Causal Explanations as a Risk Factor for Depression: Theory and Evidence

Christopher Peterson
Virginia Polytechnic Institute and State University

Martin E. P. Seligman
University of Pennsylvania

The attributional reformulation of the learned helplessness model claims that an explanatory style in which bad events are explained by internal, stable, and global causes is associated with depressive symptoms. Furthermore, this style is claimed to be a risk factor for subsequent depression when bad events are encountered. We describe a variety of new investigations of the helplessness reformulation that employ five research strategies: (a) cross-sectional correlational studies, (b) longitudinal studies, (c) experiments of nature, (d) laboratory experiments, and (e) case studies. Taken together, these studies converge in their support for the learned helplessness reformulation.

After experiencing uncontrollable events, a variety of organisms show cognitive, motivational, and emotional deficits (Maier & Seligman, 1976; Seligman, 1975). This learned helplessness phenomenon has parallels with depression in people and has been proposed as a model of this psychopathology (Seligman, 1974). When helplessness theory proved unable to account for the generality and chronicity of depressive symptoms or for self-esteem loss in depression, it was revised along attributional lines (Abramson, Seligman, & Teasdale, 1978). According to this reformulation, the boundary conditions of depression following bad events are determined by causal attributions about the events (for similar formulations, see Miller & Norman, 1979, and Roth, 1980). The central prediction of the reformulation is that individuals who have an explanatory style that invokes internal, stable, and global causes for bad events tend to become depressed when bad events occur.

Our primary purpose here is to describe the empirical investigations of this theory conducted by our research group, and to evaluate the theoretical status of the attributional model. The juxtaposition of conceptual clarification with evidence from several levels of analysis may allow a valid assessment of the theory.

Theory

Helplessness Theory: Old and New

The learned helplessness phenomenon was first described systematically by animal learning researchers at the University of Pennsylvania (Overmier & Seligman, 1967; Seligman & Maier, 1967). Mongrel dogs, following exposure to inescapable electric shock, showed striking deficits 24 hours later when placed in a shuttlebox in which the simple act of crossing a barrier would terminate shock. Unlike dogs not previously exposed to uncontrollable shock, these animals seemed helpless. They initiated few attempts to escape the shock (motivational deficit). They were not likely to follow an occasionally successful response with another (learning or cognitive deficit). They did not evidence much overt emotionality while being shocked (emotional deficit).

These deficits were interpreted in cognitive terms (Maier, Seligman, & Solomon, 1969; Seligman, Maier, & Solomon, 1971). During exposure to the electric shocks, the dogs learned that shocks were independent of responses.
Regardless of what the dogs did or did not do, the shocks occurred. This learning was represented as an expectation of future response–outcome independence (i.e., uncontrollability) that was generalized to new situations to produce the observed deficits.

The cognitive interpretation of animal helplessness has been controversial. Some researchers have attempted to explain the phenomenon in peripheral or biological terms, whereas other researchers have argued for the cognitive explanation (for reviews of this controversy, see Maier & Jackson, 1979; Maier & Seligman, 1976; Seligman & Weiss, 1980). Regardless of this controversy, psychologists interested in human adaptation were quick to sense the possible pertinence of learned helplessness to failures of human action. Two parallel lines of research with human subjects took place.

In the first, the basic helplessness phenomenon was investigated in the laboratory with human subjects (for a review, see Wortman & Brehm, 1975). In the second, helplessness theory was used to explain a variety of human difficulties (see Garber & Seligman, 1980). Perhaps the best known of these applications has been Seligman's (1972, 1974, 1975) suggestion that learned helplessness may model depression with respect to symptoms, causes, and cures.

The main problem with the original helplessness model, applied both to human helplessness in the laboratory and to natural human depression, is its failure to account for boundary conditions. Sometimes laboratory helplessness is general (e.g., Hiroto & Seligman, 1975), and sometimes it is circumscribed (e.g., Cole & Coyne, 1977). Sometimes bad events precipitate depressive reactions (occasionally transient, occasionally long lasting), and sometimes they do not (e.g., Brown & Harris, 1978; Lloyd, 1980). What determines the chronicity and generality of helplessness and depression?

Similarly, the original model does not explain the self-esteem loss frequently observed among depressives (Beck, 1967; Freud, 1917/1957). Why should individuals blame themselves for events over which they perceive no control (Abramson & Sackeim, 1977)? The simple helplessness model is silent about the chronicity and generality of helplessness and depression and about the paradox of self-esteem loss following helplessness.

To address these shortcomings, Abramson, Seligman, and Teasdale (1978) revised helplessness theory to include the individual's causal explanations of the original bad events. According to this revision, when people face uncontrollable bad events, they ask why. Their answer affects how they react to the events. Abramson et al. (1978) argued that three explanatory dimensions are relevant. First, the cause may be something about the person (internal explanation), or it may be something about the situation or circumstances (external explanation). Second, the cause may be a factor that persists across time (stable explanation), or it may be transient (unstable explanation). Third, the cause may affect a variety of outcomes (global explanation), or it may be limited just to the event of concern (specific explanation). Table 1 presents examples of these types of explanations as they might be made about the bad event "My checking account is overdrawn."

The reformulation assigns particular roles to each of these three dimensions. Internality of causal beliefs affects self-esteem loss following bad events. If the person explains a bad event by an internal factor, then self-esteem loss is more likely to occur. If a person explains the event by an external factor, then self-esteem loss is less likely to occur. Stability of causal beliefs affects the chronicity of helplessness and depression following bad events. If a bad event is explained by a cause that persists, depressive reactions to that event tend to persist. If the event is explained by a transient factor, then depressive reactions tend to be short lived. Finally, globality of causal beliefs influences the pervasiveness of deficits following bad events. If one believes that a global factor has caused a bad event, then helplessness
deficits tend to occur in a variety of different situations. If one believes that a more specific factor is the cause, the deficits tend to be circumscribed.

**Conceptual Status of Explanation and Explanatory Style**

**Causal explanations: Sufficient condition or risk factor for depression?** We used qualified language above in stating the central predictions of the reformulation. So, an internal explanation for a bad event is said to make self-esteem loss more likely, but not to cause self-esteem loss. It is important to realize that explanations and their precursor, explanatory style, are not sufficient to produce depressive deficits but rather are risk factors for such deficits (Abramson et al., 1978).

