beliefs characteristic of depression. Numerous studies from Canadian researchers have examined the psychometric properties and predictive utility of the DAS. In general, this instrument demonstrates good reliability (Dobson & Breiter, 1983), correlates with dysphoric mood (Scher et al., 2005), appears to distinguish reliably between depressed and nondepressed groups (e.g., Dobson &

Shaw, 1987), is associated with future depressive symptoms (e.g., Zuroff et al., 1990), and typically yields a stable two-factor structure (relating to affiliative and achievement needs; Cane et al., 1986, but see Moore et al., 2014).

Many other cognitive product indices have also been developed or tested by various Canadian research groups (e.g., Beshai et al., 2016; Covin et al., 2011; Dozois et al., 2003; McBride et al., 2006). Covin et al. (2011), for example, developed the Cognitive Distortions Scale (CDS) to assess common thinking biases (e.g., catastrophizing, all-or-nothing thinking) in interpersonal and achievement domains. In the CDS, after reading a definition of a cognitive distortion and a vignette example, respondents indicate the frequency with which they use each type of thinking. This measure has good psychometric properties in undergraduate (Covin et al., 2011) and clinical (Özdel et al., 2014) samples.

Conceptual Models Related to Cognitive Products

A number of Canadian researchers have also tested various conceptual models (e.g., the congruency hypothesis) related to cognitive products in depression (e.g., Bieling & Alden, 1998; Clark et al., 1992; Dunkley et al., 1997; Frewen & Dozois, 2006; Segal et al., 1992; Zuroff & Mongrain, 1987). In his congruency hypothesis, Beck (1983) argued that two personality dimensions (sociotropy and autonomy) may mediate depression. Sociotropy pertains to a set of invested beliefs and goals that emphasize the establishment and maintenance of interpersonal attachments. Individuals who are sociotropic believe that attaining acceptance, guidance, understanding, intimacy, and support, are critical for their self-worth. These individuals also tend to fear rejection, disapproval, neglect, and other adverse interpersonal situations because of the perceived threat to their self-construal. Autonomy refers to a person's self-worth investment in increasing and maintaining independence, individuality, mobility, and achievement. Situations such as failure, constriction of goals, and immobility are viewed as threats to self-worth.

Rather than a main effect model, in which a stressful event causes depression, the premise of Beck's interactional (diathesis-stress) model is that depressive symptoms are more likely to follow stressful life events when negative events match an individual's personal motivational vulnerability (congruency hypothesis). Thus, sociotropic individuals were predicted to exhibit more depressive symptoms in relation to negative interpersonal events (e.g., rejection); autonomous individuals, on the other hand, were purported to be more vulnerable to achievement related events (e.g., failure). The research literature has generally supported the congruency hypothesis (e.g., Clark et al., 1999; Segal et al., 1992; Zuroff et al., 1990; Zuroff & Mongrain, 1987).

Various Canadian researchers have also tested whether negative thinking in depression is content-specific. According to the content-specificity hypothesis, each emotional state and psychological disorder has a specific cognitive profile (e.g., sadness involves appraisals of loss or failure whereas anxiety stems from evaluations of threat or danger). Research in experimental psychopathology has generally supported the content-specificity hypothesis (see Clark et al., 1999, for review). Westra (now at York University) and Kuiper (Western University; 1997), for instance, instructed undergraduate students to make self-descriptive ratings on several adjectives sampled from the depression, anxiety, eating disorders, and personality literature. Dysphoria was uniquely related to adjectives pertaining to loss, failure, and hopelessness, whereas the themes in anxiety centered on threat and stigmatization. The idea that anxiety and depression are related to similar information processing mechanisms has also been found, although less consistently. Anxiety and depression both involve similar cognitive processes, but anxiety seems to relate more to automatic processing and attention biases whereas depression is more strongly associated with elaborative processing and memory biases (Clark et al., 1990, 1999).

