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Translating basic psychopathology research to preventive interventions: A tribute to John Abela

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Abstract

This paper highlights how the many important contributions of John R. Z. Abela's research program can inform the development and implementation of interventions for preventing depression in youth. Abela provided evidence of multiple vulnerabilities to depression including cognitive (e.g., inferential style, dysfunctional attitudes, ruminative response style), interpersonal (e.g., reassurance seeking, attachment, dependency), personality (e.g., neuroticism, self-criticism), and contextual (e.g., stress, parental depression). He introduced important methodological advances to the study of the hopelessness model of depression, especially in children, including the "weakest link" approach, cognitive priming, and idiographic measurement of stress. We briefly review what is currently known about the prevention of depression regarding intervention targets, content, outcomes, effect sizes, moderators, mediators, specificity, and durability. Next, we summarize several of Abela's contributions that are most relevant to the prevention of depression. We describe the implications of Abela's work for the development, implementation, and testing of programs aimed at preventing depression, and discuss important challenges such as the transfer of training to and the personalization of interventions so as to capitalize on individuals' strengths versus compensate for their weaknesses.

During his far too few years, John R. Z. Abela, Ph.D. had an enormous impact on clinical science in general, and the field of developmental psychopathology in particular. His endless energy, enthusiasm, and intellect facilitated his becoming an extremely prolific scholar. Abela's research had both breadth and depth; it was truly innovative, integrative, conceptually sophisticated, and methodologically rigorous. The accomplishments of John Abela are extensive and impressive, and include contributions to substantive theory, methodology, measurement development, and cross-cultural perspectives.

The purpose of the current paper is to highlight how Abela's extensive work can inform and enhance efforts at preventing depression in children and adolescents, and to suggest ways to carry this knowledge forward into the future. First, we briefly review the state of the literature regarding the prevention of depression in youth. We then describe several important findings from Abela's work that have not yet been explicitly applied to prevention, and finally, we recommend ways in which such translational work can be implemented.

Prevention of Depression in Children and Adolescents: What do we know?

Prevention programs can be classified according to the populations to whom the interventions are directed (Mrazek & Haggerty, 1994): *universal* samples are comprised of all members of a population regardless of risk; *selective* samples have a known risk factor

for the development of the disorder (e.g., offspring of depressed parents); *indicated* samples include participants with early symptoms of the disorder (e.g., sub-clinical depressive symptoms). In the past decade, good progress has been made in demonstrating the efficacy of several interventions aimed at preventing depression in these different types of samples of children and adolescents (for extensive reviews see: Brunwasser, Gillham, & Kim, 2009; Horowitz & Garber, 2006; Jané-Llopis, Hosman, Jenkins, & Anderson, 2003; Merry, McDowell, Hetrick, Bir, & Muller, 2004; Merry & Spence, 2007; Munoz, Le, Clarke, & Jaycox, 2002; Stice, Shaw, Bohon, Marti, & Rohde, 2009; Sutton, 2007). Based on this literature, below we briefly outline the current state of knowledge with regard to the prevention of depression in youth.

Targets of Depression Prevention Programs

Various types of at-risk samples have been targeted in randomized clinical trials of depression prevention programs. Such selective samples have included the following risk factors: parental depression (Beardslee et al., 1997; Clarke et al., 2001; Compas et al., 2009; Garber et al., 2009), parents with alcohol problems (Roosa et al., 1989), death of a parent (Sandler et al., 2003), parental divorce (Gwynn & Brantley, 1987; Wolchik et al., 2002), family conflict (e.g., Jaycox, Reivich, Gillham, & Seligman, 1994; Yu & Seligman, 2002), low income, minority youth (Cardemil et al. 2002, 2007) and a negative attributional style (Seligman et al., 1999). Several depression prevention trials also have been conducted with indicated samples of individuals with subsyndromal levels of depressive symptoms (e.g., Clarke et al., 2001; Gillham, Hamilton, Freres, Patton, & Gallop, 2006; Roberts, Kane, Thomson, Bishop, & Hart, 2003; Sheffield et al., 2006; Stice et al., 2006; Young et al., 2006). Effect sizes for studies using universal samples have been lower as compared to those with targeted (i.e., selective or indicated) samples (Horowitz & Garber, 2006; Merry et al., 2004; Stice et al., 2009).

Program Content

Most depression prevention programs have been adapted from evidence-based therapies for depression in adults such as cognitive-behavioral therapy (CBT) and interpersonal psychotherapy (IPT) and have been extended downward to children and adolescents. These programs have focused on cognitive restructuring and problem-solving (e.g., Gillham, Hamilton et al., 2006, Clarke et al., 2001; Pössel, Horn, Groen, & Hautzinger, 2004), coping (e.g., Compas et al., 2009; Sandler et al., 2003), social competence (e.g., Jaycox et al., 1994; Pössel et al., 2004), interpersonal relationships (Young, Mufson, & Davies, 2006), and parenting (e.g., Compas et al., 2009; Sandler et al., 2003). Other potential substantive topics that have been linked with depression in youth (e.g., Abela, Aydin, & Auerbach, 2007; Abela, Hankin, Haigh, Vinokuroff, Trayhern, & Adams, 2005; Auerbach, Abela, & Ho, 2007; Hankin, Kassel, & Abela, 2005), but have not yet been a primary focus of depression prevention programs include emotion regulation, personality (e.g., neuroticism, reassurance seeking, response style), and attachment.

Outcomes

Most depression prevention studies have examined whether the intervention reduces depressive symptoms (i.e., early treatment) or keeps symptoms from increasing (i.e., prevention; Brunwasser et al., 2009; Horowitz & Garber, 2006). A smaller number of studies have shown that diagnosed depressive disorders can be prevented (e.g., Clarke et al., 2001; Compas et al., 2009; Garber et al., 2009). Additionally, few studies have explored the effects of depression prevention programs on other outcomes (e.g., anxiety, behavior problems, academic functioning).

Effect sizes

On average, effect sizes (Efs) of depression prevention programs in children and adolescents have been small to modest. Programs that target samples at increased risk for depression (i.e., selective, indicated) have tended to have larger effect sizes than those delivered to universal samples (Horowitz & Garber, 2006; Merry et al., 2004; Stice et al., 2009), in part because the rates of depressive symptoms and disorders in the control groups are lower in universal as compared to “at-risk” samples. Although some programs have been tested in both universal and targeted samples (Brunwasser et al., 2009), few studies have directly compared these different samples within the same study (e.g., Sheffield et al., 2006).