The relationship among variables in the theory is diagrammed in Figure 1. Let us begin by looking at the extreme right-hand side of this figure. What Figure 1 attempts to explain is the general process by which the symptoms of helplessness are produced. These symptoms are passivity; cognitive deficits; emotional deficits including sadness, anxiety, and hostility; a lowering of aggression; a lowering of appetitive drives; a set of neurochemical deficits; and an increase in susceptibility to disease. In addition, the symptom of self-esteem loss is sometimes one of the symptoms of helplessness. These symptoms, taken together, look very much like the syndrome of depression. How do these symptoms come about?

In learned helplessness theory, both original and reformulated, the expectation that no action will control outcomes in the future is a sufficient condition for the production of all of these symptoms of helplessness except self-esteem loss. This expectation is represented in the center of Figure 1. Whenever and wherever this expectation occurs, the symptoms will develop. Thus, our main concern is how other processes and events conspire to bring about this expectation, resulting in the symptoms of helplessness. This expectation is usually triggered when bad events are perceived as uncontrollable. The reality of the bad uncontrollable events influences the content of the expectation. So, for example, if the bad event is blindness, and one's job is proofreading, the range of outcomes to which the expectation applies includes work.

Explanations and explanatory style also influence the expectation that no action will control outcomes in the future (see Figure 1). Explanations involving global causes tend to produce the expectation that action will not control many outcomes, which in turn produces the symptoms of helplessness in exactly that large range of situations. In parallel, if the cause of a bad event is explained by stable factors, the expectation tends to occur for a long time into the future, and therefore, the symptoms of helplessness are long-lasting. If the explanation for a bad event is internal, then the symptom of lowered self-esteem tends to be displayed. Thus, the particular explanation an individual makes for the bad event influences the generality and time course of the symptoms of helplessness, as well as the loss of self-esteem.

In the model, there are two influences on the particular explanation chosen. The first is the reality of the bad events themselves. If the bad event that sets off the expectation of helplessness is the death of one's spouse, this is a stable and global loss. The spouse will not return, and many of the activities in which one has customarily engaged will be undermined. The second influence on what particular ex-

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**Table 1**

**Examples of Causal Explanations for the Event "My Checking Account is Overdrawn"**

<table>
<thead>
<tr>
<th>Style</th>
<th>Internal</th>
<th>External</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global</td>
<td>&quot;I'm incapable of doing anything right&quot;</td>
<td>&quot;All institutions chronically make mistakes&quot;</td>
</tr>
<tr>
<td>Specific</td>
<td>&quot;I always have trouble figuring my balance&quot;</td>
<td>&quot;This bank has always used antiquated techniques&quot;</td>
</tr>
<tr>
<td>Unstable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global</td>
<td>&quot;I've had the flu for a few weeks, and I've let everything slide&quot;</td>
<td>&quot;Holiday shopping demands that one throw oneself into it&quot;</td>
</tr>
<tr>
<td>Specific</td>
<td>&quot;The one time I didn't enter a check is the one time my account gets overdrawn&quot;</td>
<td>&quot;I'm surprised—my bank has never made an error before&quot;</td>
</tr>
</tbody>
</table>
plation is made is the habitual tendency to choose certain kinds of explanations for bad versus good events. We have been able to identify individual patterns in the selection of causes over a variety of events, and we call these patterns explanatory style. The particular style that most concerns us is the depressive explanatory style, in which one tends to give internal, stable, and global explanations for bad events (it's me; it's going to last forever; and it's going to affect everything I do).

It should now be apparent why a particular explanation or explanatory style is not sufficient for the symptoms of helplessness to appear. These variables influence the expectation, but it is the expectation which is sufficient. Usually, causal explanations for an event and expectations about the consequences of an event have the same properties. For example, if the explanation for blindness is a progressive brain disease, this cause has stable and global properties, as do the consequences of blindness. But sometimes the properties of a cause and its consequences can be dissimilar. If, for example, the cause of blindness was a freak accident, the cause is unstable and specific, but the consequences are stable and global.

Because there is usually similarity between causal explanation and expectation of consequences, knowing an individual's explanation and explanatory style will usually predict helplessness deficits. Because there can sometimes be dissimilarity, an individual's causal explanation and explanatory style will not cause the deficits. And so we speak of these variables as risk factors (just as smoking is a risk factor for lung cancer), rather than sufficient conditions for helplessness and depression.

Our research focused on measuring explanatory style, because a methodological framework already existed for doing so (e.g., Harvey, Ickes, & Kidd, 1976, 1978, 1981). We chose not to focus on the expectation itself, even though it is more proximate to the symptoms, because we do not believe that a valid means of measuring expectations yet exists. Also, in most cases, we chose not to focus on particular explanations because explanatory style of necessity is a more reliable individual difference (Epstein, 1980).

Causal explanations and explanatory style: Hypothetical constructs. The term attribution has been used in a variety of ways. Some the-
orists regard attributions as the stated beliefs that compose an individual's naive psychology (Heider, 1958), whereas other theorists suggest that attributions have tacit components (Langer, 1978; Wortman & Dintzer, 1978). Controversy over how best to assess attributions has ensued (Nisbett & Wilson, 1977). We wish to make explicit that we regard an attribution as a hypothetical construct, a way for the theorist to explain observable behaviors. We do not use the notion to refer to an intervening variable (cf. MacCorquodale & Meehl, 1948).

As a hypothetical construct, causal explanation does not have a single definition nor is it exhausted by any one operation. An intervening variable is exhausted in its meaning by one class of input operations and one class of output operations. Our view of attributions contrasts with Heider's (1958) phenomenological view of attributions, in which the abilities to introspect upon and to report verbally one's causal beliefs are necessary and sufficient conditions for them. Heider (1958) regarded attributions as intervening variables. For us, on the other hand, an attribution is not a real thing, like a microphone or a typewriter. Rather, it is akin to concepts like natural selection, life, reward, preference, or—perhaps—the atom. These are all hypothetical constructs, measured with a number of different converging operations, none of which defines or exhausts the construct of interest. By this logic, not only introspections and answers to questionnaires are relevant to knowing about one's causal explanations, but so too are a variety of behavioral observations. In the studies we report, we attempt to use converging ways to infer the presence of causal explanations.

