

Cognitive, affective and social mechanisms in depression risk: Cognition, hostility, and coping style

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Although some research has assessed cognitive variables in individuals at risk for depression, few studies have specifically assessed the role of automatic thinking, and virtually no studies have assessed anger and coping in this group. The current study compared measures of these variables in a high-risk group that was defined on the basis of a previous episode of depression, and a control group comprised of low-risk/never depressed individuals. Even though neither group evidenced depressive symptoms at the time of assessment, group comparisons and regression analyses indicated that high-risk individuals reported more negative automatic thoughts than did low-risk participants and that social support seeking, self-blame, and avoidance emerged as coping predictors of risk as did higher levels of anger and hostility. These data thus suggest patterns of interpersonal, behavioural, and cognitive variables that may characterise depression risk.

A number of factors make depression a significant public-health concern. Depression is not only associated with substantial emotional distress and psychological impairment, but also with impaired interpersonal, marital, and occupational functioning (Hammen, 1991a). Perhaps the most serious

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aspect of depression, however, is its recurrent nature. Although most individuals completely recover from a depressive episode, depression is a chronic problem for many individuals. For example, depending upon the time frame and patient group sampled, recurrence rates have been found to be as high as 87% (Keller & Boland, 1998), while other studies have found recurrence rates ranging up to 75% (Angst, 1992). Moreover, an elevated risk for the recurrence of depression can extend over a decade after the index episode (Boland & Keller, 2002). The experience of a clinically significant depressive episode is thus a clear risk factor for future episodes.

Efforts to understand the risk mechanisms that underlie depression have been the focus of considerable research interest over the last decade (Hammen, 2001; Ingram & Price, 2001). The goal of much of this research has been to determine which variables might serve as mechanisms in both the onset and recurrence of depression. A great deal of this effort has focused on the assessment of the role of cognitive variables in the creation and realisation of risk. For instance, a number of studies have now documented that high-risk individuals evidence a tendency to process information in a dysfunctional fashion (Ingram, Miranda, & Segal, 1998), particularly when they encounter stressful events or mood-invoking situations (Segal & Ingram, 1994). Research has further suggested that such variables may have causal relevance for both relapse and recurrence. Specifically, Segal, Gemar, and Williams (1999) found that the intensity of negative cognitive reactions in response to a mood-induction procedure predicted depression onset up to two years after treatment. Other data have documented similar cognitive processes in children as young as eight years old (Taylor & Ingram, 1999). Such data suggest that cognitive processes that are reactive to stressful events may constitute at least some of the mechanisms that lead to relapse and recurrence.

This risk research suggests that, in at least some cases, dysfunctional cognitive processes need to be activated before they can be detected in nondepressed but vulnerable individuals (Segal & Ingram, 1994). However, because the construct of cognition encompasses a number of different processes and domains (Ingram, 1990), activation procedures may not be a prerequisite for uncovering all levels of dysfunctional cognition in high-risk individuals (Alloy & Abramson, 1999). One key cognitive process that is featured in depression theories, and that may be elevated in high-risk individuals, is negative automatic thinking (Beck, 1967; Ingram et al., 1998; Teasdale & Barnard, 1993). Beck (1967) defined automatic thoughts as depression-inducing self-statements that reflect distorted thinking patterns. Although some data suggest that automatic thinking diminishes once depression has remitted (Dobson & Shaw, 1986), such thinking may nevertheless be elevated in vulnerable individuals when compared to

nonvulnerable individuals, and may thus serve as a risk mechanism for depression.

Similarly, when compared to nonvulnerable individuals, positive automatic thoughts may be less frequent for vulnerable individuals. In fact, some data have suggested the presence of low-level dysfunctional thinking in nondepressed individuals who may be at risk for depression. For example, Ingram, Overbey, and Fortier (2001) found that nondepressed individuals who reported lower levels of maternal care, a potential risk factor for depression (see Gerlsma, Emmelkamp, & Arrindell, 1990; Parker, 1994), reported moderately elevated levels of negative automatic thinking and moderately diminished positive thinking, when compared to nondepressed individuals who reported at least average levels of maternal care. These data thus offer tentative suggestions that automatic thinking may reflect a vulnerability mechanism for depression. One purpose of the current study was to determine whether such automatic thinking factors occur in individuals who are not depressed but who are well-documented to be at heightened risk because they have previously experienced a depressive episode.