Although the data on cognitive products provided consistent evidence that depression is associated with an increase in negative thinking, researchers found that these effects generally lasted only during the depressive episode itself. That is, they appeared to be concomitants (or episode markers) rather than causes (vulnerability markers) of depression (Barnett & Gotlib, 1988; Kuiper et al., 1985). Based on the mood-congruency hypothesis (Ingram et al., 1998; Segal, 1988), research demonstrated that there may indeed be stable cognitive vulnerability markers for depression, but that priming methodologies are necessary to activate and assess them.

The rationale for priming is that the products (and processes) of self-schemas are latent until activated (Segal, 1988). Empirical data have shown that individuals who have remitted from an episode of depression demonstrate greater dysfunctional attitudes (and information processing biases) than do controls after they are primed (e.g., by negative moods) prior to cognitive assessment (Ingram et al., 1998; Segal & Ingram, 1994; Segal & Swallow, 1994). Dr. Zindel Segal (University of Toronto) and his colleagues tested whether schema activation vis-à-vis priming is related to vulnerability to relapse in depression. Segal et al. (1999), for example, compared patients who were successfully treated with either cognitive therapy or antidepressant medication. Following treatment, participants were administered the DAS, given a negative mood prime to induce a dysphoric state, and administered a parallel form of the DAS. Patients who were treated pharmacologically showed elevated DAS scores following the mood manipulation. This increase in negative thinking was not present in individuals who received cognitive therapy. Segal et al. (2006) also found that such cognitive reactivity was predictive of relapse 18 months later.

In addition to contributing importantly to our understanding of the descriptive hypotheses of the cognitive model of depression (Clark et al., 1999; Dunkley et al., 2019; Ingram et al., 1998), and the idea that cognitive reactivity may predict depressive relapse (Segal et al., 2006), research on cognitive products also shifted attention toward understanding depression more within a social context (e.g., Rector et al., 1998; Segal & Dobson, 1992), the importance of assessing the beliefs that individuals with depression have about themselves as social beings (e.g., Dozois, 2021; Dozois & Dobson, 2001b) and the importance of testing interpersonal beliefs in depression within the context of relationships (e.g., Wilde & Dozois, 2019).

Cognitive Operations/Processes

Self-Referent Encoding

Craik and Lockhart (1972), from the University of Toronto, originally proposed a depth-of-processing model to describe the various levels of memory-based processing. The basic model purported that incoming stimuli are initially processed via shallow sensory evaluation followed by deeper, more complex semantic

analyses. According to this model, highly familiar and salient content is processed at a deeper level (thus leaving a stronger memory trace) than is less meaningful material.

Early supportive evidence of this theory was derived from experiments involving a depth-of-processing incidental recall paradigm (Craik & Tulving, 1975; Kuiper & Rogers, 1979; Rogers, 1981; Rogers et al., 1977). The rationale behind this methodology is that information processing may be assessed at varying levels. University of Calgary professor Rogers et al. (1977), for example, documented that information processed in terms of its self-reference (e.g., "Describes you?") produced superior recall than did information that was evaluated according to its semantic (e.g., "Means the same as _____?"), structural (e.g., "Small letters?"), or phonemic (e.g., "Rhythmic?") properties. These results indicated that self-referent processing promotes a deeper level of encoding and yields a stronger and more elaborate memory trace than does information that is not self-referent (an effect that is likely due to both cognitive organization and the amount of elaboration self-referent information receives during encoding; see Symons & Johnson, 1997).

Soon after the discovery of the self-reference effect, studies began to materialize which adapted this conceptualization and methodology to the area of depression. University of Calgary researcher Henry Davis (1979a) first applied the depth of processing task to depression and although he found the expected self-reference effect in nondepressed controls, individuals with depression did not demonstrate superior recall for self-referent material. In subsequent studies, Davis (1979b) and Davis and Unruh (1981) showed that depressive self-referential processing was found only in individuals who experienced a longer duration of depression. These findings were the impetus for Davis' developmental approach to the self-schema. However, Davis' research generated criticism because the adjective content he used consisted mainly of positive stimuli (rather than also focusing on negative self-referent information) and was therefore not appropriate for testing self-schematic processing in clinical depression (Derry & Kuiper, 1981; Kuiper & Derry, 1981).