Control Conditions and Specificity

Effect sizes also vary depending upon the type of comparison group used; that is, Efs tend to be greater when contrasted with a no intervention or waitlist control and less when compared to an active or placebo control (Cuijpers, van Straten, Smit, Mihalopoulos, & Beekman, 2008). Whereas most depression prevention studies in youth have compared a specific intervention to a no-intervention or waitlist control, a few studies have compared a specific program with either another active program (Gillham et al., 2007; Horowitz, Garber, Ciesla, Young, & Mufson, 2007) or a nonspecific intervention (Merry, McDowell, Wild, Bir, & Cunliffe, 2004; Stice, Rohde, Seeley, & Gau, 2008). For example, the Penn Resiliency Program (PRP) has been found to be more effective than a no-intervention control, but not significantly better than a nonspecific intervention called the Penn Enhancement Program (e.g., Gillham et al., 2007) in preventing depressive symptoms (Brunwasser et al., 2009). Comparing an active intervention [e.g., Coping with Stress Course, Lewinsohn, Clarke, Rohde, Hops, & Seeley, 1996; PRP] to a structurally equivalent, but nonspecific program can help determine if any observed preventive effects are due to the specific intervention versus common, nonspecific processes.

Moderators

Examples of factors found in meta-analyses (Horowitz & Garber, 2006; Merry et al., 2004; Stice et al., 2009) to moderate the size of the effects of various depression prevention programs include type of sample (i.e., universal, selective, indicated), participant attributes (e.g., age, sex, race), characteristics of the intervention (e.g., duration, content) and the interventionists (e.g., level of training), and timing of the assessments (e.g., immediately post-intervention, follow-ups of assorted lengths). The role of various individual difference characteristics (e.g., hopelessness, neuroticism, reassurance seeking) as potential moderators of preventive interventions in youth needs further study.

Sex differences

Meta-analyses of depression prevention programs in youth have indicated that their efficacy differs for girls and boys, although the extent and direction of these differences varies depending on the data analytic method used (Garber & Downs, 2011). When “sex” has been operationalized as the percentage of participants in each study who were female (e.g., Horowitz & Garber, 2006; Stice et al., 2009), studies with a higher percentage of females have slightly larger Efs at post-intervention. Alternatively, when samples have been categorized as having higher (e.g., at or above 53%) versus lower percentages of females, and then analyzed separately, studies that had more females and those that had more males both produced significant Efs, which were not significantly different from each other. A third approach involves calculating separate Efs for girls and boys in each study, obtaining an overall weighted mean Efs for girls and boys separately, and then statistically comparing the girls’ versus boys’ Efs (Brunwasser et al., 2009; Merry et al., 2004). Results of such analyses yield low to moderate mean Efs for both males and females, with a

nonsignificant trend for the effect of the intervention compared to the control conditions to be greater for boys than girls. Thus, each method engenders different results, with the third approach of calculating the actual EfSs for girls and boys separately in each study likely producing the most precise estimate.

Mechanisms

When an intervention is shown to have a significant preventive effect, through what mechanisms does this occur? Only a few prevention studies have tested and found significant mediators of the intervention on depression (e.g., Brunwasser et al., 2009; Compas et al., 2011; Tein, Sandler, MacKinnon, & Wolchik, 2004). In at least three studies, cognitive style has been found to partially mediate the effect of the Penn Resiliency Program (PRP) on depressive symptoms (Brunwasser et al., 2009). Compas and colleagues (2011) showed that changes in children's secondary control coping and parents' positive behavior toward their child partially mediated the effects of a family group cognitive behavioral intervention on children's depressive symptoms. More tests of mediation are needed to advance theory, identify active ingredients, and determine whether the effects are specific to the intervention (e.g., Kazdin, 2008; Kraemer, Wilson, Fairburn, & Agras, 2002).

Durability of Effects

Efficacy of most depression prevention programs for youth has been found immediately post-intervention, and several have found significant short-term effects (e.g., up to six months). Evidence of longer-term effects (e.g., two or more years), however, is less common (e.g., Beardslee et al., 2012; Brunwasser et al., 2009; Compas et al., 2011; Sandler et al., 2010). The inclusion of continuation or booster sessions, online refresher courses, and training multiple members of a community (e.g., parents, teachers, peers) may help to produce more sustained effects.

In summary, despite the progress made in the last decade, much more needs to be learned in order to deliver the most efficacious and efficient depression prevention programs. In particular, more precise information is needed regarding moderators and mediators of the intervention-outcome relation. That is, which interventions work best for whom, and through what mechanisms do successful preventive interventions actually work? Have the "right" processes been targeted in existing prevention programs? We highlight here aspects of Abela's work that can most directly inform these issues and thereby guide the further development of interventions for preventing depression in children and adolescents.

Vulnerability to Depression: Who is at risk?

Recognizing that not all risk factors are necessarily causal (Kraemer et al., 1997), we can use empirically supported vulnerabilities to guide the selection of potential targets for intervention. Discovering *who* is most at risk for depression will help determine which individuals are likely to benefit from prevention and how best to allocate limited healthcare resources. Abela provided some of the best empirical support for several key vulnerabilities including cognitive styles (e.g., inferential style, rumination), interpersonal relationships (e.g., reassurance seeking, attachment, dependency), personality (e.g., neuroticism, self-criticism), and contextual risk factors (e.g., parental depression, stress).

Negative Inferential Style

A primary focus of Abela's research was refining and testing the hopelessness model of depression (Abramson, Metalsky, & Alloy, 1989), particularly as it applied to children. The three cognitive styles hypothesized to place individuals at risk of developing hopelessness depression (Abramson et al., 1989) are: inferential style about the *causes* of life events (i.e.,

attributional style; Abramson, Seligman, & Teasdale, 1978), inferential style about *consequences* -- the tendency to expect negative events to have terrible consequences, and inferential style about the *self*-- the tendency to view the self as flawed and deficient after a negative event has occurred (Abela, 2001).

The interaction between inferential styles and life stress, termed the “diathesis-stress component” of the hopelessness theory, has been the subject of extensive research (Abela & Hankin, 2008). Whereas studies of adults generally have found that attributional style serves as a diathesis for the development of hopelessness depression (e.g., Abela, 2002; Abela & Brozina, 2004; Abela, Brozina, Seligman, 2004; Abela & Seligman, 2000), findings regarding the vulnerability conferred by attributional style in children have been mixed (e.g., Abela, 2001; Abela, McGirr, & Skitch, 2007, Abela & Sarin, 2002; Brozina & Abela, 2006; Turner & Cole, 1994). Turner and Cole (1994) suggested that these discrepant findings were due to children not having true attributional styles until they developed the capacity for formal operational thought and abstract reasoning, which occurs around early adolescence. Abela (2001) argued that if this is correct, then data analyses that lumped together children of different ages would likely obscure any real effects. Therefore, he conducted a six-week prospective test of the diathesis-stress component of the hopelessness theory in children at the outer edges of the developmental period of interest, 3rd and 7th graders. Abela (2001) found that negative attributional styles interacted with life events to predict increases in depressive symptoms for 7th but not for 3rd graders.

