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How Does Anger Coping Style Affect Glycemic Control in Diabetes Patients?

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Abstract

Background—Although various forms of anger have been found to influence the psychological and physical health in many chronic illness populations, little is known about the effects of anger in diabetes patients.

Purpose—Associations between anger coping style, diabetes-related psychological distress, and glycosylated hemoglobin (HbA_{1c}) were examined in 100 diabetes patients.

Method—Participants completed the Problem Areas in Diabetes and Coping Styles questionnaires, and had HbA_{1c} assessments at study entry (Time 1=T1), six months (T2), and 12 months after T1 (T3).

Results—Linear regression analyses revealed T1 anger coping associated with T3 HbA_{1c} ($\beta=.22$, $p<.05$) but T1 HbA_{1c} did not associate with T3 anger coping ($\beta=.13$, $p=NS$). After controlling for significant covariates (of gender, age, education, type and duration of diabetes), regression analyses revealed that T2 diabetes-related psychological distress partially mediated this association.

Conclusion—These results suggested that higher levels of anger coping may promote poorer HbA_{1c} in diabetes patients by provoking greater diabetes-related distress. Areas of future research on this topic are discussed.

Keywords

anger coping; psychological distress; glycemic control; diabetes

Understanding associations between psychosocial variables and physiological dysregulation is of great interest to health psychologists. Such associations not only help to establish mind-body interactions, but they are useful in designing interventions for persons with acute or chronic illness. For persons with diabetes, associations between psychosocial factors and metabolic control are particularly important as glycemia is critical in monitoring progression and control of disease.

To effectively manage one's diabetes, coping resources have been cited as an important mechanism in managing the high levels of diabetes-related stress that often accompanies maintaining proper glycemic control (Attari, Sartippour, Amini, & Haghighi, 2006; Peyrot, McMurry, & Kruger, 1999). This study explores the association of glycemia with anger coping style, a measure of anger and an emotion-focused coping style that has been shown to reliably differentiate individuals in terms of their chronic levels of glycemic control (Peyrot & McMurry, 1992; Peyrot et al., 1999).

Anger as a coping style may be of particular interest to persons with chronic illness as it assesses a method by which one may cope with daily stressors inherent to the illness. In cross-sectional research, anger coping style has been found to correlate with higher HbA_{1c} levels in diabetes patients (Peyrot & McMurry, 1992). While experimental diabetes research studies have revealed that a rise in insulin or inducement of hypoglycemia is accompanied by an increase in anger mood (De Sonnaville et al., 1998; McCrimmon, Ewing, Frier, & Deary, 1999; Merbis, Snoek, Kanc, & Heine, 1996; Weinger, Jacobson, Draelos, Finkelstein, & Simonson, 1995), anger as a more stable trait (e.g., coping style) and its directional association with glycemia may be valuable to treatment and prevention of poor glycemic control by adding to the psychological profile of those who may most benefit from intervention.

Importantly, previous research investigating the associations of anger and hostility with frank illness (e.g., coronary heart disease) has revealed the mediating role of distress (Sieglar, Peterson, Barefoot, & Williams, 1992). Psychological distress has been shown to be a significant mediator between the presence of a chronic stressor and physiological dysregulation (Vitaliano et al., 2002). In diabetes patients, evidence suggests that diabetes-related distress uniquely contributes to glycemic control (Polonsky, Anderson, Lohrer, Welch, & Jacobson, 1995; Sultan & Heurtier-Hartemann, 2001; Weinger & Jacobson, 2001) and greater distress has been found to associate with both poorer glycemic control and emotion-focused coping (Lustman, Frank, & McGill, 1991; Peyrot et al., 1999; Sultan & Heurtier-Hartemann, 2001). Further, prospective studies have reported emotion-focused coping as an indirect influence of future HbA_{1c} by increasing diabetes-related distress (among other behavioral and psychosocial variables) in type 2 patients (Nakahara et al., 2006). No study to our knowledge has specifically investigated the prospective association of any form of anger on distress or HbA_{1c} levels in a diabetes sample.

Thus, we employed a longitudinal design to examine associations between anger coping style, diabetes-related psychological distress, and glycemic control in diabetes patients. Our goal was to explore the association of anger coping style and glycemic control across time, with particular interest in the directionality of the association. Further, we explored diabetes-related psychological distress as a temporal mediator to this association.