Building on Davis' examination of the self-reference effect in depression, Western University psychologists Derry and Kuiper (1981) used both positive and negative adjective content as depth-of-processing stimuli in the Self-Referent Encoding Task (SRET). After rating each adjective in terms of its degree of self-reference, participants were administered an incidental recall task. These researchers found that individuals with clinical depression recalled significantly more self-referent depressed content than nondepressed content; conversely, individuals without depression and psychiatric controls displayed the opposite pattern (i.e., they recalled more nondepressed than depressed self-referent adjectives).

Numerous studies have replicated and extended the initial findings of the self-reference effect in depression using a number of dependent variables (e.g., endorsement ratings, recall, recognition, reaction time, consistency ratings, and drift rate), across a number of samples (e.g., Dainer-Best et al., 2018; Dobson & Shaw, 1987; Dozois & Dobson, 2001b; Kuiper et al., 1985; Moretti et al., 1996). The data generally suggest that individuals with depression endorse more negative adjectives as self-referent, recall more negative self-referential material, and, in some instances, demonstrate more efficient processing (as evidenced by faster reaction times) of negative compared to positive content. Conversely, individuals without depression endorse, recall, and more efficiently process positive than negative information about themselves.

The progression of Canadian science from Craik and Lockhart's (1972) ground-breaking theory of how encoding impacts memory, to Rogers et al.'s (1977) demonstration that self-reference is more deeply encoded than other types of information, to Davis' (1979a) application to depression, to Kuiper's (Derry & Kuiper, 1981; Kuiper & Derry, 1981; Kuiper et al., 1985) SRET is remarkable. It is a progression that speaks volumes to the importance of keeping an open mind about science and the cross fertilization of ideas (in this instance of cognitive and clinical science). The SRET continues to be used regularly in self-concept research and in studies that assess cognitive vulnerability to depression, and its scope arguably continues to expand. As Bentley et al. (2017) contend, the SRET "has stood the test of time, and remains as relevant today as when it first emerged in the 1970s" (p. 1). Hundreds of studies have been conducted on the self-referent effect demonstrating reliably that self-referent information leaves a deep, robust, and reliable memory trace (Symons & Johnson, 1997).

Recent studies have also evaluated the psychometric properties of the SRET. Although some specific computations of self-reference (e.g., drift rate) may yield more reliable results than others (Beevers et al., 2019; Dainer-Best et al., 2018), research demonstrates that the SRET shows good test-retest reliability, internal consistency, and sensitivity to depression (Auerbach et al., 2016; Bentley et al., 2017; Dainer-Best et al., 2018; Goldstein et al., 2015; Phillips et al., 2010). Longitudinal studies also suggest that the SRET prospectively predicts increases in depressive symptomatology and depression recurrence (Connolly et al., 2016; Goldstein et al., 2015; Hayden et al., 2013; LeMoult et al., 2017). Goldstein et al. (2015), for example, found that depressive symptoms at ages six and nine years were related to higher negative, and lower positive, processing on the SRET. Lower positive processing, however, uniquely predicted increased symptoms at age nine. Thus, lower positive processing rather than higher negative processing may represent a risk factor for future depressive symptoms. Consistent with this idea, Hayden et al. (2013; Western University) found that positive (but not negative) SRET processing at age seven was associated with depressive symptomatology at ages eight and nine. In a 3-year longitudinal study, LeMoult (now at the University of British Columbia), Kircanski et al. (2017) found that self-referent encoding biases contributed unique variance in the prediction of depression recurrence.

