


Causal attributions following a cardiac event: Short- and long-term differences in health appraisals and outcomes

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Abstract

This study examined attributions generated by cardiac rehabilitation patients shortly after experiencing a cardiovascular event, exploring whether attribution type was associated with health appraisals and outcomes concurrently and 21 months later. Attributions fell into three categories: controllable behavioral ones, uncontrollable biological ones, and stress-related causes. Linking attribution type to appraisals and outcomes showed that creating a behavioral attribution was beneficial in the short-term for control appraisals, but was associated with increased anxiety symptoms 21 months later. Thus, cardiac rehabilitation providers should encourage patients to maintain a future-focus that promotes perceived control over health promotion behaviors that reduce risk for recurrence.

Keywords

beliefs, heart, outcomes, psychological distress, social cognitions

The creation of meaning to cope with trauma is seemingly universal. In her influential piece on cognitive adaptation, Shelley Taylor (1983) outlines steps commonly taken in the wake of trauma: the generation of meaning, the bolstering of perceived control, and self-enhancement. The first step in this cognitive process, the creation of meaning, is often accomplished through the generation of a causal attribution. Empirical evidence supports the process of meaning creation via causal attributions among patients diagnosed with various forms of cardiovascular disease (CVD). For example, Cameron et al. (2005) found that the most common causal attributions endorsed by myocardial infarction (MI) patients via a checklist method were stress, high cholesterol, heredity, and eating fatty foods, and that these attributions remained stable over a 6-month follow-up period. Another study found not only similar types of attributions generated by MI patients at baseline (i.e. smoking, heredity, and stress) but also some volatility in endorsement over time (Reges et al., 2011). Specifically, at 2-year post-hospitalization, more patients endorsed high cholesterol, lack of physical activity, and problems at work as causes than at baseline. Day et al. (2005) found that the most commonly endorsed attributions among CVD patients via a checklist were heredity, hypertension, high

cholesterol, physical inactivity, and poor food habits. Furthermore, nearly one-third of the sample endorsed stress/negative emotions as a causal factor in their diagnoses. A more recent study among coronary artery bypass graft (CABG) patients that used an attribution checklist found the most common causes to be stress and genes for both men and women (Dunkel et al., 2011). Results also pointed to a gender difference: whereas men were more likely than women to make an attribution to past behavior, women were more likely than men to attribute their diagnosis to destiny.

The above-mentioned studies examined attributions generated via a checklist, but other researchers have collected patients' open-ended causal explanations. For example, Martin et al. (2005) qualitatively analyzed MI patients' attributions, collapsing them into the following commonly

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cited categories: stress, comorbidities, diet, smoking, heredity, and physical inactivity. Comparisons by gender showed that men were significantly more likely to generate controllable behavioral attributions compared to women. This gender difference translated to behavioral disparities 3 months later, as men were more likely to self-report improvements in diet and exercise habits compared to women. Astin and Jones (2004) qualitatively examined CVD patients' causal attributions, collapsing them into three main categories: biological causes, behavioral causes, and stress. Results revealed gender differences, such that women were more likely than men to generate biological and stress attributions, but men were more likely than women to create behavioral attributions. The same three attributional themes were found by Bennett and Marte (2013) among cardiac rehabilitation (CR) patients, but these authors reported no gender differences in biological or stress attributions and only a trend for men to create more behavioral attributions than women. Therefore, ample evidence exists that CVD patients engage in a causal search following a cardiac event, and that these attributions typically fall into three categories: controllable behavioral causes, uncontrollable biological causes, and stress (e.g. Stafford et al., 2008).