In a further effort to clarify the nature of the diathesis-stress component of the hopelessness theory of depression in children, Abela and colleagues suggested several other factors that might account for past discrepant findings including the ways in which cognitive vulnerability and life stress were operationalized, and the use of cognitive priming before assessing inferential styles. Through these efforts, Abela and associates generated a body of research that paints a clearer picture of the diathesis-stress component of the hopelessness theory in children. This work has direct implications for the assessment and identification of at-risk youth who might benefit from preventive interventions.

Assessment of Risk: Weakest Link, Idiographic Stress, Cognitive Priming

Weakest link—One of Abela’s greatest contributions to the field was the “weakest link” approach to operationalizing children’s cognitive vulnerability to depression. When studying the diathesis-stress component of the hopelessness theory, traditionally researchers have tested how each of the three inferential styles interacted with stress in isolation of the other two. To obtain the most accurate assessment of a child’s vulnerability to hopelessness depression, however, it is necessary to examine how these styles interrelate. Abela and Sarin (2002) argued that by not considering relations among these three inferential styles, important information about an individual’s cognitive vulnerability status may be missed.

Therefore, Abela and Sarin (2002) compared different ways of operationalizing the cognitive diathesis including the traditional, additive, multiplicative, and “weakest link” approaches. They hypothesized that the most accurate way to represent an individual’s degree of cognitive vulnerability to hopelessness depression would be to use the person’s *highest* score of the three inferential style scales (i.e., most depressogenic). That is, they used the analogy that “a chain is only as strong as its weakest link” (p. 815). To operationalize cognitive vulnerability in this way, Abela and colleagues standardized a child’s scores on the consequences and self subscales of the Children’s Cognitive Style Questionnaire (CCSQ; Abela, 2001) and the generality subscale (i.e., stability and globality) of the Children’s Attributional Style Questionnaire (Seligman et al., 1984). Each child’s *weakest link* score was equal to the highest of the three standardized scores. As they hypothesized, Abela and Sarin (2002) found that the interaction of children’s weakest links

with stress predicted change in their symptoms of hopelessness depression, whereas the individual inferential styles, additive scores, and multiplicative scores did not.

Using this weakest link approach, Abela and colleagues consistently have found support for the diathesis-stress component of the hopelessness theory of depression in children, across a range of stressors including daily hassles (Abela & McGirr, 2007) negative life events (e.g., Abela & Payne, 2003; Abela, McGirr, & Skitch, 2007; Abela & Sarin, 2002; Abela & Scheffler, 2008), and parental depressive symptoms (e.g., Abela et al., 2006). These studies showed that compared to the traditional approach of examining inferential styles separately, the weakest link's interaction with stress was a better predictor of children's depressive symptoms. Moreover, in line with the hopelessness theory, the weakest link interacted with stress to predict increases in symptoms of hopelessness, but not non-hopelessness, depression (Abela & Payne, 2003; Abela, Parkinson, Stollow, & Starrs, 2009).

Contrary to earlier research (e.g., Abela, 2001; Turner & Cole, 1994), none of the studies using the weakest link approach found moderating effects of age. Turner and Cole (1994), however, had focused specifically on attributional style rather than inferential style about consequences or the self. Whereas attributional style is more sensitive to age and does not appear to emerge as a risk factor for depression until early adolescence, inferential style about consequences and the self appear earlier in development (Abela, 2001). Thus, although a negative attributional style may be less likely to be the weakest link in younger children, because inferential tendencies about consequences and the self are given equal weight to the more age-sensitive attributional style, age effects tend to disappear when the weakest link approach is used.

The immaturity of young children's thought processes does not necessarily preclude them from having negative inferential styles that interact with stress to produce an increase in depressive symptoms. Children may start forming inferential styles, especially about consequences and the self, years before adolescence, and these inferential styles may be suitable targets for prevention. Thus, inferential styles can act as diatheses in the development of hopelessness depression even in young children, but when these different inferential styles become risk factors varies with age. Understanding how developmental processes affect specific cognitive vulnerabilities is central to the creation of developmentally sensitive interventions.

Operationalizing life stress: Nomothetic vs. idiographic approach—Most studies of the diathesis-stress component of the hopelessness theory have used a nomothetic (between-subject) rather than idiographic (within-subject) approach to operationalizing stress (e.g. Abela, 2001, Conley et al., 2001, Hankin et al. 2001, Abela & Sarin, 2002, Abela & Payne, 2003). Typically, studies using the nomothetic approach test whether inferential styles assessed at one time point (T1) interact with stressful life events occurring between T1 and a subsequent time point (T2) to predict depressive symptoms at T2, controlling for depression at T1. In this design, an individual's stress level is being compared to the stress level of the whole sample and is considered "high" if it is above the mean for that sample. This approach, however, does not take into account the possibility that an individual may have experienced a significant increase in stress between T1 and T2, yet may still be below the sample mean stress level. According to the hopelessness theory, individuals who have experienced increasing stress levels would be considered to be at high risk for developing depressive symptoms, regardless of where they stand relative to the rest of the sample. Using a nomothetic approach, a person who falls below the sample mean stress level would not be considered to be at high risk for depression (Abela, Aydin, & Auerbach, 2006).

In response to this issue, Abela et al. (2006) suggested that an idiographic approach may better capture complete information about an individual's stress level when testing the diathesis-stress model. To operationalize stress from an idiographic perspective, life events are assessed at multiple time points; an individual's level of stress is determined to be high or low on the basis of where it falls relative to his or her own mean stress level. This removes the effect of individual differences between participants and allows contextual information to be maintained, leading to a more valid test of the hopelessness theory (Abela & McGirr, 2007).