Even though data on the link between cognitive variables such as automatic thinking and depression risk are important, other variables that are inherently social in their origin may also characterise depression risk. In this vein, hostility and anger are known to be associated with depression in some individuals, a fact that is reflected in the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV TR; American Psychiatric Association, 2000). The DSM notes in this regard that depressed individuals may experience increased irritability or anger, and that in some cases of depression the predominant mood state is one of irritability or hostility rather than sadness. The DSM description of anger is consistent with a variety of studies that have long shown a link between anger and both clinical and subclinical depression (Hokanson & Butler, 1992; Kendell, 1970; Riley, Treiber, & Woods, 1989; Scott, Ingram, & Shadel, 2003).

Although it is clear that elevated levels of anger occur in some depressive states, it is less clear whether increased anger is also characteristic of vulnerable individuals. That is, do those individuals who are not depressed but who are at risk show heightened levels of anger toward others? The answer to this question may have potentially important implications for understanding the recurrence of depressive states. That is, if anger tends to be elevated in vulnerability, then this variable may play an important risk function in the onset of depression, particularly in conjunction with the occurrence of some types of life events. For instance, in a social context individuals with higher levels of anger may tend to interpret the behaviour of others in ways that interfere with effective interpersonal interactions, which in turn leads to depression (e.g., Joiner, 2002; Joiner & Coyne, 1999). Before

addressing such questions, however, it is important to determine whether nondepressed individuals who are at risk for depression have elevated levels of anger, at least in relation to those who are not at risk.

An additional issue involves the possible characteristics of anger that may occur in risk states. Anger is a multidimensional construct that includes not only an affective state but also cognitive manifestations and behavioural expressions. In this vein, Buss and Perry (1992) have argued compellingly that *anger* constitutes an affective state characterised by physiological arousal. They differentiate this affective state from both *physical* aggression and *verbal* aggression, which are considered behavioural dimensions and reflect a desire to harm others. *Hostility*, on the other hand, characterises a more cognitive component composed of perceptions of unfair treatment that result in animosity toward others. Although generally correlated, such dimensions can vary independently; for example, an individual with high levels of hostility need not be either verbally or physically aggressive toward others. Little research in depression has examined the various dimensions of anger, nor has research tended to examine these dimensions in individuals who are not depressed but who are at risk.

Life stress is another variable that has consistently been linked to the depressed state. In this regard, an extensive body of research has shown that stressful life events are associated with depression (Monroe & Hadjiyannakis, 2002; Monroe & Simons, 1991). Moreover, a number of variables are known to moderate the effects of stress. Chief among these variables are coping responses, typically viewed as the response to adverse events that serve to minimise the psychological effects of these events (Lazarus & Folkman, 1984). A common perspective in coping research is that coping efforts can take two forms: problem-focused coping, used to manage the stressful event itself, or emotion-focused coping, used to regulate emotional responses to the event (Folkman, 1997). Although coping responses can be adapted to fit a particular stressful situation, it is also generally recognised that individuals can exhibit broad coping styles that tend to instigate specific coping efforts. As with several other variables, a wealth of research has examined the link between coping and depression (Johnson, Hayes, Field, Schneiderman, & McCabe, 2000), but little research has focused on particular coping styles in individuals who are not depressed but who are vulnerable to depression. Coping styles in this population may be particularly important in that certain styles may either minimise or maximise the probability of depression. That is, whereas some coping styles might help mitigate depressotypic responses to stress, other less-well-adapted approaches might increase the probability of depression when stresses occurs.