Researchers have begun to more deeply investigate how these three types of attributions affect physical and psychological recovery. Two studies suggest adaptive effects on recovery from the creation of *behavioral or modifiable/controllable causal attributions*. Blair et al. (2014) studied patients' attributions for their cardiac events, using them to predict attendance at CR. Results showed that patients who created attributions to causes that were controllable (e.g. diet and physical inactivity) were more likely to attend CR than patients who generated uncontrollable attributions, even after controlling for sociodemographic variables such as sex and distance to the nearest CR site. An intervention study by Broadbent et al. (2009) aimed to strengthen controllable behavioral attributions among patients post-MI. Results showed that the intervention group endorsed high cholesterol and physical inactivity to greater degrees post-intervention compared to the control group. Furthermore, these authors suggest that their attributional retraining was adaptive because the intervention group was more likely to return to work at the 3-month follow-up compared to the control group.

Whereas support has been found for behavioral attributions assisting in recovery, the effects of *stress attributions* on adjustment are mixed. On one hand, Dunkel et al. (2011) found that CABG patients who endorsed stress and personality causal attributions at baseline had a worsening of depressive symptoms over a 1-year period, after controlling for a host of sociodemographic and clinical factors. On the other hand, Bennett and Marte (2013) reported that baseline stress attributions were associated with improvements in a measure of energy expenditure over the course of CR, and that self-reported engagement in healthy behaviors mediated the

relation. Furthermore, little work has investigated the effect of *biological attributions* on adjustment and recovery. One study was located that examined a sample of older adults with chronic health conditions, the most common of which was heart disease (42%; Stewart et al., 2012). Results showed that endorsing an "old age" attribution (i.e. a biological one that is uncontrollable) for one's chronic health condition was associated with poor self-reported health, poor engagement in health behaviors (e.g. eating a nutritious diet), and a greater likelihood of death at the 2-year follow-up (36% vs 14%).

Hypothesis and research questions

This study was conducted to examine whether (a) attributions generated by a sample of CR patients align with previous research, (b) gender differences exist in the creation of these attributions, and (c) these attributions have effects on health appraisals and outcomes. Prior studies suggest that patients' attributions fall into three main categories: behavioral causes that are largely controllable (e.g. lack of exercise and poor diet), biological causes that are largely uncontrollable (e.g. heredity), and interpersonal stressors. These categories have emerged across studies that use open-ended questions and checklist formats, hence

Hypothesis 1. Participants' attributions will fall into three main categories: behavioral ones that are controllable, biological ones that are uncontrollable, and stress-related causes.

Given gender differences documented in the literature, we also hypothesized

Hypothesis 2. Men will be more likely to create behavioral attributions than women, whereas women will be more likely than men to create biological and stress attributions.

Because evidence is mixed on the adjustment/recovery effects of creating behavioral, biological, and stress attributions, we did not test specific hypotheses. Rather, we explored whether these types of attributions result in differences in concurrent and prospective health appraisals and outcomes through two research questions, specifically

Research Question 1. Are there concurrent differences in health behaviors, perceptions of control, and psychological and physical health status as a function of the type of attribution created at the beginning of CR?

and

Research Question 2. Are there long-term differences in health behaviors, perceptions of control, and

psychological and physical health status as a function of the type of attribution created at the beginning of CR?

Method

Participants

Baseline (i.e. Time 1) data were collected from 209 patients (65.6% male) enrolled in a CR in a Midwestern state. Ages ranged from 25 to 85 years, with an average age of 62.5 years (standard deviation (*SD*)=11.1 years). A vast majority of the sample reported their ethnic background to be European American (91.8%). Other ethnicities represented in the sample include African American (5.3%) and Native American (1.9%). Although most participants were married or living with a partner (73.2%), 12.4 percent reported being divorced, 9.1 percent were widowed, 3.8 percent were single and had never married, and 1.4 percent was separated from their partners at the time of the study. Most participants had completed high school or attended a college or trade school (47.9%), whereas 11.6 percent held a 2-year college degree, 22.2 percent held a 4-year college degree, and 15.9 percent completed a graduate degree. A majority of participants reported not working outside the home (58.7%), and the average household income range for the previous year was between US\$50,000 and US\$59,999.