The impact of the SRET to the understanding of memory processes in depression is unequivocal (Dainer-Best et al., 2018). Self-referent memory biases represent robust and stable negative cognitive biases in depression and are an important risk factor for the development and maintenance of depression (Gotlib & Joormann, 2010) and its recurrence (LeMoult et al., 2017). Some researchers (e.g., Duyser et al., 2020) also contend that negative memory biases characteristic of depressive processing may be an important transdiagnostic factor in psychopathology more generally.

Emotional Stroop Effect

Another important Canadian contribution to understanding cognitive processes in depression was the modification of the Stroop task to assess attentional biases. In the original Stroop procedure (Stroop, 1935), participants were asked to name the color of ink in which different color words are printed. Dalrymple-Alford and Budayer (1966) later revised this methodology to include both

congruent and incongruent conditions. Longer reaction times are typically shown when the word and color do not match (e.g., the letters r-e-d printed in the color green) than when they are congruent (e.g., the letters r-e-d printed in the color red). Individuals also tend to demonstrate longer latencies when naming the ink color of color words (e.g., red, yellow) than noncolor words (e.g., chair, drawer). Researchers attributed these longer reaction times to attentional interference caused by the differential strength of the competing pathways being processed (see MacLeod, 1991). Although the precise cognitive mechanism(s) underlying the Stroop effect was a matter of some dispute within cognitive psychology, the classic Stroop interference effect has been replicated in numerous studies and shown to be reliable (see MacLeod, 1991).

Drs. Ian Gotlib (who was at Western University at the time) and Doug McCann (1984; York University) first employed a modified Stroop methodology to assess schematic processing in depression. These authors reasoned that if negative schemas are characteristic of the information-processing of individuals with depression, then they should be intrinsically "primed" or attentive toward negative content (e.g., the presentation of the word "worthless"). The modified Stroop task involved naming the colors of depressed-, neutral-, and manic-content words that were presented tachistiscopically (Gotlib & McCann, 1984). The main assumption underlying this methodology is that response latency is indicative of the amount of interference a word produces (see Segal & Swallow, 1994). For example, more effort would be required to suppress the content (and name the color) of highly accessible or salient stimuli (i.e., schema-congruent words) than of less relevant stimuli, thereby lengthening reaction times. As expected, individuals with dysphoria took longer to name the colors of the depressed-content words than those of the non-depressed content. Conversely, individuals without depression did not demonstrate this differential response pattern.

Many studies have since replicated and refined the Emotional Stroop methodology for use in depression, using both clinical and analogue samples. The empirical data support the idea that individuals with depression exhibit longer response latencies to negative content than do individuals who are not depressed and that this task differs across severity of depression (see Epp et al., 2012, for a meta-analytic review). The extent to which the Emotional Stroop task reflects the operations of stable vulnerability factors for depression is, however, dubious (Evraire et al., 2015). More refined methodologies have since been developed to assess attentional biases in depression and other disorders (e.g., the dot-probe task, eye-tracking technology; see Epp et al., 2012; Evraire et al., 2015). This research supports the notion that there are attentional biases in depression; however, the difficulties seem to be related more to trouble disengaging from negative stimuli than by biases in initial orienting responses (see LeMoult & Gotlib, 2019, for review). Nonetheless, the adaptation of methods from basic cognitive to clinical science, that began with Canadian innovations (e.g, Gotlib & McCann, 1984; Segal et al., 1988; Segal & Vella, 1990), continues to advance our understanding of the various cognitive processes involved in depression.

Cognitive Structure/Organizational Coherence

Despite recommendations made by Segal (1988) and others about the importance of investigating the "clustering or interconnectedness among mental operations" in testing models of cognitive vulnerability to depression (p. 157), most research has focused on cognitive products and processes in depression. Far less is known about how these cognitive elements are organized, hierarchically structured, activated, or deactivated, although these processes may be key to understanding cognitive vulnerability to depression (Dozois & Beck, 2008). Below, we review the small body of research relevant to cognitive organization.