Although support for the diathesis-stress model has been found using a nomothetic approach to operationalizing stress (e.g., Abela & Sarin, 2002, Abela, 2001, Abela & Payne, 2003, Abela, Sakellaropoulou, & Taxel, 2007), the shortcomings of the nomothetic approach may explain some of the mixed findings in the child literature. Abela and McGirr (2007) tested both the nomothetic and idiographic approaches to defining stress in the same sample of offspring of depressed parents. Although there were main effects of stress on depressive symptoms when operationalized either way, only idiographic stress interacted with the children's weakest link to predict changes in depressive symptoms. Much of Abela's later work that found support for the diathesis-stress component of the hopelessness theory adopted an idiographic approach to operationalizing various types of stress, including daily hassles (e.g., Abela & McGirr, 2007, Abela, Morrison, Starrs, 2007, Auerbach et al., 2010) and parental depressive symptoms (e.g., Abela et al., 2006).

Cognitive priming—A third factor that Abela proposed to explain mixed findings in the diathesis-stress component of the hopelessness depression literature is the need for cognitive priming before assessing inferential styles. According to the activation hypothesis, cognitive vulnerability factors, such as negative inferential styles, tend to be latent structures that need to be primed before they can be accurately assessed (Persons & Miranda, 1992). That is, in at-risk, but currently nondepressed individuals, their negative cognitive schemata may not be readily available; priming, however, can make these negative cognitions more accessible (Abela, Brozina, & Seligman, 2004).

Cognitive priming procedures can elicit differences between depression-risk groups and controls on a variety of affective tasks (e.g., Hedlund & Rude, 1995, Teasdale & Dent, 1987). Without such priming, however, high-risk individuals may not exhibit their underlying negative tendencies. Abela and colleagues conducted several studies utilizing cognitive priming techniques in tests of the diathesis-stress model of depression (e.g., Abela, 2002; Abela & D'Alessandro, 2002; Abela & Brozina, 2004, Abela et al., 2004). Perhaps the clearest evidence of the importance of using cognitive priming techniques before assessing inferential styles comes from the study by Abela et al. (2004), which showed that primed, but not unprimed, inferential styles about causes, consequences, and self interacted with stress to predict increases in depressive symptoms. Without cognitive priming, however, support for the diathesis-stress component of the hopelessness theory was not found. Thus, the absence of priming techniques may account for past discrepant findings about the diathesis-stress component of depression.

Other Vulnerabilities

Abela recognized that other vulnerabilities in addition to inferential styles may serve as diatheses that interact with stress. In particular, cognitive processes and interpersonal relationships also may be used to identify at-risk individuals and can be targets of intervention. Abela incorporated cognitive vulnerability factors from other theories into his studies of the diathesis-stress component of depression, most notably Beck's dysfunctional attitudes (1983) and Nolen-Hoeksema's ruminative response style (1991).

Dysfunctional Attitudes—Beck’s (1983) cognitive theory of depression is also a diathesis-stress model such that depressive schemata about the self, world, and future interact with life stress to produce depression. Such schemata “contribute to the onset of a pattern of negative self-referent information processing characterized by systematic errors in thinking” (p. 112, Abela & D’Alessandro, 2002). These errors in thinking, or dysfunctional attitudes, tend to be rigid, extreme, and maladaptive. Typically, these beliefs have been measured in adults with the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978) and in youth with the Children’s Dysfunctional Attitudes Scale (CDAS; Abela & Scheffler, 2008; Abela & Sullivan, 2003).

Across multiple studies, Abela and colleagues showed that dysfunctional attitudes independently interacted with stress to predict changes in depressive symptoms over time (e.g., Abela & D’Alessandro, 2002, Abela & Skitch, 2007, Abela & Sullivan, 2003; Hankin, Fraley, & Abela, 2005). For example, in a study of 136 high school seniors applying to the University of Pennsylvania, Abela and D’Alessandro’s (2002) found a significant interaction between dysfunctional attitudes and stress, such that among students who received a negative admissions decision (high stress) only those who several weeks earlier had reported high levels of dysfunctional attitudes experienced increases in depressive symptoms. In another study, dysfunctional attitudes were included in the determination of children’s weakest links, comprising 14.5% of the sample, and again were found to interact with daily hassles to predict elevations in depressive symptoms (e.g. Abela & Scheffler, 2008).

The interaction of dysfunctional attitudes and stress in predicting depression has been found to be further moderated by self-esteem (Abela & Skitch, 2007). That is, children who endorsed high levels of dysfunctional attitudes had increased depressive symptoms following stress if they also had low self-esteem. Thus, Abela and colleagues have shown that high levels of dysfunctional attitudes represent an important diathesis for the development of depression in youth and therefore might be an important target for intervention.

Ruminative Response Style—According to Response Style Theory (Nolen-Hoeksema, 1991) rumination, which is the process of repetitively focusing attention on one’s depressive symptoms and their implications, increases an individual’s risk of experiencing more severe and persistent depression. Whereas inferential style involves the *content* of thoughts about causes, consequences, and the self, rumination reflects a particular *way* of thinking rather than specific content. Abela and colleagues consistently found a significant relation between ruminative response style and depressive symptoms in children (Abela, Aydin, & Auerbach, 2007; Abela, Vanderbilt, & Rochon, 2004), adolescents (Skitch, & Abela, 2008), and young adults (Hankin et al., 2007; Sarin, Abela, & Auerbach, 2005), children from other cultures (Hong et al., 2010) and in offspring of depressed parents (Abela et al., 2005; Abela, Aydin, & Auerbach, 2007). In contrast to inferential styles, which tend to predict increases in depressive symptoms following exposure to stress, Abela and colleagues generally found a main effect of rumination on depression (Abela, Parkinson, Stolow, & Starrs, 2009; Hankin, Fraley, & Abela, 2005; Sarin et al., 2005; although see Skitch & Abela, 2008 for an exception).

Abela reported mixed results regarding sex differences in the tendency to ruminate. Whereas some studies showed that girls were more likely to engage in rumination than their male counterparts (Abela, Parkinson, Stolow, & Starrs, 2009; Skitch & Abela, 2008), other studies did not find sex differences in rumination (Abela, Aydin, & Auerbach, 2007; Abela, Vanderbilt, & Rochon, 2004; Hong et al. 2010). Hong et al. (2010) reported no sex differences in the amount of rumination, although they also showed that a ruminative

response style was associated with longer and more severe symptoms of depression for girls but not boys.

Additionally, Abela et al. (2007) found no sex differences in the use of rumination, but boys were more likely than girls to engage in distraction and problem-solving in response to stress. Whereas rumination tends to be associated with higher levels of depressive symptoms, distraction and problem-solving tend to be linked with lower depression levels (Abela et al., 2007). Thus, preventive interventions not only should aim to reduce rumination, but also should increase the use of other coping strategies such as distraction and problem-solving.