Participants in this study varied widely in the diagnosis for which they were referred to CR. The most common diagnosis, for which 39.0 percent were referred, was the placement of a stent. The other most common diagnoses were CABG (17.6%), MI with the placement of a stent (13.8%), and valve replacement or repair (9.0%). All participants were stratified by risk for disease progression on the basis of the American Association of Cardiovascular and Pulmonary Rehabilitation (2004) guidelines. Risk stratification assignments of low (59.4%), moderate (36.1%), or high (4.4%) were made by CR staff and based on participants' diagnoses, prior cardiac events if appropriate, and current risk factors (e.g. smoking, concurrent ailments, and diet). Participants attended an average of 16.1 exercise sessions (*SD*=7.1) during their 12-week CR programs.

Follow-up data were collected from 112 participants (53.6%) 21 months later. The follow-up sample was 67.0 percent male with an average age of 63.7 years (*SD*=10.2 years), 91.9 percent were European American, 76.8 percent were married or living with a partner, 43.8 percent had completed high school or attended a college or trade school, most (i.e. 58.6%) did not work outside the home, and the average household income range for the previous year was between US\$60,000 and US\$69,999. A series of chi-squares and independent sample *t*-tests were conducted to determine whether the follow-up sample differed from the baseline sample on any

demographic or study variable. Results showed that the follow-up sample was older ($M=63.68$), better educated ($M=5.00$), and had lower risk stratifications for disease progression ($M=1.36$) than the full baseline sample ($M_s=60.59, 4.45, \text{ and } 1.56$, respectively). Therefore, some caution is warranted in generalizing prospective findings to the full sample.

Procedure

Participants were recruited through a Phase II CR program in a Midwestern city. Phase II CR programs typically run for 12 weeks and are primarily comprised of monitored exercise classes. Recruitment for the study occurred in two phases. First, CR staff members summarized the study's objectives and procedures during introductory, intake interviews with new patients. If patients provided preliminary written consent, their contact information was forwarded to our research team. The second phase of recruitment occurred when our research team members called these interested patients within 1 week of their intake interviews. During these phone calls, more detailed information about the study was provided to patients, and if they expressed interest, a study packet with a consent form, Time 1 questionnaire, and postage-paid return envelope was mailed to them. Research team members communicated to prospective participants that they were not obligated to participate if they received a study packet by mail. If, after reading through the materials, a patient consented to being in the study, he/she was asked to sign the consent form and return it, along with the completed questionnaire, in the enclosed envelope. Follow-up questionnaires (i.e. Time 2) were mailed to participants 21 months later, approximately 18 months following completion of CR. Participants returned their completed Time 2 questionnaires to us in postage-paid envelopes. All study procedures were approved by the appropriate hospital and university institutional review boards.

Measures

Questionnaires comprising several standardized measures, as well as items specifically designed for this study, were used at Time 1 and Time 2.

Cardiac attributions. At Time 1, using an open-ended question, participants were asked what they believed to be the main cause of their cardiac events: "If you had to pick one major cause for your heart condition, in your own words, what would that cause be?" Participants were provided a space in which to write their answers. The use of a single, open-ended question to assess causal attributions for cardiac events is supported by results of French et al. (2005), who found that although MI patients can identify multiple

possible causes, most focused on a single cause for their cardiac events.

Cardiac symptom experiences. Patients' experiences of cardiac-related symptoms were assessed with three questions adapted from Rose (1962) at Time 1 and Time 2. Participants were asked how many times during the preceding 2 weeks they suffered from three common CVD-related symptoms: (a) pain in chest, (b) pressure or heaviness in chest, and (c) shortness of breath. Responses were made on a 5-point scale (1 = *never* and 5 = *more than 15 times*) and summed, with high scores indicating frequent experience of symptoms.

Engagement in health-promoting behaviors. Participants' engagement in healthy behaviors was assessed with six items adapted from Naslund and Fredrikson (1993) at Time 1 and Time 2. Items measured how often during the past month participants engaged in a variety of healthy and unhealthy behaviors (e.g. eating red meat, fruits, and vegetables and engaging in light or moderate intensity workouts) on a 7-point scale (1 = *never* and 7 = *more than once a day*). Responses were summed so that higher values reflect engagement in more healthy behaviors.