Attributional Style Questionnaire. One way in which we measure explanations and explanatory style is with a questionnaire that we developed for this purpose: the Attributional Style Questionnaire (ASQ; Peterson, Semmel, et al., 1982). This self-report instrument yields scores for the explanation of bad and good events with internal versus external, stable versus unstable, and global versus specific causes. The format reflects the fact that we wanted questions to assess how much respondents used particular values of the three dimensions. We ask subjects to generate their own cause for each of a number of events, and then to rate that cause themselves along 7-point scales corresponding to the internality, stability, and globality dimensions. The ASQ does not constrain or create the causal explanations provided by the subject, but at the same time it allows simple and objective quantification of responses by asking the subject to rate the internality, stability, and globality of the causes.

The questionnaire is group administered, and the following directions appear on the first page of the booklet:

Please try to vividly imagine yourself in the situations that follow. If such a situation happened to you, what would you feel would have caused it? While events may have many causes, we want you to pick only one—the major cause if this event happened to you. Please write this cause in the blank provided after each event. Next we want you to answer some questions about the cause. To summarize we want you to:

1. Read each situation and vividly imagine it happening to you.
2. Decide what you feel would be the major cause of the situation if it happened to you.
3. Write one cause in the blank provided.
4. Answer three questions about the cause.
5. Go on to the next situation.

Because we are interested in style—cross-situational explanations—we describe 12 different hypothetical events. Half are good events (e.g., you meet a friend who compliments you on your appearance), and half are bad events (e.g., you go out on a date, and it goes badly). After each event are parallel questions. First, the subject is asked to write down the one major cause of the event. Then the subject is asked to rate the cause along the three explanatory dimensions. The wording of the various questions reflects the specific event to be explained, and the example in Table 2 illustrates the nature of these questions.

The three ratings of each cause are scored in the directions of increasing internality, stability, and globality. Total scores are formed separately for the bad and good events simply by summing the appropriate items and dividing the sum by 6. Table 3 reports the internal consistencies, intercorrelations, and test–retest reliabilities of these scales for a sample of undergraduates at the State University of New York at Stony Brook (Peterson, Semmel, et al., 1982).

Several things should be noted about these data. First, the individual scales have modest reliabilities, ranging between .44 and .69. One reason for these low reliabilities is the small
number of items—six—in each scale. Second, the scales are substantially intercorrelated within good events and within bad events. For this reason, we usually combine them into overall composites for good events and bad events, thereby bolstering reliability to more acceptable levels (alphas of .75 and .72, respectively). Third, causal explanations for good events are independent of causal explanations for bad events. Conceptions that confound the two (e.g., Rotter, 1966) incorrectly average over two quite different phenomena. Fourth, the stability of explanatory style scores is respectably high, as we had hoped in assuming that they operationalize a characteristic style of causal explanation.

**Criterion validity of the ASQ.** What is the validity evidence for this questionnaire? We present support for the construct validity below. However, two recently completed studies speak to the criterion validity of the ASQ: the degree to which the questionnaire predicts naturally occurring causal explanations. Do answers on the ASQ reflect the natural, unconstrained causal explanations an individual makes?

To test if the scores obtained with the ASQ are more than questionnaire artifacts, we asked 66 adults (31 males, 35 females) on the University of Pennsylvania campus to describe in writing, using 250 to 300 words, the two worst events involving themselves during the last year (Peterson, Bettes, & Seligman, 1982). Instructions stressed descriptive accuracy, and there was no mention of causal explanation. The subjects then completed the ASQ, which had not been mentioned prior to this point. This procedure allowed us to see (a) if causal accounts are spontaneously included in descriptions; (b) if individuals are consistent across different explanations in terms of their internality, stability, and globality; and (c) if these spontaneous explanations converged with scores of the ASQ.

We had first developed a procedure by which spontaneous explanations in written material could be extracted and scored for internality, stability, and globality (Peterson, Luborsky, & Seligman, 1983). A researcher reads the material and notes descriptions of bad events involving the writer for which a causal explanation is made (identified by such key phrases as “because of,” “as a result of,” and so on). Next, each event description and the accompanying explanation are transcribed verbatim and are shown singly to independent and blind judges who rate the explanation with 7-point scales corresponding to its internality, stability, and globality. These judges use the definitions from the ASQ (Peterson, Semmel, et al., 1982). Finally, the ratings by the different judges are combined into a profile for the individual.

For this particular study, there were four judges. Coding reliabilities, estimated by Cronbach’s (1951) alpha, were satisfactory: .93, .89, and .90, for the internal, stable, and global ratings, respectively. Table 4 presents the ratings made by the judges for this excerpt from a written description (emphasis added):

> About four months ago, he called me on the telephone from ______, where he’s been working. He told me that our relationship was over, that he didn’t want to see me ______

2 For a newly revised version of the ASQ with 24 bad events, reliabilities are substantially improved: .66 for internality, .85 for stability, and .88 for globality.
Table 3
Reliabilities and Intercorrelations of the Attributional Style Questionnaire

<table>
<thead>
<tr>
<th>Dimension</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good events</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Internality</td>
<td>(.50)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Stability</td>
<td>.62*</td>
<td>(.58)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Globality</td>
<td>.38*</td>
<td>.59*</td>
<td>(.44)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Composite</td>
<td></td>
<td></td>
<td></td>
<td>(.75)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bad events</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Internality</td>
<td>.11</td>
<td>.01</td>
<td>-.03</td>
<td>(.46)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Stability</td>
<td>-.17</td>
<td>-.07</td>
<td>.03</td>
<td>.18*</td>
<td>(.59)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Globality</td>
<td>-.15</td>
<td>.04</td>
<td>.24*</td>
<td>.28*</td>
<td>.45*</td>
<td>(.69)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Composite</td>
<td></td>
<td>.02</td>
<td></td>
<td>(.72)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Test-retest correlations

| M       | 5.26 | 5.36 | 5.11 | 5.25 | 4.29 | 4.14 | 3.87 | 4.12 |
| SD      | .79  | .68  | .80  | .62  | .84  | .71  | 1.07 | .64  |
| Test-retest correlations | .58* | .65* | .59* | .70* | .64* | .69* | .57* | .64* |

Note. N = 130. From Peterson, Semmel, et al., (1982). Figures in parentheses, on diagonal, are reliabilities estimated by Cronbach’s (1951) alpha. Test-retest correlations over 5 weeks (n = 100).