Interpersonal Vulnerabilities—Interpersonal theories of depression posit that depressed individuals perpetuate a cycle of negative interpersonal exchanges that tend to exacerbate their depression (Coyne, 1976). These negative interactions often take the form of excessive seeking of reassurance from others in order to alleviate concerns about their worth and lovability, even if such assurances have been repeatedly provided (e.g., Joiner, Metalsky, Katz, & Beach, 1999). Although, at first, others typically respond to depressed individuals with concern and support, as the depressed person continues to seek reassurance, others may become irritated and rejecting, which then often leads to further exacerbation of the depressed person's symptoms.

Abela focused on two important interpersonal vulnerabilities for depression: excessive reassurance-seeking (Abela, Morrison, & Starrs, 2007) and insecure attachment (Abela et al., 2005; 2009). Indeed, excessive reassurance-seeking in children is another type of diathesis that interacts with stress to predict increases in depressive symptoms across time (Abela, Morrison, & Starrs, 2007; Abela, Zuroff, Ho, Adams, & Hankin, 2006). Using experience sampling over eight weeks in children of depressed parents, Abela et al. (2007) showed that higher levels of reassurance-seeking predicted increases in depressive symptoms following exposure to daily hassles. Abela et al. (2006) found an interaction with age such that excessive reassurance seeking was associated with greater elevations in depressive symptoms following increases in either hassles or parents' depressive symptoms for older but not younger children. Abela and colleagues (2006) suggested that this moderating effect of age may occur because as children get older, reassurance seeking is "increasingly perceived as developmentally atypical and consequently may become increasingly more apt to be responded to in a negative manner" (p. 184), thereby increasing stress and exacerbating depressive symptoms.

The notion that reassurance-seeking may exert its harmful effect through the negative responses it elicits from others is further supported by the fact that higher reassurance-seeking predicts more dependent interpersonal stressors (Shih, Abela, & Starrs, 2009). Thus, factors that are normative and adaptive at younger ages may begin to confer risk as children transition to adolescence (Fichman et al., 1996). This highlights the importance of taking development into account both when studying vulnerability factors and when determining appropriate targets for intervention.

With regard to poor attachment quality, Abela and colleagues (Abela, Hankin, Haigh, Vinokuroff, Trayhern, & Adams, 2005) showed in a study of offspring of depressed parents that insecure attachment was significantly related to children's level of current depressive symptoms. Moreover, insecure attachment interacted with excessive reassurance-seeking to predict children's current depressive symptoms and past depression diagnoses. In a prospective study of offspring of depressed parents, Abela et al. (2009) found that children who reported high levels of negative attachment cognitions at baseline subsequently reported more depressive symptoms when their parents were experiencing high as compared

to low levels of depressive symptoms. Thus, another important target of interventions may be to reduce reassurance seeking, particularly in older children, and to improve attachment in offspring of depressed parents.

Personality

Abela and colleagues have demonstrated that various personality characteristics constitute risk factors for depressive symptoms. In particular, neuroticism (Auerbach et al., 2010; Hankin, Fraley, & Abela, 2005; Hankin, Lakdawalla, Carter, Abela, & Adams, 2007), behavioral inhibition (Brozina & Abela, 2006), self-criticism and dependency (Abela, Skitch, Auerbach, & Adams, 2005; Abela & Taylor, 2003; Abela, Webb, Wagner, Ho, & Adams, 2006; Adams, Abela, Auerbach, & Skitch, 2009), self-esteem variability (Auerbach, Abela, Ho, McWhinnie, & Czaikowska, 2010), and dispositional pessimism (Abela, Auerbach, & Seligman, 2008) all have been linked to depression. Despite the stability of these constructs, Abela's work highlights that these traits and their correlates may be valuable targets for prevention and treatment.

Neuroticism, or negative emotionality and emotional instability, reflects an individual's perceptions and experiences of the world as threatening or distressing (Watson, Clark, & Harkness, 1994; Widiger et al., 1999). Neuroticism has been shown to predict prospective elevations in depression (e.g., Kendler et al., 2002; Krueger, 1999; Widiger, Verheul, & van den Brink, 1999) and has been theorized to be associated with cognitive vulnerability to depression (e.g., Clark, Watson, & Mineka, 1994; Hankin & Abramson, 2001). In a study of healthy college students, Hankin, Fraley, and Abela (2005) showed that neuroticism predicted both negative cognitions and depressive symptoms assessed daily over 35 consecutive days.

In children, neuroticism was found to interact with self-esteem to predict increases in depressive symptoms (Auerbach, Abela, Ho, McWhinnie & Czaikowska, 2010). Specifically, high neuroticism and low self-esteem significantly predicted increases in depressive symptoms, although unlike cognitive styles, neuroticism did not predict increases in depressive symptoms following negative events. Auerbach and colleagues (2010) speculated that perhaps neuroticism was a more distal predictor of depressive symptoms that had its greatest impact in the presence of other risk factors rather than in response to stress alone.

Abela and colleagues also studied self-criticism and dependency as personality vulnerabilities to depression. For example, following an increase in daily hassles, children high in self-criticism reported more depressive symptoms, whereas those low in self-criticism did not (Adams et al., 2009). Similarly, adults who were low in self-criticism did not experience increases in depressive symptoms following negative events, whereas those with high levels of self-criticism who also had low self-esteem reported more depressive symptoms in response to stress (Abela, Webb et al., 2006). Thus in children, self-criticism alone was sufficient to confer risk for depression, whereas in adults additional risks such as low self-esteem may be necessary to predict depression in those high in self-criticism.

Findings have been more mixed with regard to the relation between dependency and depression, particularly in children. Abela and Taylor (2003) reported that dependency was not a risk factor for depression in school children in grades 3 and 7, and suggested that perhaps dependency may be a normative characteristic in younger children as compared to adolescents and adults. In adults, however, high dependency predicted increases in depressive symptoms following negative life events in those with high self-esteem, whereas in adults with low self-esteem, high dependency was associated with chronically elevated depressive symptoms regardless of the number of daily hassles (Abela et al., 2006).

One mechanism suggested as underlying the relation of the personality vulnerabilities of neuroticism, self-criticism, and dependency and depression is the amount of dependent stress generated by individuals with these characteristics. For example, highly neurotic people with elevated depression levels have been shown to generate stressors over time (e.g., Kendler, Gardner, & Prescott, 2003; Lakdawalla & Hankin, 2008). In children of parents with a history of depression, Shih, Abela, and Starrs (2009) found that higher levels of self-criticism significantly predicted more dependent stressors, and greater interpersonal dependency predicted higher levels of dependent interpersonal stressors in girls. Levels of self-criticism and dependency, however, were not related to the amount of independent stressors these children experienced. Thus, these personality vulnerability factors may increase risk for depression specifically by generating stress. An important implication for intervention is that whereas changing an individual's personality (e.g., neuroticism, self-critical or dependent tendencies) may be difficult, targeting the consequences of these traits, such as the generation of dependent stressors, might be a more direct route to reducing and preventing depression.