In sum, a variety of studies have assessed cognition, anger, and coping in depression, but with the exception of cognitive variables, few studies have focused on these variables in nondepressed but high-risk individuals. In the

area of cognition, some research has found that dysfunctional cognition normalises in remission, but it is yet unclear whether there are relative differences between vulnerable and nonvulnerable individuals on these variables. Similar vulnerability questions have been largely unaddressed in the areas of anger and coping. Given these issues, the purpose of the current study was to examine automatic thinking patterns, the various elements of anger, and different coping styles as possible risk markers in individuals vulnerable to depression. Depression risk can be operationalised in a variety of different ways (Ingram et al., 1998), but, as previously noted, data consistently show that the experience of a previous episode of depression is a significant risk factor for depression recurrence. In the current study, participants evidencing a previous episode of clinically significant depression were compared on a range of automatic thinking, anger, and coping dimensions with individuals who had not experienced a previous episode.

METHOD

Participants and procedure

Research participants consisted of 104 students who participated in partial fulfilment of requirement for their introductory psychology classes. Participants were selected from a pool of approximately 1000 students who took part in a mass testing session and who completed, among other measures, a screening measure of past depressive symptoms and the Beck Depression Inventory (BDI; Beck, 1967). The screening measure consisted of the nine items from the DSM-III-R criteria for depression. Participants were asked to indicate whether they had experienced any of these symptoms, at any point in the past, for a period of at least two weeks. Previously depressed participants were those who indicated that they had experienced at least five of these symptoms for a two-week period, and who had this episode confirmed by the Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon, & First, 1989). Participants were defined as never depressed if they indicated that they had experienced no more than three of these nine symptoms for a period of two weeks. To ensure that no individual in the sample was currently depressed, participants were only selected if they scored 7 or less on the BDI, or roughly the mean of the mass-tested sample.

Participants were invited to an additional session where they completed measures of hostility, coping, and automatic thinking. In addition, they were readministered the BDI to ensure that they were not experiencing depressive symptoms at the time of this additional assessment. Participants were also administered a modified version of the BDI that inquired about the experience of depressive symptoms over the course of the previous year. Data from these administrations are described in the results section. The

SCID module for past depression was also administered to confirm classification of participants as either formerly depressed or never depressed. If individuals who had indicated an episode of depression during the mass screening did not meet these additional criteria, their data were excluded from the study. Participants' data were also excluded if their depressive episode was part of a bipolar disorder. Data for never-depressed participants were excluded if they met three or more depression criteria on the SCID. Data were also excluded for any participants who scored above 7 on the readministered BDI. The final sample consisted of 50 never depressed/low-risk (64% women) participants, and 54 previously depressed/high-risk (76% women) participants.

Measures

Depressive symptomatology. The Beck Depression Inventory (BDI; Beck, 1967) was used to assess current depressive symptomatology. The BDI is a 21-item self-report measure of a range of depressive symptoms. Each item is answered on a 0 to 3 scale with total scores ranging from 0 to 63. Beck, Steer, and Garbin (1988) examined research assessing the psychometric properties of the BDI over a period of 25 years and have shown that this measure consistently demonstrates strong reliability and validity.

Previous depression screening. The screening measure used during the mass testing session has been used in previous studies (e.g., Ingram & Ritter, 2000) and was adapted from DSM-III-R criteria for a major depressive episode. The measure presents participants with a description of each of the nine depressive symptoms listed in the DSM-III-R and asks them to answer true or false for each individual symptom according to whether or not they have experienced the symptom at some point in their life for at least a two-week period. As noted, the SCID was used to confirm past diagnostic status.

Positive automatic thoughts. Positive thoughts were measured with the Automatic Thoughts Questionnaire-Positive (ATQ-P; Ingram & Wisnicki, 1988). The ATQ-P is a 30-item self-report instrument that measures the frequency of occurrence of positive automatic thoughts, or positive self-statements. Each item represents a thought and respondents rate the frequency of occurrence of this thought, or a similar thought, within the past week. Occurrence frequency is rated on a 5-point scale ranging from 1 (*not at all*) to 5 (*all the time*). Scores range from 30 to 150. Reliability and validity of the ATQ-P have been examined in numerous studies and are quite adequate (Ingram, Kendall, Siegle, & Guarino, 1995). Reliability in the present study was .79.