* p < .05.

anymore. I felt devastated. I tried to argue, but what could I say? I’m still flipped out. I guess I’m just no good at relationships. I’ve never been able to keep a man interested in me. I talked to my roommate about it all, right after he called me. She knows me real well, plus she went to high school with ———. She told me to forget about him and find someone else. She said I made a mistake and the best thing to do was move on. There was never a chance for a lasting thing. But how could I misjudge things so badly? When I get stars in my eyes I get carried away.

All of the descriptions contained at least one causal explanation. We concluded that causal explanations are spontaneously offered, even when they are not explicitly prompted. Ratings of the first explanation offered by an individual were correlated with those of the second, and significant correlations were obtained. Furthermore, when these ratings were combined across explanations by the same individual, they correlated respectably with the corresponding dimensions of the ASQ. Exact values are reported in Table 5.

We carried out a second study to see if the content analysis of explanatory style was valid in patient populations as well as in normal populations (Castellon, Ollove, & Seligman, 1982). Toward this end, we gave 40 outpatients the Schedule for Affective Disorders and Schizophrenia (SADS) diagnostic interview (Spitzer & Endicott, 1977) in the same session as we gave them the ASQ and the Beck Depression Inventory (BDI; Beck, 1967). These patients were diagnosed unipolar depressive episode by this interview. The first two questions of the SADS interview ask the patient (a) why treatment was sought and (b) the origin of their symptoms. We performed a blind content analysis—exactly as is described above—of the causal explanations offered in answer to the first two questions of the SADS interview.

Content analysis seems valid for a severely depressed population. The composite score of the ASQ for bad events correlated significantly with the composite score for bad events obtained by this content analysis (r = .38, p < .02). Furthermore, explanatory style, measured in both ways, correlated with severity

Table 4
Example of Content Analysis

<table>
<thead>
<tr>
<th>Event explanation</th>
<th>Judge</th>
<th>Int</th>
<th>Sta</th>
<th>Glo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Our relationship was over. (because) I’m just no good at relationships. I’ve never been able to keep a man interested in me.</td>
<td>A</td>
<td>7</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>How could I misjudge things so badly? (because) When I get stars in my eyes I get carried away.</td>
<td>B</td>
<td>5</td>
<td>6</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 5

<table>
<thead>
<tr>
<th>Dimension</th>
<th>M</th>
<th>SD</th>
<th>Consistency r with corresponding ASQ score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internality</td>
<td>3.71</td>
<td>1.54</td>
<td>.25**</td>
</tr>
<tr>
<td>Stability</td>
<td>3.78</td>
<td>1.62</td>
<td>.37**</td>
</tr>
<tr>
<td>Globality</td>
<td>3.13</td>
<td>1.43</td>
<td>.33**</td>
</tr>
<tr>
<td>Composite</td>
<td>3.54</td>
<td>1.27</td>
<td>.36**</td>
</tr>
</tbody>
</table>

Note. n = 66. ASQ = Attributional Style Questionnaire.
*p < .10. **p < .05. ***p < .001.

of depressive symptoms, as measured by the BDI.

These two studies have several important implications. First, the important constructs of the helplessness reformulation—explanations and explanatory style—are more than responses to questions about hypothetical events. Individuals offer unsolicited causal explanations about important bad events, and these explanations are consistent with each other along the dimensions of internality, stability, and globality. Second, the ASQ has criterion validity. Internality, stability, and globality ratings of spontaneous explanations converged with the corresponding scales of the questionnaire.

Explanatory style versus reality. As we argued above, explanations are determined by both situational and dispositional factors. That the reality of an event can determine explanations is undeniable. Also, we prefer the term explanation to attribution because attribution connotes projection too strongly. In saying that reality partly determines explanations, we are not saying that causal explanations are therefore accurate (Langer, 1978; Nisbett & Wilson, 1977). Rather, we are saying that causal beliefs are not entertained in a vacuum, and appropriate investigations of the helplessness reformulation must not look only at dispositional determinants of explanations.

A number of studies have shown manipulations of reality to affect causal explanations, which in turn affect laboratory helplessness (see reviews by Abramson et al., 1978; Miller & Norman, 1979; and Roth, 1980). A representative study is that of Tennen and Eller (1977), who instructed subjects given unsolvable problems that the task was either easy or difficult. Failure at an easy task encourages explanations in terms of lack of ability, whereas failure at a difficult task results in explanations in terms of task difficulty. Subjects given "easy" unsolvable problems showed later problem-solving deficits, whereas subjects given "difficult" unsolvable problems showed facilitation.

Situational factors also affect explanations and depressive reactions to events outside the laboratory. Peterson and Conn (1982) investigated the reactions of people who had just ended a romance. Although most subjects had depressive symptoms and sad affect following the breakup, the most severe reactions were by those whose partner left them for someone else. In contrast, the least severe reactions were by those whose romance ended because their partner was attending a different college.

If one’s partner leaves for someone else, the answer to the why question tends to be in terms of stable and global characteristics of the person left behind. Many external explanations (e.g., my partner is not interested in romance) are implausible, as are interpretations in terms of transient and circumscribed causes. In contrast, if romance ends because of distance, one need not see oneself as causing the breakup. Furthermore, one need not believe that the causes will persist, or will be pervasive. Indeed, subjects whose romance ended because of distance were more optimistic about achieving future relationships than were subjects whose partner left for someone else.

Individual dispositions in the explanations are needed to explain why different individuals have different reactions to the same events. Why do some people become helpless following unsolvable problems and others not (Alloy, Peterson, Abramson, & Seligman, 1984; Dweck & Licht, 1980)? Why do some people become depressed following bad events and others not (Lloyd, 1980; Metalsky, Abramson, Seligman, Semmel, & Peterson, 1982)? The reformulation claims that people susceptible
to helplessness and depression interpret these bad events in internal, stable, and global terms.