Control appraisals. Perceptions of control were measured with two items adapted from Bennett et al. (2005) that focus on CVD-related *recovery* and *prevention*: "In general, how much personal control do you think you have in recovering from your cardiac event?" and "How much personal control do you think you have in preventing another cardiac event?" Responses to these two questions were made on a 4-point scale (1 = *very little* and 4 = *total*) at Time 1 and Time 2.

Symptoms of anxiety. Participants completed the Beck Anxiety Inventory (BAI; Beck and Steer, 1990) to measure the degree to which they experienced symptoms of anxiety at Time 1 and Time 2. The BAI presents 21 common symptoms of anxiety such as difficulty breathing and hands trembling. Respondents were asked to report the frequency they experienced these symptoms along a 4-point scale (0 = *not at all* and 3 = *severely*). Scores can range from 0 to 63, with high scores reflecting high symptoms of anxiety.

Symptoms of depression. Depressive symptoms were measured with the Beck Depression Inventory-II (BDI-II; Beck et al., 1996) at Time 1 and Time 2. The BDI-II asks participants to rate their degree of agreement with 21 items tapping symptoms of depression such as sadness, loss of pleasure, and fatigue. Respondents rated their degree of agreement with these items along a 4-point scale (0 = *I do not feel sad* and 3 = *I am so sad or unhappy that I can't stand it*). Possible scores range from 0 to 63, with high scores reflecting high depressive symptoms.

Results

Table 1 provides descriptive statistics, coefficient alphas (where appropriate), and correlations for all study variables.

Hypothesis 1: content analyses of attributions

The sample of 209 participants generated 244 different attributions for their cardiac events. Seven participants left the attribution question blank and were excluded from the analyses presented below. Of the remaining 202 participants, most (168; 83%) generated only one attribution for their cardiac events; however, 26 participants generated two attributions (13%) and eight participants generated three attributions (4%). In order to test Hypothesis 1 (i.e. that attributions will fall into behavioral, biological, and stress themes), content analyses of these attributions followed a multi-step process. First, four coders reviewed a random sample of 25 percent of the attributions, devoid of any identifiers, in order to develop a set of common themes (or coding categories). Individually, coders generated a list of themes into which the random sample of attributions fell. Second, a group meeting was held wherein themes were compared, discussed, and amended, resulting in a set of 13 agreed-upon coding categories. Third, the four coders independently assigned another random sample of 25 percent of the attributions into these 13 coding categories. Fourth, a meeting was held to discuss the codings, resolve any discrepancies, and revise the coding categories if needed. Consensus was reached on the assignment of attributions to the coding categories, and no revision to those coding categories was necessary. Fifth, the four coders individually reviewed and assigned the remaining 75 percent of the attributions into the coding categories. Sixth, a group meeting was held wherein the coders discussed and resolved all discrepancies. Across all attributions, coders agreed 91 percent of the time, yielding a free marginal kappa of .90.

Table 2 provides the 13 coding categories, the number of attributions assigned to each category, and examples from each coding category. Results showed that the most common type of attribution generated by participants was hereditary (25.8%), followed by poor diet (18.0%), stress (10.2%), and poor self-care (9.8%). Consistent with Hypothesis 1, these 13 categories can be further collapsed into three broad causal themes found in the literature: (a) behavioral (controllable) causes (poor diet, poor general self-care, lack of exercise, smoking, and obesity); (b) biological (uncontrollable) causes (heredity, non-cardiac-related conditions, cardiac-related conditions, and age); and (c) stress. The personality and miscellaneous categories ($n=15$) were dropped from subsequent analyses. For the current sample, 92 participants (45.5%) generated a behavioral cause, whereas 90 participants (44.6%) created a biological cause, and 24 participants (11.9%) believed stress to be a cause. A

Table 1. Descriptive statistics, coefficient alphas, and correlations for all study variables.