Relations among vulnerabilities—Thus, Abela and colleagues have demonstrated that not only do cognitive vulnerabilities (e.g., inferential styles and ruminative response styles) predict increases in depression, but also certain personality characteristics (e.g., neuroticism, self-criticism, dependency) confer risk for depressive symptoms. These various cognitive and personality vulnerabilities have been found to be correlated with each other and with depression, although they also can be differentiated from each other. The three cognitive vulnerability factors (i.e., negative inferential style, dysfunctional attitudes, and ruminative response style) are theoretically and empirically distinct risk factors for depression. In a sample of several hundred college students, Hankin et al. (2007) conducted exploratory and confirmatory factor analyses and demonstrated that rumination was a separate vulnerability factor for depression distinct from neuroticism, negative cognitive style, and dysfunctional attitudes; dysfunctional attitudes also formed a discriminable factor independent of the other vulnerability constructs. The factor analysis further revealed that neuroticism was independent of the cognitive vulnerabilities (i.e., inferential style, dysfunctional attitudes, ruminative response style) but closely linked to self-esteem and depressive symptoms (Hankin et al., 2007). Hong et al. (2010) reported that higher levels of rumination were associated with more depressive symptoms and episodes, even when controlling for neuroticism. Thus, although the various vulnerabilities are correlated with each other, they each contribute unique variance to the prediction of depression, and therefore should be considered separately when constructing and evaluating interventions for depression.

Both cognitive and personality styles are potential targets for prevention and treatment. It is even more likely, however, that personality characteristics will serve as moderators of the efficacy of some interventions on reducing or preventing depression. A clear recommendation is that these personality traits should be assessed prior to intervening, and then tested as potential moderators. Once a significant moderator of the effects of the intervention on depression has been found, then the next step will be to identify the mechanisms that account for the differential outcomes for individuals high versus low on the particular trait. Through this iterative process, it will be possible to further refine the interventions to fit different levels and types of vulnerabilities.

Development of Cognitive Vulnerabilities

Given the centrality of negative cognitive/inferential styles as risks for depression, preventing their development in the first place is another logical target for intervention. How do these cognitive vulnerabilities develop? Abela and colleagues conducted several investigations relevant to this question.

Abela et al. (2005) speculated that interpersonal factors, particularly insecure attachment, may influence the onset of depression through the development of negative cognitions and dysfunctional attitudes. Some support for this view was found in a prospective study of young adults whereby insecure attachment style was associated with higher levels of dysfunctional attitudes, which in turn, predicted lower self-esteem and subsequent elevations in depressive symptoms (Hankin, Kassel, & Abela, 2005). Thus, these cognitive factors mediated the relation between insecure attachment and depression in adults.

In a cross-sectional study of factors that possibly contributed to the development of cognitive vulnerability, Sarin and Abela (2005) found distinct correlates of the three types of inferential styles. Children's inferential style about causes was associated with higher levels of negative life events and lower levels of social support from peers; inferential style about consequences was associated with recent negative life events and pessimistic feedback from parents about the consequences of these events; inferential style about the self was linked to histories of emotional and sexual abuse. Thus, distinct social and contextual factors may be involved in the development of the different inferential styles.

Emotional abuse, in particular, has been linked to the development of negative cognitions because the abuser sends direct messages to the child, especially about the child's self-worth (Rose & Abramson, 1992). In a sample of offspring of depressed parents, Gibb and Abela (2008) found that children's reports of emotional abuse from parents and verbal victimization from peers were associated with negative changes in their inferential styles about consequences and self-characteristics and depressive symptoms over a 12-month follow-up.

Thus, disrupted interpersonal relationships are one important source of information that directly feeds into children's developing views of themselves, others, and the world. Early interventions [e.g., Parent-Child Interaction Therapy (PCIT); Positive Parenting Program (Triple P)] that teach parents how to increase positive and reduce negative interchanges with their young children might be used to help prevent the emergence of negative cognitive styles. Although such parenting programs have been found to effectively reduce behavior problems in young children (e.g., for a review see Thomas & Zimmer-Gembeck, 2012), their specific impact on children's inferential style has not been studied.

Another way to manage the effects of the social context on the development of cognitive vulnerabilities would be to expose children to individuals who can provide them with more positive messages. For example, regular contact with supportive role models such as teachers, coaches, or older siblings could help strengthen children's "cognitive muscles" so that they can counteract the negative impact of dysfunctional relationships on their developing inferential styles. Gibb and Abela (2008) recommended that future studies are needed "to determine whether the presence of one or more supportive individuals in a child's life who provide adaptive inferential feedback following negative events buffers the effects of emotional abuse from parents or verbal victimization from peers on the development of negative inferential styles and depression" (p. 173).

Parental Depression as a Vulnerability to Depression in Children

Parental depression is one of the most salient risk factors for the development of psychopathology, particularly depression, in children (e.g., Beardslee, Gladstone, & O'Connor, 2011; Goodman, 2007). Recently, the Institute of Medicine (England & Sim, 2009) designated parental depression to be a major public health priority. Abela's research program investigated several important risk factors relevant to the prevention of depression in high-risk youth.

Abela and colleagues found that offspring of depressed parents have higher levels of depressive symptoms (Abela, Skitch, Auerbach, & Adams, 2005), more insecure attachments and greater reassurance seeking (Abela, Hankin, Haigh, Vinokuroff, Trayhern, & Adams, 2005; Abela, Zuroff, Ho, Adams, Hankin, 2006), greater exposure to stress, particularly their parents' depressive symptoms (Abela, Skitch, Adams, & Hankin, 2006; Abela, Zuroff et al., 2006), and more negative cognitions (Abela, Skitch, Auerbach, & Adams, 2005) as compared to children of nondepressed parents. Moreover, Abela and colleagues also found evidence consistent with diathesis-stress models of depression in offspring of depressed parents. For example, children with depressive inferential styles reported greater elevations in depressive symptoms following increases in their parent's level of depressive symptoms (i.e., the stressor) than did children who did not exhibit such styles (Abela, Skitch, Adams, & Hankin, 2006). Children with high levels of reassurance seeking reported greater elevations in depressive symptoms following increases in their parents' symptoms than did children with low levels of reassurance seeking. Offspring of depressed parents also were at higher risk for developing depressive symptoms in response to their parent's increasing symptoms if the children had a greater depressogenic weakest link (Abela, Skitch, Adams, & Hankin, 2006). Finally, children's negative attachment cognitions have been found to interact with elevations in parents' depressive symptoms to predict increases in children's symptoms (Abela, Zinck, Kryger, Zilber, & Hankin, 2009).