If reality is ambiguous enough, a person may project and impose habitual explanations. According to the reformulation, individuals have a characteristic way of explaining events. They are consistent in the way they use internal (vs. external), stable (vs. unstable), and global (vs. specific) causes to explain bad events. Thus, when reality is ambiguous, the ASQ works as a projective test and may be used to measure a person's characteristic explanatory style. If this style invokes internal, stable, and global causes, then the person tends to become depressed when bad events occur. This is the central prediction of the reformulation, and we now describe converging evidence in support of this prediction.

**Research strategies.** We use a variety of research strategies: (a) cross-sectional correlational studies, (b) causal modeling with longitudinal data, (c) experiments of nature, (d) laboratory experiments, and (e) case studies. Each class of investigation addresses a more stringent prediction. The cross-sectional studies seek merely to correlate explanatory style and depressive symptoms. The longitudinal studies investigate whether a depressive style precedes depression, consistent with a predisposing role. The experiments of nature look at how naturally occurring bad events and explanatory style interact to produce a subsequent depressive reaction. The laboratory experiments see if experimentally manipulated bad events interact with explanatory style to result in laboratory helplessness, a depressive analogue. Finally, the case studies ascertain the applicability of the reformulation to real lives.

**Evidence**

**Cross-Sectional Correlational Investigations**

In this section, we describe several investigations of the hypothesis that depressive symptoms correlate with the use of internal, stable, and global causes to explain bad events. These studies are cross-sectional, and employ a variety of populations, using the ASQ. Depressive symptoms were assessed by self-report questionnaires and by psychiatric diagnosis.

**Study 1: ASQ and Depression**

In the first investigation of the reformulation, Seligman, Abramson, Semmel, and von Baeyer (1979) administered the ASQ to a sample of 143 college students at the University of Pennsylvania, along with the short form of the BDI (Beck & Beck, 1972), a 13-item self-report instrument that assesses the severity of common depressive symptoms (for validity evidence among undergraduates, see Bumby, Oliver, & McClure, 1978). As predicted by the helplessness reformulation, depressive symptoms among undergraduates correlated with internal ($r = .41, p < .001$), stable ($r = .34, p < .001$), and global ($r = .35, p < .001$) explanations for bad events (composite $r = .48, p < .001$).

**Study 2: Spontaneous Explanations and Depression**

We have already described our investigation of spontaneous explanations (Peterson, Bettes, & Seligman, 1982). Sixty-six adults wrote essays from which causal explanations of bad events were extracted and rated for internality, stability, and globality. These explanations were consistent and converged with the corresponding scales of the ASQ. These subjects also completed the short form of the BDI.

ASQ scores correlated with depressive symptoms as predicted by the reformulation: internality ($r = .44, p < .001$), stability ($r = .42, p < .001$), globality ($r = .29, p < .02$, and composite ($r = .45, p < .001$), replicating the results of Study 1. In addition, when ratings of the extracted explanations were averaged across different causes offered by the same individual, these scores also correlated with depressive symptoms: internality ($r = .36, p < .005$), stability ($r = .29, p < .02$), globality ($r = .33, p < .01$), and composite ($r = .39, p < .001$).

The studies so far described support the predictions. However, these studies employed somewhat restricted samples: upper middle-class college students who were on the whole not seriously depressed. To extend the empirical support for the depressive explanatory style, we conducted parallel investigations with three different samples: lower-class women, children, and psychiatric inpatients.

**Study 3: Lower-Class Women**

Depressive disorders among the lower class have sometimes been regarded as mainly somatic (Schwab, Bialow, Brown, Holzer, & Stevenson, 1967). Accordingly, the relevance
of a cognitive model like the helplessness reformulation may be questioned. For this reason, we asked women from the lower socioeconomic class to respond to the ASQ and the BDI (Navarra, 1981). Forty-one women (average age = 38 years) on welfare in Philadelphia were recruited through newspaper advertisements as research participants. In an interview format, they answered the questions of the ASQ and of the long form (21 items) of the BDI (Beck, 1967).

As in the other samples, causal explanations for bad events correlated with depression, although the stability dimension did not reach statistical significance: internality \( r = .42, p < .01 \), stability \( r = .12, \ ns \), globality \( r = .50, p < .001 \), and composite \( r = .50, p < .001 \).

**Study 4: Children**

The helplessness reformulation purports to be a general theory that should explain depression across the life span (Seligman & Peterson, in press). Explanatory style should correlate with depressive symptoms among children in the same pattern as among adults. Accordingly, we conducted an investigation to see if the depressive explanatory style characterized depressed children (Seligman et al., 1984).

We tested 96 children from two Philadelphia elementary schools that consisted predominantly of white, middle-class children. Approximately equal numbers of boys \( n = 50 \) and girls \( n = 46 \) from the third, fourth, fifth, and sixth grades participated. The subjects completed the Children's Depression Inventory (CDI; Kovacs & Beck, 1977) and a Children's Attributional Style Questionnaire (CASQ), which we developed. During class-time, the researcher read the questionnaires aloud while the children silently read their copies. The questionnaires were completed twice by the same children, at an interval of 6 months.

The CDI is a 27-item questionnaire that measures the severity of emotional, motivational, cognitive, and somatic symptoms of depression. Each item consists of three self-report statements graded in severity from 0 to 2. The child is instructed to choose the option corresponding to how he or she has been feeling during the preceding 2 weeks.

The CASQ is a forced-choice instrument. We found in pilot work that young children had trouble with the adult ASQ, particularly globality. Hence, we used a force-choice format in which hypothetical good or bad events involving the child were followed by two possible explanations, which varied one of the explanatory dimensions while holding the other two constant. Sixteen questions pertain to each of the three dimensions; half refer to good events, and half to bad events. Examples of items from the CASQ are shown in Table 6.