Negative automatic thoughts. Negative thoughts were measured with the Automatic Thoughts Questionnaire-Negative (ATQ-N; Hollon & Kendall, 1980). The ATQ-N is a 30-item self-report instrument used to measure the frequency of occurrence of automatic negative thoughts, or negative self-statements associated with depression. Like the ATQ-P, each item represents a thought and respondents rate the frequency of its occurrence on a 5-point scale (1 = *not at all*, 5 = *all the time*). The ATQ-N possesses good validity and reliability (Hollon & Kendall, 1980), with a reliability coefficient of .91 in the current study.

Coping style. The Revised Ways of Coping Checklist (WOC; Vitaliano, Russo, Carr, Maiuro, & Becker, 1985) was used to assess the ways that people tend to cope with stressful situations. The WOC is a 57-item checklist describing a broad range of behavioural and cognitive coping strategies that individuals might use during stressful episodes. Respondents are first asked to describe the most stressful event during the last month, and then to check several different coping strategy items on a 4-point scale ranging from 1 (*never used*) to 4 (*regularly used*). The WOC contains eight subscales: *Problem-Focused Coping* (15 items; e.g., “Made a plan of action and followed it”), *Wishful Thinking* (8 items; e.g., “Wished the situation would go away or somehow be finished”), *Avoidance* (10 items; e.g., “Refused to believe it had happened”), *Seeks Social Support* (6 items; e.g., “Talked to someone about how I was feeling”), *Blames Self* (3 items; e.g., “Realized you brought the problem on yourself”), *Blames Others* (6 items; e.g., “Took it out on others”), *Counting One’s Blessings* (6 items; e.g., “Focused on the good thing in my life”), and *Religiosity* (3 items; e.g., “Prayed about it”). Scores are obtained by summing the responses for each scale. Vitaliano et al. (1985) suggest adequate reliability and validity for all scales but the *Counting One’s Blessings* and *Religiosity* subscales. Reliabilities for each subscale ranges from .69 (*Counting One’s Blessings*) to .89 (*Problem-Focused Coping*)

Aggression. The Aggression Questionnaire (AQ; Buss & Perry, 1992) was used to examine several dimensions of anger and aggression. The AQ consists of 29 items that are divided into four subscales: *Physical Aggression* (9 items; e.g., “Once in a while I can’t control the urge to strike another person”), *Verbal Aggression* (5 items; e.g., “I tell my friends openly when I disagree with them”), *Anger* (7 items; e.g., “I flare up quickly but get over it quickly”), and *Hostility* (8 items; e.g., “I am sometimes eaten up with jealousy”). Items are answered on a 5-point scale ranging from 0 (*very uncharacteristic of me*) to 5 (*very characteristic of me*). Evidence of satisfactory validity and reliability are provided by Buss and Perry (1992). Reliability coefficients for each subscale were satisfactory (.82 to .90).

RESULTS

Data were analysed in two ways. In the first set of analyses, group comparisons were conducted to examine whether high- and low-risk groups differed on any of the variables. In the second set of analyses, regression equations were computed to assess potentially unique associations between the variables and risk status.

Risk group comparisons

Although gender was not included as a variable in the research design per se, it was nevertheless important to consider potential gender effects in the analyses. This seems particularly true for the hostility variables, since it seems reasonable to suspect that men and women may differ in their expression of at least some of these behaviours and emotional states (Buss & Perry, 1992; Graham & Wells, 2001). Before examining the variables, however, it was also important to examine potential differences in BDI scores. Even though research participants were only selected if they fell into the nondepressed range on the BDI, it was still possible that differences might emerge if one group was predominantly at the high end of this range and the other at the low end. A 2 (High-risk/Low-risk) \times 2 (Female/Male) analysis of variance on the readministered BDI found no significant effects for BDI scores assessing the past week (high-risk $M = 3.69$, low-risk $M = 3.31$). However, consistent with selection on the basis of a past episode of depression, a significant main effect was found for risk status on the BDI version covering the past year, with high-risk research participants evidencing higher BDI scores ($M = 5.41$) than low-risk participants ($M = 2.86$), $F(1, 103) = 5.84$, $p = .02$. No other significant effects were found. To ensure that results were not affected by differences in BDI scores for either time period, both BDI versions were used as covariates in subsequent analyses. In all cases, adjusted means are reported.¹