Thus, parental depression can act as a stressor itself that interacts with other cognitive or interpersonal vulnerabilities to predict increases in children's symptoms. Parental depression also can serve as a distal precursor of children's depression, mediated by insecure attachment or negative cognitive styles in the children. The implications of this work for prevention are clear.

First, although not all children of depressed parents develop psychopathology, and not all children with psychopathology have a depressed parent, offspring of depressed parents are definitely at elevated risk and therefore are appropriate targets for preventive intervention. Several prevention programs have been found to reduce depressive symptoms and disorders in offspring of depressed parents (e.g., Compas et al., 2009; Garber et al., 2009). More research is needed to identify which children are most and least likely to respond to these preventive interventions, what mechanisms account for these effects, and how best to disseminate these programs more broadly in the community.

Second, a potentially important target for preventing depression in at-risk offspring would be to eliminate or at least reduce depression in the parents. A small but growing literature has shown a significant relation between improvements in parents' depression and changes in their children's psychopathology and functioning (Gunlicks & Weissman, 2008). Questions remain, however, about the strength, direction, and underlying causes of this covariation (Garber, Ciesla, McCauley, Diamond, & Schloedt, 2011). Studies are needed that randomize depressed parents to an active, state-of-the-art treatment versus a no treatment (e.g., placebo) or usual care control condition, and examine whether changes in children's outcomes are indeed the direct result of parents' depression remitting as a function of treatment.

Third, preventive interventions can be constructed to alter some of the consequences of the parents' depression on the developing child. For example, depressed parents can be taught strategies for building more secure parent-child attachment relationships (e.g., Lieberman, 1992). In addition, depressed parents can learn to reduce the negative messages they communicate to their children about the causes and consequences of negative events and about their child's self-worth.

Finally, interventions exist or can be developed further that work directly with at-risk children to teach them skills for preventing depression. For example, offspring of depressed parents can be taught ways of coping with stress, particularly the stress of having a depressed parent (e.g., Compas et al., 2009). High-risk offspring also can learn strategies for countering their dysfunctional beliefs and negative inferential styles (e.g., Clarke et al., 2001).

Although evidence exists of the efficacy of certain interventions for preventing depression in offspring of depressed parents (e.g., Clarke et al., 2001; Compas et al., 2009; Garber et al., 2009), further randomized controlled depression prevention trials are needed. In particular, we do not know the incremental benefits or relative efficacy of interventions that target reducing parental depression, altering the impact of parental depression on their children, or intervening directly with the children for preventing depression in these high risk offspring. That is, what combination of interventions maximizes the beneficial preventive effects on children? Conversely, can we streamline the intervention down to its bare essential active ingredients necessary for preventing depression in these children?

Additional Implications of Abela's Research for the Prevention of Depression in Youth

Assessment

Weakest link—A major contribution of Abela's work that has not yet been applied to prevention involves the assessment of risk. Most prevention studies have not selected participants on the basis of cognitive vulnerabilities, and the few that have (e.g., Seligman et al., 1999) did not use the methodologies recommended by Abela's findings. In particular, the weakest link method has not been applied to the identification of at-risk children based on either their inferential styles or other cognitive diatheses such as dysfunctional attitudes or self-esteem. Given that many depression prevention programs involve cognitive restructuring (e.g., Clarke et al., 2001; Jaycox et al., 1994), obtaining a comprehensive and more precise evaluation of a child's specific cognitive vulnerabilities might improve the efficacy of such programs by individualizing the intervention to address their weakest link, or conversely, to strengthen their least negative beliefs (Morris, Ciesla, & Garber, 2008). Abela and Scheffler (2008) explicitly recommended that: "interventions should take a broad approach towards assessment of cognitive vulnerability and subsequently implement interventions that target each child's unique area(s) of weakness. This contrasts with approaches that uniformly target a limited number of specific cognitive vulnerabilities (e.g., attributional style)" (p. 346).

One intriguing question for future studies is what happens to each of the three inferential styles as a function of a preventive intervention? As the weakest (i.e., most negative) inferential style improves, what happens to the other two "less" weak links? Does an intervention that targets one inferential style necessarily improve the other two? Is there a particular order in which these inferential styles should be tackled? Should we construct programs that are specific to one type of cognition or should interventions aim more broadly to affect all of them? If a broader approach is better, then how can information about an individual's weakest link be used to further increment the efficacy and efficiency of the intervention?

Priming—A second assessment recommendation derived directly from Abela's research is that some form of cognitive or emotional priming should be used when attempting to identify vulnerable individuals. Some risk factors, such as negative inferential styles, may not be readily accessible through direct questioning, particularly in children. Therefore, to

increase the chances of identifying people who are genuinely at risk, using priming to “awaken” their latent vulnerabilities is recommended.

Transfer of Training

The concept of priming is not only important for assessing vulnerabilities, but it also is relevant to the state of individuals during implementation of the intervention. That is, will participants learn better if they are actually experiencing the negative cognitions at the time they are learning to deal with them? This is a major challenge for prevention science more generally. In contrast to treatment, which is provided when patients are experiencing acute symptoms, preventive interventions are offered when individuals have no or few symptoms; at least two problems may arise as a result. First, individuals who are not currently or have never had symptoms of depression might be less motivated to prevent them. Second, individuals might not learn to challenge their negative cognitions or other vulnerabilities when they are not actively experiencing them at the time of the training sessions. The literature on transfer of training (e.g., Barnett & Ceci, 2002) and state-dependent learning (Overton, 1991) could provide interesting directions for prevention research. Whether priming a person’s specific vulnerabilities or depressive symptoms actually facilitates the uptake of the skills taught in preventive interventions is an important question that needs to be addressed.

Personalization: Compensatory versus Capitalization Approaches

Another critical issue for the prevention of depression is how do we efficiently match an individual’s specific risk factors to the most relevant intervention for that person? Abela provided a wealth of information about various vulnerabilities to depression. Once we have identified an individual’s risk profile and we have interventions that address these vulnerabilities, the next big question is whether the intervention should target individuals’ weaknesses by providing them with compensatory strategies, or should a capitalization approach that directly builds on their existing abilities be used (Rude & Rehm, 1991)?