Prime-Target Relatedness

Segal and his colleagues (Segal, 1988; Segal & Gemar, 1997; Segal et al., 1995; Segal & Vella, 1990) conducted an ingenious series of studies using a variation of the Emotional Stroop task to infer interconnectedness of the self-schema in depression. After creating lists of idiographically derived self-descriptive traits for each participant, these experimenters administered the modified Stroop task. Participants read the prime word (which varied in terms of its relatedness to the target adjective), named the color of the target, and then recalled the prime. Individuals who were "schematic" for a particular content domain, were expected to take longer to color-name target words when the prime and target were related than when they were not. Consistent with this prediction, individuals with depression displayed longer reaction times for color-naming negative target words when the primes were self-descriptive than when they were not. This prime-target relatedness effect was not found for nonpsychiatric controls, although Segal and Vella (1990) also found the relatedness effect for extremely nondescriptive words in both groups of individuals with depression and controls. This latter finding suggests that individuals with depression may have an organized store of both positive and negative information. Segal and Gemar (1997) used a similar methodology (using interpersonal phrases instead of prime words) to investigate cognitive organizational changes following cognitive-behavioral therapy (CBT). Patients who had improved after treatment demonstrated less of a prime-target relatedness interference effect for negative adjectives. Individuals who remained depressed, on the other hand, continued to display high levels of cognitive interference for negative self-descriptive material. No relationship was found between post-treatment status and positive interference scores.

While these studies suggest that individuals with depression have an organized structure of negative self-relevant information, they do not rule out the possibility that prime-target relatedness may be due to activation (rather than organization) of self-schematic processes. It is plausible, for instance, that self-descriptive negative primes competed for attention in those individuals with depression more so than did other primes, thereby slowing reaction times for naming the colors of target words. Given that there were no psychotherapy or pharmacotherapy control groups, it was also not possible to conclude that changes in Stroop latencies were a direct result of CBT rather than a function of symptomatic improvement. Nonetheless, these studies represented an important step toward assessing the structural properties of schemata in depression.

Psychological Distance

Drs. David Dozois (Western University) and Keith Dobson (University of Calgary) developed the Psychological Distance Scaling Task (PDST; Dozois & Dobson, 2001a, 2001b) as a way to more directly examine cognitive organization or structure in

depression. On a computer screen or digital device, respondents place self-referential adjectives on two-dimensional space based on self-descriptiveness and valence. The distance among the adjectives is then computed for positive and negative content, with the assumption that smaller distances among adjectives reflect greater interconnectedness or consolidation of self-referent content and larger distances among adjectives indicate less interconnectedness or consolidation (see Dozois, 2021; see Figure 1 for a simplistic schematic of this idea).

Individuals with depression show well-interconnected negative content and loosely clustered positive content, a finding has been demonstrated in adults (e.g., Dozois & Dobson, 2001b; Dozois & Frewen, 2006), youths (Dozois et al., 2012; Lumley et al., 2012), and individuals with past depression (e.g., Dozois & Dobson, 2003). Seeds and Dozois (2010) also found that the interaction of cognitive organization and life stress predicted depressive symptoms at one-year follow-up after controlling statistically for baseline symptoms. In addition, although it can be modified via effective evidence-based treatments (Dozois et al., 2009; Quilty et al., 2014) cognitive organization appears to predict depressive symptoms beyond negative schema content (Lumley et al., 2012) and persists despite symptom improvement (Dozois & Dobson, 2001a; Dozois, 2007). Dozois and Dobson (2001a) administered the PDST and information processing tasks (Emotional Stroop, SRET) to a sample of females with depression. Participants were retested at 6-month follow-up when half of the sample continued to experience clinically significant depression and the other half remitted. Negative information processing was found only during the acute phase of depression and improved significantly once depression improved (suggesting that this variable operates more as state than trait marker). In contrast, negative cognitive organization remained stable across time in those individuals who no longer met diagnostic criteria for major depression. This finding was replicated in a subsequent study which also found that the stability of negative cognitive organization was specific to interpersonal self-referent content (Dozois, 2007). These results suggest that negative interpersonal self-structures may be vulnerability factors for depression and its recurrence (see Dozois, 2021, for a review). Dr. Margaret Lumley (University of Guelph) and her colleagues have expanded this research to focus on positive schema structures and demonstrated that this construct may provide unique variance to the prediction of depression (Keyfitz et al., 2013; Lumley et al., 2012).