Automatic thinking. A 2 \times 2 analysis of covariance (ANCOVA) on ATQ-N scores found only a main effect for risk, $F(1, 99) = 4.01$, $p = .048$, with high-risk participants reporting more negative thoughts ($M = 55.34$, $SE = 2.04$) than low-risk participants ($M = 49.63$, $SE = 1.94$). For ATQ-P scores, a marginal effect was found for risk ($p = .09$), with a significant interaction between risk and gender, $F(1, 99) = 4.05$, $p = .047$. Interaction means are presented in Table 1. As can be seen from this table, the interaction was driven by high scores for males in the low-risk group.

¹ Analyses without BDI scores found an identical pattern of significant and nonsignificant results.

TABLE 1
 Mean positive automatic thoughts questionnaire scores as a
 function of risk and gender

<i>Risk</i>	<i>Gender</i>	
	<i>Male</i>	<i>Female</i>
High	103.94 (5.22)	107.67 (3.01)
Low	119.23 (4.58)	106.44 (3.40)

Aggression. A 2×2 ANCOVA on verbal aggression scores found a marginally significant effect for risk status ($p = .09$), but a significant effect for gender, $F(1, 99) = 7.96$, $p = .006$, with men reporting more verbal aggression ($M = 14.88$, $SE = 0.784$) than women ($M = 12.34$, $SE = 0.485$). The interaction was not significant. For physical aggression, the ANCOVA indicated a significant main effect for risk, $F(1, 99) = 16.39$, $p = .001$, with high-risk participants reporting more physical aggression ($M = 21.49$, $SE = 0.868$) than low-risk participants ($M = 16.57$, $SE = 0.827$). A significant effect was also found for gender, $F(1, 99) = 50.17$, $p = .001$, with men reporting more physical aggression ($M = 23.31$, $SE = 1.00$) than women ($M = 14.76$, $SE = 0.649$). For hostility, only a main effect was found for risk, $F(1, 99) = 6.94$, $p = .01$, showing that high-risk participants reported more hostility ($M = 18.81$, $SE = 0.778$) than low-risk participants ($M = 15.94$, $SE = 0.741$). No other effects approached significance. Finally, significant effects for risk, $F(1, 99) = 6.03$, $p = .016$, and gender, $F(1, 99) = 4.23$, $p = .043$, were found for anger. These results indicated that high-risk participants reported more anger ($M = 16.43$, $SE = 0.737$) than did low-risk participants ($M = 13.90$, $SE = 0.702$) and that men reported more anger ($M = 16.22$, $SE = 0.851$) than did women ($M = 14.11$, $SE = 0.551$).

Coping. For the “blames others” subscale, the ANCOVA indicated a significant main effect for risk, $F(1, 99) = 10.34$, $p = .002$, with high-risk individuals reporting more other-blame ($M = 13.79$, $SE = 0.57$) than low-risk participants ($M = 11.26$, $SE = 0.54$). No other effects were significant. For “self-blame” a risk main effect was also found, $F(1, 99) = 9.78$, $p = .002$, with high-risk participants reporting more self-blame ($M = 7.81$, $SE = 0.26$) than low-risk participants ($M = 6.69$, $SE = 0.24$). Again, no other significant effects were found. Similarly, for “wishful thinking” only a significant main effect for risk was found, $F(1, 99) = 5.03$, $p = .027$, with high-risk participants reporting more wishful thinking ($M = 21.80$, $SE = 0.66$) than low-risk participants ($M = 19.94$, $SE = 0.69$). On the avoidance subscale, a significant