	1	2	3	4	5	6	7	8	9	10	11	12
1. Healthy behaviors, T1	–	.03	–.12	–.13	–.28*	–.32*	.45*	.09	–.06	–.15	–.21*	–.26*
2. Recovery control, T1		–	.64*	–.31*	–.32*	–.36*	.12	.49*	.25*	–.20*	–.18	–.33*
3. Prevention control, T1			–	–.28*	–.24*	–.28*	.10	.32*	.26*	–.20*	–.12	–.27*
4. Cardiac symptoms, T1				–	.55*	.48*	–.21*	–.28*	–.20*	.58*	.40*	.49*
5. Anxiety symptoms, T1					–	.77*	–.23*	–.29*	–.19	.36*	.56*	.67*
6. Depressive symptoms, T1						–	–.26*	–.34*	–.25*	.39*	.54*	.76*
7. Healthy behaviors, T2							–	.10	.15	–.14	–.19	–.29*
8. Recovery control, T2								–	.53*	–.22*	–.29*	–.27*
9. Prevention control, T2									–	–.41*	–.29*	–.33*
10. Cardiac symptoms, T2										–	.53*	.49*
11. Anxiety symptoms, T2											–	.62*
12. Depressive symptoms, T2												–
N	200	201	201	202	194	184	109	110	110	110	109	103
M	23.20	3.03	2.75	5.31	8.16	9.87	26.78	2.92	2.77	4.65	7.42	8.80
SD	5.30	0.65	0.74	2.42	7.74	8.98	4.81	0.66	0.69	2.36	7.59	7.72
Coefficient alphas	.65	–	–	.67	.89	.93	.60	–	–	.82	.91	.91

Probabilities are expressed only to $p < .05$ level; * $p < .05$.

Table 2. Coding categories and examples of attributions assigned to each category.

Coding category	Examples
Heredity ($n = 63$)	“Both parents had heart disease” “Family history” “Genetics”
Poor diet ($n = 44$)	“Bad eating” “Eating unhealthy everyday”
Stress ($n = 24$)	“Stress brought on by myself and others” “Stress, work and home life”
Poor general self-care ($n = 24$)	“Not taking proper care of myself” “Not living right”
Lack of exercise ($n = 19$)	“Sedentary lifestyle” “Lack of proper exercise”
Smoking ($n = 17$)	“Smoking while inactive (driving truck)”
Non-cardiac-related conditions ($n = 12$)	“Sleep apnea” “42 years as an insulin dependent diabetic, type 1”
Cardiac-related conditions ($n = 10$)	“Infection around valve to heart” “Poor circulation, clogged arteries”
Obesity ($n = 8$)	“Obesity” “Somewhat overweight”
Age ($n = 7$)	“Getting older”
Personality ($n = 4$)	“My intensity”
Miscellaneous ($n = 11$)	“Overdue” “Birth defect”
Blank ($n = 7$)	

total of 11 participants (5.4%) created both behavioral and biological attributions.

Hypothesis 2: gender differences

Chi-square analyses were conducted to test whether men created more behavioral attributions than women, and whether

women created more biological attributions than men. Because very few participants created stress attributions, gender comparisons were not tested. Results revealed no gender differences. Men were just as likely as women to make behavioral attributions ($\chi^2(1, n=202)=2.49, p=.11$), while women were just as likely as men to make biological attributions ($\chi^2(1, n=202)=0.16, p=.68$). Thus, Hypothesis 2 was not supported.

Table 3. Mean comparisons of outcomes by attribution categories.