The compensatory approach aims to change, reduce, or improve the person’s vulnerabilities. Presumably, eliminating what places the person at risk will prevent the onset of depression. For example, adolescents with high levels of social difficulties (e.g., conflict with parents or peers) have been found to benefit from an intervention that targeted improving interpersonal relationships (Gunlicks-Stoessel et al., 2010; Young, Mufson, & Gallop, 2010). A potential problem with the compensatory approach, however, is that a person’s vulnerabilities might be especially resistant to change, thereby making it even more difficult to effectively implement the intervention.

In contrast, the capitalization model focuses on enhancing a person’s strengths. The treatment literature indicates that youth with more cognitive and behavioral resources benefit the most from CBT (e.g., Brent et al., 1998; Lewinsohn, Clarke, Rohde, Hops, & Seeley, 1996; Rohde, Seeley, Kaufman, Clarke & Stice, 2006). No matter how much we build up a person’s assets, however, the chances of their developing depression likely will remain high as long as their vulnerabilities persist.

The dilemma of how best to individualize interventions can be addressed in several ways. Hankin and Young (2012) have proposed a matching study that compares the responses of individuals with cognitive versus interpersonal vulnerabilities to a cognitive behavioral or an interpersonal preventive intervention. Such matching approaches also can be applied within an area of vulnerability such as among the different inferential styles (e.g., causes, consequences, self), interpersonal vulnerabilities (e.g., reassurance seeking, dependency), or personality types (e.g., neuroticism, behavioral inhibition).

Abela's work has shown that both cognitive and interpersonal vulnerabilities increase risk for depression; therefore, targeting both probably makes sense. Indeed, several existing depression prevention programs include both cognitive and social components (e.g., Jaycox et al., 1994; Pössel et al., 2004). Ideally, preventive interventions will simultaneously reduce individuals' specific vulnerabilities while also expanding their strengths. For example, with regard to response styles (Nolen-Hoeksema, 1991), interventions could both reduce ruminative responses to stress and at the same time increase the use of more adaptive strategies such as distraction and problem-solving. Given limited time and resources, interventions that can be personalized to directly target both a person's specific limitations and skills are likely to produce the most effective and cost efficient outcomes.

Another variation of the matching approach would be to address a person's vulnerabilities sequentially. That is, first identify the individual's weakest link and provide the most relevant intervention. Then, move on to the next most vulnerable factor and implement the corresponding intervention. Thus, a prevention program can be comprised of multiple modules each of which targets a particular vulnerability. The order in which these modules are delivered can be personalized to fit each individual's *hierarchy of risk*. One potential limitation of such personalization, however, is that the more individualized a prevention program becomes, the more likely it will need to be delivered one-on-one rather than in a group format, thereby making it less efficient and more expensive. Given that most depression prevention programs for youth have been delivered in a group format (e.g., Brunwasser et al., 2009; Clarke et al., 2001; Spence, Sheffield, & Donovan, 2003), more work is needed to determine how best to integrate these seemingly mutually exclusive goals.

One related complication is how to configure the groups with regard to personal vulnerabilities. Are the interventions more effective when all members share a common risk factor (e.g., negative inferential style) so that the sessions can really target the vulnerability? Conversely, is it better if the group is comprised of a mixture of individuals with different types and degrees of vulnerabilities so they can learn from each other? This is an as yet unanswered empirical question.

Finally, although it is easier to treat the various vulnerabilities categorically, it probably makes more sense to take a more dimensional perspective. That is, most individuals fall somewhere along a continuum on any particular risk factor, rather than simply either "having or not having" the characteristic. Therefore, creating a profile indicating where on the continuum an individual falls with respect to each of the vulnerabilities of interest probably will be more useful than categorizing them as either/or for both identifying who is at risk and measuring how much they change as a function of the intervention.

Implications for the Content of Preventive Interventions; Mechanisms: What to target

Discovering *who* is at risk for depression will help determine which individuals should be targeted for prevention. Such "selective" samples have included youth who were offspring of depressed parents (Beardslee et al., 1997; Clarke et al., 2001; Compas et al., 2009; Garber et al., 2009), exposed to specific stressors such as family conflict, parental divorce, death of a loved one (Gillham et al., 1995; Sandler et al., 2003; Wolchik et al., 2002), high in anxiety (Lock & Barrett, 2003; Lowry-Webster, Barrett, & Lock, 2003), characterized by negative cognitive styles (Seligman et al., 1999), or had prior depressive episodes (Clarke et al., 2001; Garber et al., 2009). The content of the prevention programs with these samples, however, has varied regarding how much express attention has been paid to the risk factor(s) for which the participants were selected. For example, some depression prevention studies using offspring of depressed parents as the index of risk have emphasized cognitive restructuring and problem solving, and only briefly addressed the parents' mood disorder in particular (Clarke et al., 2001; Garber et al., 2009). In contrast, other studies of at-risk

offspring have explicitly attempted to alter the consequences of the parents' depression on their behaviors toward their children and provide the children with skills for coping with their parents' depression (Compas et al., 2009). Both of these approaches have been shown to be efficacious; their relative efficacy has not been tested, however.

Even if an underlying cause of depression is not malleable (e.g., genes), the more proximal endophenotypes that link distal causes to subsequent depression (Roberts & Kendler, 1999) may be amenable to change. For example, the temperamental characteristic of "stress reactivity" may be the phenotypic expression of a genetic predisposition. Training stress-reactive individuals to reduce their initial response to negative life events and cope with the stress once it has occurred may be an effective strategy for preventing subsequent depression when such individuals are faced with adversity. Moreover, identifying the specific mechanisms associated with a particular child's risk will allow us to better individualize the prevention program for him or her.

There may be gender differences in those malleable characteristics. Thus, future research efforts should identify the risk processes that increase the likelihood of depression in females as well as males, recognizing that these etiologic pathways may differ. Basic knowledge of what accounts for sex differences in depression then can be translated into the construction of gender-sensitive preventive interventions. A more comprehensive understanding of the mechanisms through which depression emerges in females and males is needed in order to design the most gender appropriate interventions for preventing it.

In summary, the field of psychology has suffered a tremendous loss by the passing of John Abela. His contributions to our understanding of the risks for the development of depression were extensive, and not easily reviewed in a single paper. Had Abela been able to continue his work, it is clear that he would have had a significant impact on the prevention of depression as well. We hope that the current review provides at least the beginning of a framework for carrying forward Abela's unfinished work.

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