The CASQ is scored by assigning a 1 to each internal or stable or global response, and a 0 to each external or unstable or specific response. Scales are formed by summing these scores across the appropriate questions for each of the three dimensions, separately for good
Table 7
Children’s Attributional Style Questionnaire Scores and Depression

<table>
<thead>
<tr>
<th>Dimension</th>
<th>Good events</th>
<th></th>
<th></th>
<th></th>
<th>Bad events</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>M</td>
<td>SD</td>
<td>$r_{11}$</td>
<td></td>
<td></td>
<td></td>
<td>r with CDI</td>
</tr>
<tr>
<td>Internality</td>
<td>4.61</td>
<td>1.48</td>
<td>.32</td>
<td>.53**</td>
<td>.63**</td>
<td></td>
<td></td>
<td>-.34**</td>
</tr>
<tr>
<td>Stability</td>
<td>4.21</td>
<td>1.91</td>
<td>.55</td>
<td>.61**</td>
<td>.52**</td>
<td>.64**</td>
<td>.45**</td>
<td></td>
</tr>
<tr>
<td>Globality</td>
<td>4.67</td>
<td>1.58</td>
<td>.40</td>
<td>.54**</td>
<td>.31</td>
<td>.66**</td>
<td>.45**</td>
<td></td>
</tr>
<tr>
<td>Composite</td>
<td>13.49</td>
<td>3.72</td>
<td>.66</td>
<td>.71**</td>
<td>.66**</td>
<td>.66**</td>
<td>.51**</td>
<td></td>
</tr>
</tbody>
</table>

|                   | Bad events |          |          |          |            |          |          |          |
| Internality       | 2.30       | 1.57     | .43      | .63**    |            |          |          |          |
| Stability         | 2.40       | 1.40     | .42      | .52**    |            |          |          |          |
| Globality         | 1.88       | 1.27     | .31      | .64**    |            |          |          |          |
| Composite         | 6.58       | 2.77     | .50      | .66**    |            |          |          |          |

|                   | Children's Depression Inventory (CDI) | 7.71       | 6.28     | .86      | .80**      |          |          |          |

Note. From Seligman et al. (1984). $r_{11}$ is internal consistency estimated by Cronbach’s (1951) alpha.

*p < .05, **p < .001.

... events and for bad events. Scores for each of the scales range from 0 to 8.

Table 7 presents the reliabilities and stabilities of the questionnaires completed by the children at Time 1. Statistics at Time 2 were essentially the same, and are not shown. CASQ subscales were only modestly reliable. More satisfactory reliabilities were obtained by combining the subscales (separately for good events and for bad events). Alpha for the good events composite was .66, and for bad events was .50 (see Table 7). The CASQ scales and composites were consistent over the 6-month interval, showing explanatory style to be a somewhat stable individual difference among children, just as it is among adults.

CDI scores were highly reliable and highly consistent over the 6 months, implying that childhood depression, at least as measured here, is not a transient phase (Tesiny & Lefkowitz, 1982). As predicted, explanatory style correlated with depressive symptoms (see Table 7). The explanation of bad events with internal, stable, and global causes covaried with CDI scores, as did the reverse style for good events. Considering the modest reliabilities of the CASQ, these correlations are substantial; depression among school children is closely tied to the causal explanations they pick.

This study broadens the empirical base of the depressive explanatory style by showing it among adults, not just at the symptom level but also in terms of explanatory style (Schulterbrandt & Raskin, 1977).

Study 5: Depressed Patients

These four studies have two shortcomings. First, few of the individuals were seriously depressed. Although the helplessness reformulation regards mild and severe unipolar depression as continuous, at least with regard to cognitive characteristics (Seligman, 1978), this is an empirical issue. Second, the research so far described does not show the “depressive” style to be specific to depression. Perhaps it is a general characteristic of psychopathology.

We therefore conducted a study in which formally diagnosed unipolar depressed inpatients completed the ASQ (Raps, Peterson, Reinhard, Abramson, & Seligman, 1982). We compared their scores with those of two other patient groups. The first comparison group was medical and surgical patients, to control for being hospitalized (Taylor, 1979). The second was nondepressed schizophrenics, to control for severity of psychopathology and length of hospitalization (Raps, Peterson, Jonas, & Seligman, 1982). Although a schizophrenic group is not an exhaustive control for psychopathology, it provides a first step toward assessing specificity.

We administered the ASQ to male veterans hospitalized at the Northport Veterans Administration Medical Center: 30 unipolar depressed patients, 15 nondepressed schizo-
phrenic patients, and 61 nondepressed medical and surgical patients. As shown in Table 8, depressives explained bad events with more internal, stable, and global causes. These results confirm, in a sample of unipolar depressed patients, the association between explanatory style for bad events and depressive symptoms predicted by the helplessness reformulation. Such a style is not a general characteristic of psychopathology, because schizophrenic patients did not show it.

Two other studies have also correlated the ASQ with depressive symptoms in severe unipolar depressed patients. Eaves and Rush (1984) compared 31 depressed female psychiatric patients, diagnosed as unipolar, major affective disorder, with an age-, sex- and education-matched control group of 17 people.

The effects were striking. Depressed patients, whether endogenous, nonendogenous, or unremitted, all differed from the control group in terms of explanations for bad events (all ps < .0001). Table 9 presents these data.

For these patients, sensitivity of the ASQ, defined as the probability of a correct diagnosis of depression by the test given that the patient is depressed (i.e., the percentage of true positives; Lusted, 1968, pp. 108–109), was 61% for internality for bad events, 58% for stability for bad events, and 77% for globality for bad events. Specificity of the ASQ, defined as the probability of a correct diagnosis of nondepressed by the test given that the patient is nondepressed (i.e., the percentage of true negatives; Lusted, 1968, pp. 108–109), was 94% for internality for bad events, 94% for stability for bad events, and 77% for globality for bad events.
for bad events, and 88% for globality for bad events. These compare favorably to the best biological tests of depression (Eaves & Rush, 1982).

In addition, explanatory style for bad events significantly correlated with the total amount of time the individual was depressed, the average length of episode, and the length of the current episode for nonendogenously depressed individuals. This was particularly the case for the stability measure, which correlated .79 with the total time of depression, .76 with the average length of episode, and .71 with the length of the current episode (all ps < .001). Theoretically, these data suggest that stability for bad events reflects, and may predict, how long a depressive episode will last.

Another study of unipolar depressed patients was conducted by Persons and Rao (1981). Forty-nine patients completed the ASQ, as well as other measures. Upon admission, the internality score for bad events correlated .60 with the BDI. The globality score for bad events correlated .53, whereas the stability score for bad events did not correlate significantly with the BDI. Thirty-four of these patients were followed through discharge, and changes in explanatory style and BDI scores were measured. Explanatory style change over therapy (largely chemical) accounted for 49% of total change in depression. This was the most powerful variable correlated with change in depression.