	Behavioral/controllable cause		Biological/uncontrollable cause	
	Yes	No	Yes	No
Time 1 outcomes (at the beginning of CR)				
Engagement in healthy behaviors ($n=200$)	21.78***	24.37	24.21*	22.38
Recovery control appraisals ($n=201$)	3.17**	2.91	2.94	3.10
Prevention control appraisals ($n=201$)	2.95**	2.59	2.60**	2.87
Cardiac symptom experiences ($n=192$)	5.14	5.45	5.33	5.29
Symptoms of anxiety ($n=194$)	8.09	8.23	6.96†	9.10
Symptoms of depression ($n=184$)	9.51	10.17	9.18	10.45
Time 2 outcomes (21 months after Time 1)				
Engagement in healthy behaviors ($n=109$)	26.40	27.10	27.48	26.09
Recovery control appraisals ($n=110$)	2.95	2.91	2.85	3.00
Prevention control appraisals ($n=110$)	2.93	2.67	2.69	2.85
Cardiac symptom experiences ($n=110$)	5.23*	4.28	4.11*	5.22
Symptoms of anxiety ($n=106$)	10.07***	5.66	5.30*	9.51
Symptoms of depression ($n=96$)	8.54	8.82	8.42	9.06

ANCOVA: analysis of covariance.

Asterisks in the “yes” columns in the upper half of the table reflect significant differences between the “yes” and “no” means as indicated by an independent samples t -test. Asterisks in the “yes” columns in the lower half of the table reflect significant differences between the “yes” and “no” means as indicated by an ANCOVA, controlling for baseline levels of the outcome.

† $p < .05$; * $p < .05$; ** $p < .01$; *** $p < .001$.

Research Questions 1 and 2: differences in outcomes by attribution category

The two research questions tested in this study examined possible concurrent and long-term differences in health behaviors, control appraisals, and physical and psychological health status as a function of attribution category generated at the beginning of CR. Because very few participants created stress attributions, this category was dropped in subsequent analyses. Table 3 provides results of mean comparisons on these different outcomes by whether participants endorsed a behavioral/controllable cause (yes/no) and a biological/uncontrollable cause (yes/no). The top half of Table 3 provides comparisons on outcomes at Time 1 by attribution category, whereas the bottom half of the Table provides comparisons on outcomes at Time 2.

Concurrently, independent sample t -tests showed that participants who made a behavioral attribution at the beginning of CR engaged in fewer healthy behaviors ($t(198) = -3.54, p < .001; d = .50$), but had higher recovery control ($t(199) = 2.95, p < .01; d = .42$) and prevention control appraisals ($t(199) = 3.52, p < .01; d = .50$), than their counterparts who did not make a behavioral attribution. Additionally, participants who made a biological attribution at the beginning of CR engaged in more healthy behaviors ($t(198) = 2.46, p < .05; d = .35$) and trended toward experiencing fewer anxiety symptoms ($t(192) = -1.92, p = .06; d = .28$), but had lower prevention control appraisals ($t(199) = -2.65, p < .01; d = .38$), than their counterparts who did not make a biological attribution.

The longitudinal comparisons in the lower half of Table 3 were done with analyses of covariance (ANCOVAs), controlling for baseline levels of the outcomes. Results showed that participants who made a behavioral attribution at Time 1 experienced more cardiac symptom experiences at Time 2 than their counterparts who did not create a behavioral attribution ($F(2, 110) = 30.90, p < .001; \eta^2 = .04$). The reverse occurred for those who created a biological attribution: they experienced fewer cardiac symptoms than participants who did not create a biological attribution ($F(2, 110) = 31.42, p < .001; \eta^2 = .05$). Significant differences were also found for symptoms of anxiety. Participants who created a behavioral attribution at Time 1 experienced significantly more anxiety symptoms at Time 2 than participants who did not create a behavioral attribution ($F(2, 106) = 33.68, p < .001; \eta^2 = .12$). The same pattern as above emerged for participants who created biological attributions: they reported significantly fewer anxiety symptoms than participants who did not create a biological attribution ($F(2, 106) = 28.02, p < .001; \eta^2 = .05$).