Method

Participants

Persons with type 1 and type 2 diabetes between the ages of 18 and 75 were recruited by mail and/or during a medical appointment at the Joslin Diabetes Center (JDC) in Boston, MA. The JDC Committee on Human Studies approved the protocol and voluntary written informed consent was obtained from each participant before the study. The study was designed to investigate how psychosocial variables influence glycemic control and intended to inform the development of tailored interventions targeting those in poor glycemic control.

At their initial appointment, 145 patients completed a questionnaire packet and had their glycosylated hemoglobin (HbA_{1c}) percentage assessed. These participants were given the same battery three times over a one year period (T1 = baseline, T2 = approximately six months after

T1, and T3 = approximately 1 year after T1). Of the 145 patients who entered the study, 31 did not participate at T2 and an additional 13 did not participate at T3. These 44 participants never returned to the JDC for a clinic appointment within the study time frame. One patient had insufficient baseline data to be included in the study. Hence, the data for 100 patients were analyzed for this study. Attrition analyses are reported in more detail below.

Measures

Anger coping style (ACS) was measured using the three items from the anger coping subscale of the Coping Styles Questionnaire (CSQ) (Peyrot & McMurry, 1985; Peyrot et al., 1999; Wilson, Moore, Randolph, & Hanson, 1982). Participants were asked to respond to a four-point Likert scale ranging from “not at all like me” to “very much like me” indicating the extent to which the statement reflected their typical coping style. The CSQ, although diabetes nonspecific, was chosen as it was previously used in research on associations of coping styles, stress, and glycemic control in diabetes patients (Peyrot & McMurry, 1985, 1992; Peyrot et al., 1999). High scores indicate higher levels of anger coping (scale range 3–12) and have been associated with higher HbA_{1c} (Peyrot & McMurry, 1985, 1992). In the current sample, the Cronbach α for the anger coping subscale was 0.75.

Diabetes-related emotional distress was assessed by the Problem Areas in Diabetes Scale (PAID), a 20-item measure that assesses a broad range of feelings related to living with diabetes and its treatment, including depressed mood, worry, and fear (Polonsky et al., 1995). Patients were asked to respond on a five-point Likert scale (“not a problem” to “serious problem”) indicating the extent to which the feeling was currently a problem. High scores indicate higher diabetes-related distress (scale range = 0–100). The PAID has high internal consistency and validity (Cronbach’s α = 0.95, Polonsky et al., 1995; Welch, Jacobson, & Polonsky, 1997). A similar α (0.94) was found in the current study. The PAID is also sufficiently sensitive to change (Welch, Weinger, Anderson, & Polonsky, 2003).

Glycemic Control was assessed by the percent of glycosylated hemoglobin (HbA_{1c}). HbA_{1c} is the definitive measure of chronic glycemic control that is used in major clinical trials of diabetes (Diabetes Control and Complications Trial, 1993). The JDC laboratory was used for all assessments (high-performance liquid chromatography ion capture method; Tosch Medics, San Francisco, CA; reference: 4.0–6.0%). These methods conform to the Diabetes Control and Complications Trial Research Group (1993) standardized methods.

Statistical Analyses

Pearson correlation coefficients were estimated to examine associations between ACS, PAID, and HbA_{1c} at each time point and at all combinations of assessments across time. Stability of variables was explored using intraclass correlation coefficients for single measures and mean changes were assessed using repeated measures analysis of variance.

Attrition analyses were conducted to compare groups that completed the study to those that dropped out at T2 or T3. Analyses of variances (and independent-samples *t*-tests) were used to test for differences in demographic variables, anger coping style, distress, and glycemic control between the groups. In addition, the direction and magnitude of correlations between completers and non-completers on key variables were explored.

Linear regression analysis was used to examine the association between anger coping and HbA_{1c}. Age and years of education were included in the final models as they were associated with the dependent variables of these analyses. Type of diabetes, duration of diabetes, and sex were also tested but not included in the final models as they did not associated with HbA_{1c} or

ACS at T3. These covariates were chosen and examined *a priori* as they represent demographic and clinical variables that may influence the association of HbA_{1c} on psychosocial variables.