Future Directions and Conclusions

As is clear from our review, Canadian scientists who study cognitive mechanisms in depression have made major contributions to the field. However, there is, of course, much work left to be done concerning the cognitive bases of depression vulnerability. In this section, we provide an overview of several broad domains we see as worthy priorities for future research on cognitive vulnerability to depression. While these priorities apply broadly to this research domain (i.e., their importance is not constrained to Canadian research), many Canadian depression researchers have ongoing, relevant programs of work that are well positioned to make valuable contributions; we look forward to seeing this literature develop and mature in the years to come.

Integrative Models of Cognitive Vulnerability

Research on cognitive vulnerability has a rich tradition of the use of rigorous, well-controlled, experimental (or quasi-experimental) designs that maximize internal validity. However, the well-established etiological heterogeneity of depression calls for complementary tests of complex models that are oftentimes challenging to model in a laboratory setting, necessitating naturalistic studies of depression that emphasize external validity. Indeed, studies examining associations between a single, putatively etiologically relevant factor (e.g., an index of cognitive vulnerability) measured from a single vantage point (e.g., self-report) and depression are increasingly supplanted in the literature by multidisciplinary studies that integrate biological, cognitive, and environmental/contextual indices of risk. However, the field is still grappling with the evidence that the broader domain of psychological science may be failing to produce robust findings (Open Science Collaboration, 2015; Sharpe & Goghari, 2020). While the failure to attend closely to considerations of measurement is an underappreciated contributing factor to the replication crisis (Pashler & Wagenmakers, 2012; a point addressed later in this section), it is also the case that increasingly complex models may have a lower likelihood of replicability (Sanbonmatsu et al., 2021), both concerns that psychological scientists will need to address in developing increasingly complex models of depression's etiology.

As an example, in the early 2000s, reports of an interaction between the serotonin transporter promoter polymorphism (5-HTTLPR) and life stress in predicting depression appeared (Caspi et al., 2003). The field greeted initial tests of gene-environment interaction (GXE) as novel instantiations of diathesis-stress models of depression with tremendous excitement; this literature was subsequently extended by findings of associations between the 5-HTTLPR and markers of cognitive vulnerability to depression (e.g., Hayden et al., 2008) as putative endophenotypes, or genetically mediated vulnerabilities to depression. These approaches (i.e., tests of GXE and genetic association studies of indices of cognitive vulnerability) are consistent with the well-established literature showing that both heritable and environmental factors contribute to depression and that there is a heritable basis for markers of cognitive vulnerability (Lau et al., 2006). However, concerns about the replicability of studies of GXE followed shortly thereafter, with critics noting concerns about statistical power to detect the likely small effects of candidate genes; indeed, even seemingly inconsequential factors like the scaling of indices of stress can increase Type I error (see Dick et al., 2015, for a more extended discussion of these and other issues). While depression researchers from Canada and elsewhere (e.g., Harkness et al., 2015) have provided compelling responses to these critiques, as well as suggestions on how to strengthen such study designs, the question of how to best model the genetic and environmental etiological influences on depression vulnerability remains open and will require multidisciplinary, collaborative efforts to provide adequate answers.