Discussion

This study was conducted to examine whether (a) attributions generated by a sample of CR patients would align with previous research showing that the most common causes of CVD are behavioral, biological, and stress-related; (b) gender differences exist in the creation of these attributions; and (c) these attributions have effects on health appraisals and outcomes. Consistent with Hypothesis 1, patients' explanations fell into three main categories: biologically based ones

that were uncontrollable, behaviorally based ones that were controllable, and stress-related causes. Biological and behavioral attributions were created by almost equal proportions of participants, whereas stress was mentioned by fewer. Within the biological theme, the most commonly mentioned attribution involved heredity, whereas within the behavioral theme, the most commonly cited cause was poor diet. These results suggest that controllable and uncontrollable causes are equally salient to patients as they engage in a causal search to understand their diagnoses. Our results depart from past studies in the relatively low number of patients who endorsed stress as a cause. It is possible this is an artifact of our relatively affluent and educated sample, a majority of which no longer worked outside the home. By extension, it may be that these participants were subjected to fewer stressors than participants in other studies who were younger or lower in socioeconomic status (e.g. Cameron et al., 2005; Reges et al., 2011). Given that these stress-related findings differ from other studies, caution is warranted in generalizing beyond this sample.

Based on prior studies, Hypothesis 2 predicted gender differences in the types of attributions created: whereas men were predicted to make more behavioral attributions than women, women were predicted to generate more biological and stress attributions than men. Results did not support this hypothesis, however, as no gender differences emerged for behavioral or biological attributions (and too few stress attributions were generated to test for gender differences). Results imply that men and women are equally likely to draw on controllable and uncontrollable causes when creating meaning following a cardiac event. This may reflect a change in gender norms surrounding the role of behavior (i.e. exercise) in the promotion of physical health. Whereas older generations of women may have avoided physical exertion, women of all ages now endorse attitudes that embrace the central role of physical activity in health promotion (e.g. Ransford and Palisi, 1996; Rydwick et al., 2012). Our results also likely reflect a more accurate view of CVD risk factors by both men and women, recognizing that biological and behavioral risk factors often co-occur and interact to shape one's risk for CVD.

This study also sought to explore (through Research Questions 1 and 2) whether attributions made at the beginning of CR would differentiate between patients in their self-reported health appraisals and outcomes concurrently, and whether any longitudinal differences would be sustained 21 months later. At the beginning of CR, results showed that participants who created a behavioral attribution reported engaging in fewer health-promoting behaviors in the preceding month compared to those who did not generate a behavioral attribution. The opposite was true for those who created a biological attribution. Together, these differences suggest that patients had insight into the origins of their disease. For example, people who cited "poor diet" or "lack of exercise" as causes self-reported fewer of

those corresponding behaviors than participants who did not generate behavioral attributions. Significant differences also emerged for appraisals of control over recovery from CVD and control over prevention of recurrence. Participants who generated a behavioral cause reported significantly more perceived control over recovery and prevention compared to participants who did not create a behavioral attribution. Conversely, participants who generated a biological attribution felt significantly less perceived control over prevention than those who did not generate a biological attribution. Again, this pattern of results suggests insight into the origins and course of treatment for patients following a CVD event.

Although the cross-sectional results are encouraging on account of participants' seemingly accurate views of the origins and trajectories of CVD, the benefits associated with making behavioral attributions seem to disappear longitudinally. Patients who created behavioral attributions at baseline reported significantly more cardiac symptom experiences 21 months later compared to those who did not create a behavioral attribution. The same difference can be seen in symptoms of anxiety: patients who created a behavioral attribution at baseline experienced significantly more anxiety symptoms 21 months later than those who did not create a behavioral attribution. The direction is opposite for those who created biological attributions at baseline: they experienced significantly fewer cardiac and anxiety symptoms 21 months later compared to participants who did not generate biological attributions. This pattern suggests that believing one's behavior caused CVD is associated with psychological distress that can manifest as anxiety symptoms, or may translate to slower physical recovery operationalized as the experience of cardiac symptoms.