TABLE 2
Mean social support seeking scores as a function of risk and gender

<i>Risk</i>	<i>Gender</i>	
	<i>Male</i>	<i>Female</i>
High	14.39 (0.78)	16.91 (0.46)
Low	15.83 (0.69)	15.52 (0.52)

risk main effect was found, $F(1, 99) = 6.72, p = .01$, showing that high-risk participants reported more avoidance ($M = 23.69, SE = 0.685$) than low-risk individuals ($M = 21.22, SE = 0.650$). Finally, for “social support seeking” only a marginal main effect was found for risk ($p = .08$), but a significant interaction between risk and gender emerged, $F(1, 99) = 5.16, p = .025$. Interaction means are presented in Table 2, which shows that the interaction is due to high-risk females reporting a greater tendency to seek social support, and high-risk males reporting less of this tendency. No other WOC subscales evidenced significant differences.

Regression analyses

We performed regression analyses on the automatic thinking, coping, and aggression scales to determine which variables might be unique predictors of risk. In each case, risk status was the criterion variable. In addition, BDI and gender were entered first to control for their effects. For ATQ scores the analysis indicated an adjusted r^2 of .191, $p = .002$, with only ATQ-N scores contributing significantly ($\beta = .41, t = 3.78, p = .002$).

For coping, the analyses indicated an adjusted r^2 of .173 that was significant, $p = .002$. Three coping variables emerged as significant predictors, blames self ($\beta = .39, t = 3.19, p = .002$), seeks social support ($\beta = .28, t = 2.12, p = .04$), and avoidance ($\beta = .33, t = 2.13, p = .04$). For the aggression scales, the analysis indicated a significant adjusted r^2 of .158, $p = .01$. Of the four aggression-scale variables, only anger ($\beta = .29, t = 2.15, p = .04$) and hostility ($\beta = .27, t = 2.11, p = .04$) emerged as significant predictors. To further understand the link between risk and these processes, these significant variables were entered into a regression analysis. This regression was significant, adjusted $r^2 = .392, p = .001$. Other than avoidance, all variables remained significant: ATQ-N ($\beta = .34, t = 4.14, p = .001$), hostility ($\beta = .29, t = 4.36, p = .001$), anger ($\beta = .16, t = 2.06, p = .04$), blames self ($\beta = .272, t = 4.26, p = .002$), and seeks social support ($\beta = .17, t = 2.10, p = .04$).

DISCUSSION

Several results emerged from these analyses. First, high-risk (formerly depressed) individuals and low-risk (never depressed) individuals differed on aspects of each of the three constructs examined. For automatic thinking, high-risk individuals reported more negative automatic thoughts than did low-risk participants. Additionally, an interaction emerged for positive thinking, with low-risk males evidencing the highest ATQ-P scores. Significant differences between risk groups were also found on the self-blame, other-blame, and avoidance scales, with a significant interaction on social support seeking. This interaction showed that high-risk women were the most likely to report using this strategy and high-risk men were the least likely. Social support, self-blame, and avoidance emerged in the regression analysis as significant predictors of risk status, although avoidance dropped out after further analyses. Finally, men reported more verbal and physical aggression than women, and high-risk participants reported more physical aggression than low-risk participants. Higher levels of anger and hostility were also found for high-risk participants.

Among these variables, dysfunctional automatic thinking can be considered a cornerstone of several cognitive approaches to depression (Beck, 1967; Ingram et al., 1998). Interestingly, some previous data have suggested that negative thinking may characterise the disordered state, but subsequently normalises in remission. For instance, in one of the first studies to assess the stability of cognitive measures in depression, Dobson and Shaw (1986) found evidence to suggest that negative thinking diminishes when depressed individuals enter remission. Interestingly, Dobson and Shaw found that ATQ-N scores dropped from 86.71 in the depressed state to 55.93 in remission; a mean that is remarkably consistent with the mean obtained for the high-risk individuals in the current study ($M = 55.34$). The current data thus suggest that although negative automatic thinking may diminish when depression remits, mild to moderate levels of elevation may remain for high-risk individuals when compared to low-risk individuals.