Testing Causal Mechanisms and Unique Contributions of Cognitive Vulnerability

Along similar lines, the issues that surround tests of models of cognitive vulnerability as mediators of depression risk is a less well-known point of contention. The relevant literature is replete with

studies seeking to link distal etiological factors to depression via cognitive mechanisms. However, Bullock et al. (2010) provide an incisive analysis of how tests of mediation are highly subject to bias, especially in the context of research that is not truly experimental, because any unmeasured factor that influences the hypothesized mediator and outcome similarly will serve to inflate estimates of the effect of the mediator (i.e., omitted-variables bias). More specific to the current discussion, unmeasured influences that serve to increase both cognitive vulnerability and depression will artificially inflate estimates of cognitive vulnerability as a mediator of depression. This concern cannot be readily dismissed given that it is not challenging to generate a list of influences that are potentially common to both cognitive vulnerability and depression.

Even in the context of true experiments, tests of mediation are still especially vulnerable to threats to inference for several reasons, including cases in which an experimental manipulation influences other mediators, alongside the target of the manipulation. To give an example relevant to this literature, in studies in which attentional bias to threat is experimentally induced as a mediator, any other cognitive systems (e.g., memory) that are similarly influenced will bias estimates of mediation. Indeed, Bullock and colleagues specifically note the challenges this issue poses in the context of the study of cognitive mediators. Given that most psychopathologists would agree that many of our independent variables of interest influence outcomes through multiple causal pathways (e.g., maternal depression likely disrupts multiple systems implicated in offspring depression risk), it is challenging, if not impossible, to include all potential pathways in a single study.

Tests of the incremental validity of cognitive vulnerability, and the use of covariates generally speaking, require thoughtful consideration of issues that are underappreciated but truly important (Westfall & Yarkoni, 2016). Oftentimes, especially in non-experimental research, depression and other researchers are interested in the predictive utility of a variable for an outcome after controlling for the influence of other causal variables, or in how a putatively causal variable influences an outcome after having adjusted for nuisance variables that are not of substantive interest. In studies of cognition and depression, for example, it is common practice to test whether an index of cognitive vulnerability influences depression "above and beyond" the influence of other relevant variables (e.g., depressive symptoms measured concurrent to the cognitive marker). However, given that measurement error of predictor variables contributes to the proportion of variance accounted for by the predictor, "noise" can readily be mistaken for veridical predictive value (see Westfall & Yarkoni, 2016) for a more extensive discussion of the problem of *residual confounding*). Westfall and Yarkoni call for a greater use of structural equation modeling (SEM)-based statistical approaches to address this particular issue, a useful suggestion with which we agree. Unfortunately, some study designs and methods are more amenable to SEM (e.g., questionnaire measures) than are others. Thus, there is no easy solution to this problem and other important concerns surrounding the use of covariates (e.g., Miller & Chapman, 2001) are underappreciated.

Measuring Cognitive Vulnerability

Earlier, we alluded to the role of measurement in the so-called replicability crisis. As noted by Curran and Willoughby (2003), our capacity to conduct valid tests of theory rests upon the rigor of our statistical models and measures. While these authors were speaking in the context of developmental theory, this statement applies broadly to psychological science. Indeed, many of the aforementioned obstacles to the development of robust models of cognitive vulnerability and depression are potentially addressed (or at least, better understood) by greater attention to measurement considerations. While the field tends to prioritize tests of causal mechanisms in the context of publication and research funding, the potential of such tests is oftentimes limited by suboptimal measurement approaches. Even measures in the field that are well established (and used by investigators for their very longevity) are oftentimes long overdue for a fuller investigation of their psychometric properties with larger samples, using contemporary methods of data analysis. Findings that indices of cognitive vulnerability drawn from multiple levels of analysis (e.g., behavioral tasks, self-reports) show low convergence is often interpreted as evidence that nonredundant cognitive systems drive cognitive vulnerability; however, it is also possible that certain indices of depressive cognition, even ones widely used in the field, are psychometrically flawed or at least require a consideration of best practices in their usage (e.g., the dot-probe; Meissel et al., 2021). The etiological complexity of depression notwithstanding, we see methodologically focused research that develops new, improved indices of depressogenic cognition, in conjunction with stringent tests of the reliability and validity of widely used indices of cognitive vulnerability in large samples of depressed individuals, who are obviously at clear risk for the disorder, as crucial to advancing the field. The value of tests of causal models of cognitive vulnerability to depression is constrained by the construct validity of indices of risk.