Overall, then, these three studies show that severely depressed patients have an insidious explanatory style characterized by internal, stable, and global explanations for bad events involving the self. This style is continuous with the findings in mild depression and not a general characteristic of psychopathology, inasmuch as schizophrenic patients did not show it. In addition, explanatory style is an index that is both sensitive and specific. Finally, changing explanatory style is a good correlate and may be a good predictor of changing depression. This last finding is particularly suggestive for therapy, because techniques that encourage an individual to change his or her explanatory style may relieve depression (Seligman, 1981).

Disconfirming Studies

Several researchers have correlated depressive symptoms with the use of internal, stable, and/or global explanations for bad events and obtained nonsignificant correlations (e.g., Hammen & deMayo, 1982). Some have concluded that the helplessness reformulation is therefore wrong (e.g., Coyne & Gotlib, 1983). How do we reconcile this with the substantial support found for a depressive explanatory style?

Conclusions based on accepting the null hypothesis are always problematic, because nonsignificant results may alternatively reflect an insensitive or inappropriate method. This may be the case for studies that have failed to find a depressive explanatory style. Peterson and Raps (1983) observed that these studies tend not to distinguish explanations per se from explanatory style. As a result, they operationalize style with single-item questionnaires, running the risk that the particular real event about which respondents offer explanations is the overriding determinant of that explanation. This technique minimizes the role of the person's cognitive style. If respondents are asked to offer explanations about several (hypothetical) bad events, then the chances are greater that the average of these explanations will reflect a characteristic style.

This is really a logical argument, another way of phrasing the statistical truisms that reliability and thus validity are served by increasing the number of observations above one (Epstein, 1980). To illustrate this empirically, Peterson and Raps (1983) classified a number of cross-sectional investigations of the explanation–depression link, noting the numbers of events about which explanations were made in each study. As Table 10 shows, confirming studies were more likely than were disconfirming studies to ask about several events. Although the disconfirming studies may be correct in concluding that contextual characteristics are importantly related to depression, they are incorrect in further concluding that explanatory style is unrelated, because they usually did not assess style in a psychometrically or theoretically reasonable way.

Conclusions

Severity of depressive symptoms is often correlated with the habitual use of internal, stable, and global causes to explain bad events involving the self. This depressive explanatory style has been demonstrated with a variety of
procedures in a variety of populations. Some evidence suggests that it may be specific to depression. Studies failing to find the depressive explanatory style tend to use inappropriate methods. So, there is a clear relationship between a particular explanatory style and depression. But does this style put one at risk for later depression? Several other possibilities are compatible with this correlation.

First, there is the possibility of a tautological relationship of depressive explanatory style and depressive symptoms. For example, many answers that count toward the self-report of depression are phrased in terms of internal, stable, and global causes, for example, "I will always fail at whatever I do." Perhaps a bad explanatory style is just a special symptom of depression. This is not the case. If we eliminate such items from the BDI and correlate explanatory style with the remaining items, the correlation does not go down (Peterson, Schwartz, & Seligman, 1981). In addition, as we report below, such a style precedes and predicts who, among people not depressed, will later become depressed.

Second, there is the possibility that depression wholly causes the bad explanatory style. On this view, when one becomes depressed, the depressive explanatory style is activated as a result. A study by Mukherji, Abramson, and Martin (1982) argues against this possibility. They induced a depressed mood in undergraduates with the Velten (1968) procedure. Although mood induction was successful, explanatory style as measured by the ASQ was unaffected.

Third, there is the possibility that some third variable, such as biochemical deficits or a preexisting style of turning anger inward, causes both depression and the depressive explanatory style. The remaining lines of research attempt to untangle these various possibilities from the process predicted by the reformulation.

In the next section, we begin this untangling by describing evidence for the next most stringent prediction of the reformulation: that depressive explanatory style precedes depression.

### Longitudinal Investigations

According to the reformulation, explanatory style is a risk factor for depression. It must precede the occurrence of depressive symptoms. Because cross-sectional studies are momentary, they cannot address this prediction. However, several longitudinal investigations have been conducted in which explanatory style was assessed at one time and consequences at a later time.

### Study 6: Children

We have already described our research with grade-school children, who completed mea-

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Table 10

Cross-Sectional Investigations of the Reformulation and the Number of Events for Which Explanations Were Made

<table>
<thead>
<tr>
<th>Studies</th>
<th>No. of events</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disconfirming</td>
<td></td>
</tr>
<tr>
<td>Danker-Brown and Baucom (1982)</td>
<td>1</td>
</tr>
<tr>
<td>Feather and Davenport (1981)</td>
<td>1</td>
</tr>
<tr>
<td>Garber and Hollon (1980)</td>
<td>1</td>
</tr>
<tr>
<td>Gong-Guy and Hamm (1980)</td>
<td>5</td>
</tr>
<tr>
<td>Hammen and Cochran (1981)</td>
<td>5</td>
</tr>
<tr>
<td>Hammen and deMayo (1982)</td>
<td>1</td>
</tr>
<tr>
<td>Peterson and Conn (1982)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td><strong>2.14</strong></td>
</tr>
<tr>
<td>Confirming</td>
<td></td>
</tr>
<tr>
<td>Blaney, Behar, and Head (1980) – One</td>
<td>6</td>
</tr>
<tr>
<td>– Two</td>
<td>6</td>
</tr>
<tr>
<td>Eaves and Rush (1984)</td>
<td>6</td>
</tr>
<tr>
<td>Janoff-Bulman (1979)</td>
<td>4</td>
</tr>
<tr>
<td>Peterson, Bettes, and Seligman (1982) – One</td>
<td>2</td>
</tr>
<tr>
<td>– Two</td>
<td>6</td>
</tr>
<tr>
<td>Peterson, Schwartz, and Seligman (1981)</td>
<td>18</td>
</tr>
<tr>
<td>Raps, Peterson, Reinhard, Abramson, and Seligman (1982)</td>
<td>6</td>
</tr>
<tr>
<td>Seligman, Abramson, Semmel, and von Baeyer (1979)</td>
<td>6</td>
</tr>
<tr>
<td>Seligman et al. (1984)</td>
<td>8</td>
</tr>
<tr>
<td>Sweeney, Shaefller, and Golin (1982)</td>
<td>6</td>
</tr>
<tr>
<td>Zemore and Johansen (1980)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td><strong>6.25</strong></td>
</tr>
</tbody>
</table>