With regard to the aggression-scale scores, high- and low-risk individuals differed on all scales as, predictably, did men and women. However, in the regression analyses, which controlled for gender, anger and hostility emerged among these variables as the most consistent predictors of risk. Anger is known to characterise some depressed states, and in some cases hostility or irritation may be the primary symptom of depression rather than sad affect (Scott et al., 2003). However, the current data show that increased levels of anger and hostility may also characterise the vulnerable state. As such, because anger is largely an interpersonal variable, these data may provide a glimpse into interaction patterns that may be problematic, and may thus constitute some of the mechanisms of depression risk. In particular,

heightened levels of anger and hostility may provide fertile ground for conflicted interpersonal transactions that lead to relationship turmoil.

High-risk individuals' coping styles may further contribute to interpersonal difficulties and risk. It is unlikely that any particular coping strategy is inherently ineffective or dysfunctional. Yet, the particular combination of coping strategies displayed by high-risk individuals does suggest a strategy that is difficult to view as particularly effective. That is, although they reported engaging in problem-focused coping as much as low-risk individuals, high-risk individuals also endorsed a coping strategy that relies on self-blame, seeking social support, and to some degree avoidance. Along with increased anger and hostility, such findings can be seen as generally consistent with interpersonal approaches to depression. For instance, a key concept in interpersonal approaches is that depressed individuals seek reassurance and support, but that emergent interpersonal dynamics make the continuation of support difficult for others to maintain (Coyne, 1976, 1999; Joiner, 2002). Self-blame and avoidance of problems rather than seeking effective solutions, along with anger, hostility, and negative self-statements, would seem to be a combination of factors that would strain social relationships and render support-seeking not particularly effective.

Broadly in line with these factors, the current data may have some relevance for understanding some of the interpersonal sources of stress that vulnerable individuals experience. In this regard, Hammen (1991b, 1992, 1996) has advanced a stress-generation hypothesis about depression and suggested that depressed individuals may not only be the passive "victims" of stressful life events, but may play a role in creating at least some of the stress they encounter. Empirical data have supported this hypothesis in depression and have shown that depressed individuals engage in a variety of behaviours that appear to generate stress, such as poor problem solving and interpersonal incompetence (e.g., Daley, Hammen, Burge, & Davila, 1997; Flett, Hewitt, Garshowitz, & Martin, 1997; Potthoff, Holahan, & Joiner, 1995). The bulk of empirical support for this hypothesis has been obtained for individuals who are depressed. The current data extend support for this idea to risk for depression and highlight some possible pathways to the generation of stress. For example, heightened levels of anger and hostility may create problematic interactions that, if not creating stress, at least set the stage for stress. When combined with a tendency to blame oneself and to think negative thoughts, while also seeking social support and avoiding problem solving, these factors would appear to be likely candidates for creating and maintaining interpersonal dynamics that generate stressful environments.

Although gender differences were not the prime focus of the current study, two interactions emerged in the current data that may shed some light on these processes in depression risk. For instance, given a wealth of data on

gender differences in the incidence of depression (Nolen-Hoeksema, 2002), it seems reasonable to conclude that in this study low-risk men were the least vulnerable group. Interestingly, the interaction showed that this group endorsed the highest levels of positive thoughts. Several investigators (e.g., Ingram, Slater, Atkinson, & Scott, 1990; Lightsey, 1994, 1999) have suggested that enhanced scores on the ATQ-P may reflect a cognitive coping tendency that relies on positive self-statements in the face of negative mood provoking events. A possible protective or buffering mechanism for this group might thus be the tendency to think a greater number of positive self-relevant thoughts than other groups of people.