These findings are surprising, and depart from several prominent attribution theorists. For example, Janoff-Bulman (1979) suggests that behavioral self-blame attributions should aid psychological adjustment because behavior is malleable. Character, on the other hand, is predicted to be distressing because one's disposition is presumed to be immutable. To the extent that heredity and biological factors are uncontrollable, Janoff-Bulman's ideas would suggest behavioral attributions, but not biological attributions, aid in recovery and adjustment. However, these results strongly challenge the notion that creating a behavioral attribution is adaptive through enhancements in control appraisals; although differences in recovery and prevention control were seen cross-sectionally, no differences remained 21 months later. Rather, our findings imply that an attribution that absolves someone of control (e.g. heredity) can have a positive impact, and that rehabilitation efforts should use a forward orientation coupled with enhancements in control over *future* behaviors (Bennett et al., 2013).

These findings may have implications for CR providers. First, results suggest patients should not be encouraged to ruminate about past behaviors in an attempt to identify one

or more that caused their cardiac event. In fact, avoiding a behavioral attribution may protect patients from distress, which in turn could negatively affect their motivation or self-efficacy for sustained physical activity. Within the context of CR, the creation of a behavioral attribution may create anxiety about needing to change that behavior, which could interfere with the confidence being formed through continued participation in CR. Therefore, maintaining a future orientation, with a focus on building confidence to control future behaviors, seems warranted. Second, CR's strong focus on building exercise capacity seems to affect all patients regardless of the type of attribution created at baseline. That is, there were no differences found by attribution category in engagement in healthy behaviors. In fact, paired sample *t*-tests among participants who completed questionnaires at both times showed that regardless of whether behavioral or biological attributions were created, they made significant improvements in healthy behaviors from Time 1 to Time 2 (*t*s ranged from -4.13 to -5.17). Thus, as an intervention, CR seems quite effective in increasing health promotion behaviors.

Limitations and future directions

These data are the first of which we are aware to link open-ended cardiac attributions to self-rated health appraisals and outcomes nearly 2 years after a cardiovascular event. However, several study limitations are noteworthy. First, results are based on an ethnically homogenous, relatively affluent sample of CVD patients participating in a CR program. Research shows that referral rates to CR are low (approximately 56%; Brown et al., 2009), with rates of participation in CR among eligible patients even lower (approximately 19%; Suaya et al., 2007). Therefore, this sample likely represents a motivated group of patients that may differ from others not participating in CR. In order to increase generalizability of findings, future research should recruit a more heterogeneous sample of CVD patients, including ones who are not enrolled in CR. Second, all data in this study were self-reported; thus, they are subject to shared method variance, social desirability, and other response biases. Future research would benefit from clinical assessments of distress, cross-informant ratings of social-cognitive constructs, and observations of exercise-related behaviors. Third, attrition occurred between the assessments, resulting in a loss of approximately one-half the sample at Time 2. Although attrition over time points is normal, especially with follow-ups over many months (as was the case in this study), some caution is warranted when interpreting the longitudinal findings. Comparisons revealed that the sample used for the longitudinal analyses differed from the full sample in age, education, and risk stratification. Therefore, future research should attempt to reduce attrition at follow-up. Finally, these data cannot speak to the stability of cardiac attributions made. Future

research should measure patient-generated attributions over time to determine whether these causal searches yield stable meanings about the origins of one's CVD.

Conclusion

This study examined types of causal attributions generated by CR patients shortly after experiencing a cardiac event, with results showing that biological and behavioral ones were generated in equal proportions. Despite predictions, no gender differences emerged. Thus, men and women were equally likely to create biological (uncontrollable) and behavioral (controllable) attributions for their cardiac events. Linking attribution type to outcomes showed that creating a behavioral attribution seemed to be beneficial in the short-term for control appraisals, and that participants displayed insight into the origins of their diagnoses by making either behavioral or biological attributions. However, over time, any benefit to making a behavioral attribution disappeared, as it was associated with increased anxiety symptoms 21 months later. These findings suggest that CR providers should discourage patients from looking backward when creating meaning, but rather should encourage patients to maintain a future-focus that promotes perceived control over health promotion behaviors.

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