Complete mediation was assessed as per Baron and Kenny's guidelines (1986). In analysis of T3 HbA_{1c}, variables were entered using the following steps: step 1, significant covariates; step 2, anger coping style at T1; step 3, diabetes-related psychological distress at T2. Standardized beta weights and associated *p*-values are reported in the text. T1 HbA_{1c} was included in step 1 in analyses of change across time.

Results

Demographic, Psychosocial, and Clinical Variables

Data for the demographic, psychosocial, and clinical variables of the 100 participants with complete data assessed at baseline are given in Table 1. Overall, men (*n*=44) and women (*n*=56) did not differ on metabolic or psychosocial variables, with the exception of diabetes-related psychological distress at T1, where women were higher (*M*=38.1, *SD*=23.9 vs. *M*=25.0, *SD*=16.6 for men; *p*<.01). However, this difference was not statistically significant at T2 or T3.

Differences in psychosocial and metabolic variables were also tested between type 1 (*n*=63) and type 2 (*n*=37) patients. HbA_{1c} at T2 was the only differentiating variable between the groups, with type 1 patients in worse control at T2 (8.0 ± 1.5 versus 7.4 ± 1.4 ; *p*<.05). As such, all patients were considered together; however, type of diabetes was tested as a potential covariate in all models.

The stability of mean scores on anger coping style, diabetes-related distress, and HbA_{1c} were moderately high (intraclass correlation coefficients: ACS = .74, PAID = .83, HbA_{1c} = .72). Repeated measures analyses of variance revealed that the mean scores for ACS and PAID decreased across time (*p*'s<.01) while the mean HbA_{1c} levels did not change. Table 2 provides intercorrelation coefficients of the variables across all time points, and also shows the key variables' association with age and years of education, as they were the only covariates associated with T3 HbA_{1c} and therefore used in the final models of the regression analyses.

Attrition Analysis

Because the attrition rate was significant, comparisons were made between those who completed all three assessments and those who did not. In terms of means and percentages, those who did not return after baseline (*n*=31) did not differ on any demographic, psychosocial or clinical variables at T1 from the completers (*n*=100; all *p*'s = NS). While correlation coefficients at baseline for ACS-PAID and PAID-HbA_{1c} were similar in magnitude and direction for completers and non-completers, completers showed a higher correlation between ACS and HbA_{1c} (*r*=.28, *p*<.01) than non-completers (*r*=.05, *p*=NS).

Associations between Anger Coping Style and Glycemic Control

T1 anger coping associated with HbA_{1c} at T3 ($\beta = .22$, *p*<.05), but the reversal of these regressions showed that T1 HbA_{1c} did not associate with T3 anger coping ($\beta = .13$, *p*=NS). As age and years of education were covariates that associated with HbA_{1c}, they were included in the final models.

Tests of Temporal Mediation

Diabetes-related distress (PAID) was tested as a temporal mediator of the association between T1 anger coping and T3 HbA_{1c}. To fulfill the requirements of temporal mediation, each variable at its hypothesized time point (anger coping at T1 and diabetes-related psychological distress

at T2) was entered into the equation to examine whether it was associated with glycemic control at T3. Table 3 illustrates that T1 anger coping predicted T3 glycemic control when T2 PAID was not entered in the regression equation ($\beta = .20, p < .05$), but the beta was reduced to more than half when PAID was in the equation ($\beta = .08, p = \text{NS}$). Simultaneously, the standardized beta for PAID was significant ($\beta = .26, p < .05$) and anger coping style at T1 predicted T2 PAID ($\beta = .47, p < .001$). Table 3 also shows the effect of age in the prediction of T3 HbA_{1c} with and without T2 PAID in the equations.

Predicting Change in HbA_{1c}

To test whether anger coping at T1 predicted change in HbA_{1c} across time, T1 HbA_{1c} was entered into the regression equation at Step 1. Anger coping at T1 did not predict increases in HbA_{1c} ($\beta = .08, p = \text{NS}$). However, the two other pathways remained significant with T1 HbA_{1c} in the final model: T1 anger coping predicted T2 diabetes-related distress ($\beta = .48, p < .001$), and T2 PAID predicted worse T3 HbA_{1c} ($\beta = .25, p < .01$). No covariates were significant beyond the effect of T1 HbA_{1c}. Hence, these results indicated that anger coping at T1 did not directly predict worsening glycemic control from T1 to T3, but it did so indirectly via T2 PAID. Interestingly, diabetes-related distress at T2 predicted worsening glycemic control across time.