Social support seeking was the other variable that witnessed a gender by risk interaction, which demonstrated that high-risk women were the most inclined to seek out social support as a way to cope, and that high-risk men were the least likely. There is little doubt that, when used appropriately, social support seeking may be an effective coping method. However, interpersonal approaches suggest that an excessive reliance on seeking social support is at best a minimally effective short-term strategy, and at worst may exacerbate depression (Marcus & Nardone, 1992). Hence, if high-risk women rely “too much” on this process they may be inclined to suffer the coping setbacks of the sort featured by interpersonal models, whereas high-risk men may underutilise this process. Social support seeking might thus lead to different gender outcomes in depression, with women using a process that maintains or exacerbates depression, and men using a process that might lead to the social isolation that frequently accompanies depression. Moreover, because support seeking, unlike positive automatic thinking, must rely on the participation of other people to be effective, seeking social support as a strategy may leave some individuals, in particular high-risk women, to the vicissitudes of other’s behaviours. Thinking and coping processes may thus be relevant to understanding some of the interpersonal dynamics that lead to gender differences in depression.

The differences that were obtained, while significant, were not of large magnitudes. Such consistent but moderate differences would not be unexpected in an examination of risk groups who are not depressed. It may be, however, that small differences during the nondepressed state become magnified under stress so that small variations in these processes in the high-risk state may turn into larger and more virulent variations when stress becomes significant. For example, network models of depression suggest that dysfunctional cognitive structures emerge under stress and provide access to larger and more elaborate negative networks that lead to a cognitive spiral into depression (Ingram, 1984; Ingram et al., 1998; Teasdale & Barnard, 1993). Hence, an enhanced tendency to think negatively about the self when nondepressed may spiral into substantially higher levels of negative thinking when life events become intense.

Similarly, although modest elevations in anger and hostility may create problematic social environments that predispose to external strife and internal distress, these elevations may become exacerbated when difficult life events are encountered. In this regard, *social exits* are life events reflecting the dissolution of key supportive and attachment relationships, and have been found to be consistently related to the occurrence of depression (Brown & Harris, 1978, 1989; Monroe & Hadjiyannakis, 2002). If depression is occasioned by a social exit, individuals with a pre-existing tendency toward anger and hostility may become more angry and more hostile, both or either of which seem quite likely to interfere with effective problem resolution. Moreover, as already noted, high levels of anger and hostility may create problematic interpersonal dynamics that diminish prospects for continued social support, and more generally tax a coping style (self-blame and avoidance) that also becomes magnified under stress.

The current data are correlational and hence definitive conclusions about causality cannot be drawn. Casualty is a notoriously difficult process to verify, even in longitudinal studies (Ingram & Siegle, 2002). Thus, as with virtually all depression and depression-risk studies, any causal implications of these data must be considered speculative. Rather than providing strong conclusions about causality, the current data suggest several intriguing possibilities about some of the interpersonal and cognitive processes that may serve as links between stress and the onset of depression. Additionally, caution is always appropriate in the interpretation of self-report data, but there is reason to think that the measures used in the current study may reflect a reasonably accurate portrayal of the thoughts and behaviours in which high-risk individuals engage. That is, the patterns of data are not consistent with self-reporting biases in that the operation of such biases should lead at-risk individuals to respond more negatively on all measures. On the BDI, for instance, and consistent with the way risk was defined, high-risk individuals reported more depressive symptoms in the past year than low-risk individuals, but reports of depressive symptoms in the past week did not differ. Although high-risk participants did evidence higher scores than low-risk participants on all of the aggression scale scores, only anger and hostility were consistently related to risk status. Likewise, some but not all of the WOC scales differed, with no significant differences found for problem-focused coping, religiosity, or wishful thinking. If there are limitations to these data because of the use of self-report measures, they do not appear to be due to self-reporting biases.

In summary, the current data found that high-risk individuals differed in several respects from low-risk individuals. In the case of negative automatic thinking, anger and hostility, self-blame, avoidance, and social support-seeking, high-risk individuals evidenced a pattern of responses that might provide important clues about the way that risk processes are eventually

transformed into depression. The cross-sectional nature of these results suggests that these conclusions must be considered speculative, but these results do provide some intriguing clues about the role of variables that have not received much attention in the depression-risk literature.

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