Relatedly, other important research questions in the field are similarly positioned within the domain of measurement. For example, the field currently lacks measures that will allow us to study the developmental psychopathology of cognitive vulnerability to depression, despite the relevance of this topic for early intervention and preventative efforts. Neither heterotypic nor homotypic continuity in cognitive vulnerability can be understood in the absence of measures that validly represent the same construct over time (i.e., measurement invariance; see Curran & Willoughby for a more detailed definition and explanation of this issue). Going forward, scientists should dedicate intensive resources to the development of indices of cognitive vulnerability that permit the study of its initial development and change over time.

Similarly, the development and validation of culturally sensitive models of cognitive vulnerability to depression, another key goal for the field, necessitates an investigation of the measurement properties of widely used indices of cognitive vulnerability that have been validated in Whites but not in populations historically underserved by psychological scientists. Research such as this is but one small aspect of a broader diversification of scientific inquiry that is urgently needed in the study of psychopathology writ large, a topic beyond the scope of the current paper and worthy of its own dedicated review. We assert that it is time for the field to move beyond treating ethnicity as a covariate and to instead thoroughly explore substantive questions surrounding the role of ethnicity, culture, historical oppression, and other issues relevant to intersectionality in validating and refining models of cognitive vulnerability to depression. Integrating diversity issues and basic science on cognitive vulnerability to depression will also have important implications for enhancing the effectiveness of cognitive approaches to treating depression in non-majority populations (Metzger et al., 2021), a related and critical issue in the field.

We have highlighted what we see as critical future directions in the field as well as some of the challenges that scientists will face as they strive to advance what is known about cognitive vulnerability. We see a consideration of measurement issues as a relatively underappreciated concern in the field and have highlighted the value of studies of measurement invariance and the use of SEM to improve our measures and study designs. However, there is no single approach or methodology that can address all these challenges; indeed, it would be highly counterproductive to privilege one approach over others, as science progresses through the aggregation of knowledge gleaned from multiple vantage points and diverse perspectives. Greater collaboration across laboratories and multidisciplinary approaches will therefore be critical, moving forward.

Final Comments

We have reviewed the rich tradition of contributions made by Canadian scientists to the study of cognitive vulnerability to depression. It is a challenging yet exciting time to study cognitive vulnerability and we hope that this overview provides useful guidance to scientists interested in enhancing the rigor of our methods and theories. We very much look forward to the exciting contributions Canadian scholars will make to our understanding of cognitive vulnerability to depression.

Résumé

Depuis plus de quatre décennies, les psychologues du Canada font d'importantes contributions à l'amélioration de la compréhension de la vulnérabilité cognitive à l'égard de la dépression. Cet article met en relief certaines de leurs exceptionnelles contributions ainsi que le rôle important qu'ont joué les scientifiques canadiens en vue d'approfondir notre compréhension des produits cognitifs (par ex., les attitudes dysfonctionnelles), des activités et des opérations cognitives (par ex., l'attention, l'encodage, les biais de mémoire), et des structures cognitives (c.-à-d. l'organisation cognitive) qu'implique la dépression. Après cette revue, les auteurs présentent les recherches qui ont incorporé la vulnérabilité cognitive et d'autres facteurs de risque de la dépression, discutent de certaines des importantes questions relatives aux mesures dans le domaine de la recherche sur la vulnérabilité cognitive et suggèrent des orientations pour d'éventuelles recherches.

Mots-clés : vulnérabilité cognitive, dépression, traitement de l'information, schémas, structure de schéma

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