Discussion

When faced with a chronic illness such as diabetes, ineffective coping strategies may have severe negative consequences on both mental and physical well-being. This study investigated the associations between anger coping style, diabetes-related distress and glycemic control in diabetes patients. In light of preliminary reports suggesting trait forms of anger may be a risk factor for diabetes (Golden et al., 2006) and the long-standing knowledge of the effect of poor coping on distress and HbA_{1c} levels in these patients (Attari et al., 2006; Peyrot et al., 1999), this study demonstrates how anger coping style may negatively influence glycemic control in diabetes patients.

One advantage to our study design was the ability to investigate whether anger coping better associated with future HbA_{1c} levels, or visa versa. We found that anger coping associated with future HbA_{1c}, while the reverse was not true. This finding lends much promise to the potential usefulness of anger-management interventions aimed at bettering diabetes-related outcomes, particularly given the success of other types of psycho-educational interventions in diabetes health (e.g., Gallegos, Ovalle-Berumen, & Gomez-Meza, 2006; Welch et al., 2003). Further, given that diabetes-related distress was found to partially mediate this association, cognitive behavioral or psychoeducational skill-training interventions in anger management may be useful for reducing diabetes-related distress in patients who frequently use anger as a means for coping and reacting to their disease. Similar treatment techniques have been used successfully in women caregivers of a relative with dementia, where in addition to reducing anger and increasing self-efficacy, positive coping strategies increased in those who participated in the anger management arm (Coon, Thompson, Steffan, Sorocco, & Gallagher-Thompson, 2003). Future research would benefit from the continued assessment of anger as a coping strategy and the impact of anger-management as a means to improve quality of life and physical health in the diabetes population.

Although anger coping at T1 associated with HbA_{1c} levels one year later, our data did not support a prediction of a change in HbA_{1c} levels. This finding may be explained in part by the fact that HbA_{1c} did not change significantly over the course of the year. Perhaps one year was not sufficient time to elucidate the long-term effects of anger coping on changing HbA_{1c} in an observational study. Further, these results may illustrate the point that HbA_{1c} is difficult to change, even in a group of research participants who are regularly seeking treatment. In fact, it is not uncommon for HbA_{1c} to remain unchanged even after educational or psychosocial

interventions as assessed in periods of less than one year (Bradshaw et al., 2007; Moreland et al., 2006; Thomas & Miceli, 2006). Despite this, our study showed that higher T1 anger coping predicted greater T2 diabetes-related distress, and greater T2 diabetes-related distress predicted increases in HbA_{1c}, even after controlling for T1 HbA_{1c}. Thus, these results suggest that diabetes-related psychological distress is a critical variable in predicting change in glycemic control over time.

These results are consistent with known mechanisms that may help to explain the association between distress and glycemic control including elevated stress hormones such as corticotropin-releasing factor and cortisol, and poorer health habits such as overeating and inactivity. These outcomes may result from higher distress levels and poorer health habits, as research in non-diabetics has found that these independently predict factors associated with insulin resistance (see Vitaliano et al., 2002 for a discussion). Regimen adherence and self-efficacy have also been found to mediate distress and glycemia and may be useful in future investigations (Nakahara et al., 2006; Peyrot et al., 1999; Stewart et al., 2003).

Age was an important covariate in these analyses, as not only was it associated with HbA_{1c} and anger coping style, but it was also a significant contributor to the association of anger coping and HbA_{1c} (Tables 2 and 3). These findings corroborate existing research showing both anger and HbA_{1c} tend to decrease with age (Diabetes Control and Complications Trial/ Epidemiology of Diabetes Interventions and Complications Study Research Group, 2007; Gross et al., 1997; Phillips, Henry, Hosie, & Milne, 2006; Schieman, 1999). Future investigations may benefit to explore the mechanisms of these associations, which may include increased adaptive coping, self-care patterns, or acceptance.

Despite the fact that the mediation model satisfied the criteria for temporal mediation as put forth by Baron and Kenny (1986), we acknowledge that temporal priority is a necessary, but not sufficient, condition for causality and we cannot be certain that the order of the variables over the limited timeframe is inflexible. Other limitations include the significant attrition rate over the course of this study. Although there were no differences between completers and non-completers in means and percentages for the key variables, we did find that completers had a higher correlation between anger coping style and HbA_{1c} than those who did not complete all assessments. This correlation may have over-inflated the association between anger coping and glycemia that we found in our study sample. Further, our assessments did not include treatment type, beyond the distinction between type 1 and type 2 diabetes. Exogenous insulin was not specified in the models tested, but this variable may have elucidated differences between insulin users and non-insulin users. Additionally, a primarily Caucasian ethnic makeup limits the extent to which our results may be applicable to other ethnic groups. Thus, future research is warranted to insure the generalizability of our findings.

Despite these limitations, the current results suggest that anger coping style may influence diabetes-related distress and that this distress may, in turn, influence HbA_{1c} in diabetes patients struggling with glycemic control. Previous research has shown anger, anxiety, and distress to be associated with increased physical illness, immune deficiencies, cholesterol, cardiovascular disease and death (Suinn, 2001). In diabetes, these negative emotions have shown to be associated with mental functioning and accuracy of blood glucose symptom perception (Paschalides et al., 2004; Wiebe, Alderfer, Palmer, Lindsay, & Jarrett, 1994). Our study aimed to elucidate the association between negative emotions and mental and physical health in persons with diabetes by exploring the association of anger coping with both mental and physiological markers. Continued investigation of anger coping as a vulnerability factor for distress and glycemic control in diabetes would be useful to inform interventions, aid clinical practice, and improve overall mental and physical health in those living daily with diabetes.

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Table 1

Means (*M*) and Standard Deviations (*SD* or %) for Demographic, Clinical, and Psychosocial Variables at Time 1, n=100

Variables	(<i>M</i> ± <i>SD</i>) or (%)	Range
<i>Demographic Characteristics</i>		
Age	49.1 ± 15.8	18–75
Duration (years)	19.1 ± 13.2	1–51
Education (years)	15.5 ± 2.9	8–20
Sex (female)	56	
Ethnicity (Caucasian)	90	
Type of diabetes (Type 1)	63	
<i>Psychosocial and Clinical Variables</i>		
Anger Coping Style	6.63 ± 2.29	3–12
Diabetes-related Distress	32.32 ± 21.88	1.25–90
HbA _{1c} (%)	7.92 ± 1.41	4.6–13.2

Table 2

Intercorrelation Coefficients Between Anger Coping Style, HbA_{1c}, Diabetes-related Distress, and Key Covariates Across Time

	1	2	3	4	5	6	7	8	9	10	11
1. T1 ACS	--	.74***	.71***	.54***	.49***	.45***	.28**	.25*	.27**	-.22*	-.08
2. T2 ACS		--	.77***	.49***	.45***	.46***	.20	.08	.20	-.28*	.05
3. T3 ACS			--	.40***	.46***	.43***	.18	.15	.22*	-.24*	.04
4. T1 PAID				--	.83***	.84***	.20*	.28**	.27**	-.15	-.08
5. T2 PAID					--	.86***	.17	.29**	.36***	-.17	-.09
6. T3 PAID						--	.10	.24*	.29**	-.18	.01
7. T1 HbA _{1c}							--	.73***	.71***	-.29**	-.14
8. T2 HbA _{1c}								--	.79***	-.35***	-.22*
9. T3 HbA _{1c}									--	-.33**	-.21*
10. Age										--	.06
11. Education											--

Note. ACS = Anger Coping Style; PAID = Problem Areas in Diabetes Scale; T1 = time 1, T2 = time 2, T3 = time 3.

* $p < .05$.

** $p < .01$.

*** $p < .001$

Table 3
Regressions of Time 3 HbA_{1c} on Time 1 Anger Coping Style (ACS) and Time 2 Diabetes-Related Distress (PAID)

Without PAID				With PAID			
Step	Variable	Unst. β	Stand. β	Variable	Unst. β	Stand. β	
1	Age	-0.02	-0.28**	Age	-0.02	-0.26**	
	Education	-0.08	-0.18	Education	-0.07	-0.17	
	ACS	0.12	0.20*	ACS	0.05	0.08	
2							
3	-	-	-	PAID	0.02	0.26*	

Note. Without distress: $F(3, 94) = 6.94^{***}$, $R^2 = .18$. With distress: $F(4, 93) = 6.96^{***}$, $R^2 = .23$.

* $p < 0.05$,
** $p < 0.01$,
*** $p < .001$.