Note. From Peterson and Raps (1983). All studies correlated depressive symptoms with the use of internal, stable, and/or global explanations for bad events involving the self. Disconfirming studies were those that found no significant correlations ($p > .05$); confirming studies were those that found significant correlations ($p < .05$); Not all studies investigated all three explanatory dimensions. Not included are studies that found mixed support for the reformulation.
sures of explanatory style and depressive symptoms at two times, 6 months apart (Seligman et al., 1984). The cross-sectional data from this research supported the reformulation. Do the longitudinal data?

The question of interest is whether explanatory style at Time 1 predicts depressive symptoms at Time 2, above and beyond the level of depression at Time 1. As predicted by the reformulation, the depressive explanatory style for bad events was correlated with subsequent depressive symptoms, even when initial CDI scores were partialed out (partial $r = .26, p < .02$). So, explanatory style for bad events may be a risk factor for depression among children.

Nolen-Hoeksema (1983) performed a similar study using 168 children from Grades 3–5. Children who were both nondepressed in January and had a nondepressive explanatory style remained nondepressed in March. In contrast, children who were nondepressed in January but had a depressive explanatory style were likely to be depressed in March ($p < .01$).

Several recently completed studies using adults address the same question. Golin, Sweeney, and Shaefer (1981) and Firth and Brewin (1982) administered measures of both explanatory style and depressive symptoms at two times. Both studies provided support for the strong claim that explanations affect depression to a greater degree than depression affects explanations. In the Golin et al. (1981) study, causal priority of stability and globality for bad events was demonstrated for 180 college students over a 1-month period. In the Firth and Brewin (1982) study, causal priority for stability for bad events was demonstrated for 16 female patients over a 5-week period.

In another longitudinal study, O'Hara, Rehm, and Campbell (1982) administered the ASQ to 170 women in their second trimester of pregnancy, along with several other cognitive-behavioral assessments. The strongest predictor of level of depression, 3 months postpartum, was explanatory style for bad events, which significantly correlated with BDI scores even when prepartum level of depression was held constant.

In similar studies, Manly, McMahon, Bradley, and Davidson (1982) found no support for the hypothesis that explanatory style predicts depression following childbirth, whereas Cutrona (1983) replicated the findings of O'Hara et al. (1982) and also showed that explanatory style predicted speed of recovery from postpartum depression.

**Conclusion**

These studies mostly support the prediction that depressive explanatory style precedes depressive symptoms. Explanatory style may be a risk factor for later depression, allowing identification of individuals at risk by the way they explain bad events. Primary prevention of depression should then be focused on such individuals (but see Lewinsohn, Steinmetz, Larson, & Franklin, 1981, and Peterson et al., 1981).

The general flaw of these studies as tests of the reformulation is that they do not manipulate or assess bad life events. According to Abramson et al. (1978), a depressive explanatory style per se is not sufficient for depression. It is only when bad events occur and are interpreted in terms of internal, stable, and global causes that depressive symptoms are more likely to ensue. Because these studies did not look at bad events, they did not test this prediction. The studies in the next section test the next strongest proposition that a preexisting bad explanatory style followed by bad events makes depression more likely.

**Experiments of Nature**

The ideal way to test the helplessness reformulation is to measure the explanatory style of individuals, and then to choose at random half of these individuals to experience some important bad event. The most severe depression is predicted to ensue for subjects with the preexisting bad explanatory style who also then experienced the bad event. The obvious ethical dilemma can be partly solved by a quasi-experimental method in which naturally occurring bad events are the manipulation. We report two studies conducted by our research group. In the first, the bad event was an unsatisfactory grade on a midterm examination. In the second, the bad event was imprisonment in a penitentiary. We also describe several other experiments of nature that are pertinent to the reformulation.

**Study 7: Midterm Experiment**

This investigation was a prospective study of college students and their reactions to a low
grade on a midterm examination (Metalsky et al., 1982). According to the helplessness re-
formulation, students who habitually explain bad events in terms of internal, stable, and
global factors are more likely to feel depressed on learning that they received a low grade than
are students who tend to explain bad events in terms of external, unstable, and specific fac-
tors.

The participants were undergraduates in an
introductory psychology course at the State
University of New York at Stony Brook, who
completed questionnaires for this research
during classtime. At Time 1, they took the
ASQ and a questionnaire about their aspira-
tions for the class midterm. Students indicated
the grades with which they would be happy
and unhappy. At Time 2, 11 days later, just
prior to the midterm examination, students' level of depressed mood was assessed with
the Multiple Affect Adjective Check List
(MAACL; Zuckerman & Lubin, 1965), a
measure of transient depressed mood. At Time
3, another 5 days later, immediately following
receipt of the midterm grade, students again completed the MAACL.

Subjects were considered to have received
a low grade if their midterm grade was less
than or equal to the grade with which they said they would be unhappy (n = 53). Students
were considered to have received a high grade
if their midterm grade was equal to or greater
than the grade with which they said they would be happy (n = 28). To test our predictions,
explanatory style scores for bad events were correlated with standardized residual gain
scores on the MAACL depression scale from
Time 2 to Time 3, separately for the two
groups. Explanatory style for bad events pre-
dicted increases in depressed mood for stu-
dents who received low grades—internality
(r = .34, p < .02), stability (r = .04, ns), and
globality (r = .32, p < .02)—but not for stu-
dents who received high grades—internality
(r = .12, ns), stability (r = .36, p < .10), and
globality (r = .22, ns). Additional correlational
analyses revealed that explanatory style was independent of the actual grade received and
of grade aspirations. Thus, several possible confounds are ruled out.

The theoretical significance of these findings
is that they support the reformulation's claim
to be a diathesis–stress model, where the de-
pressive explanatory style is a constitutional weakness (diathesis) and the low midterm
grade is the environmental stressor (stress), which interact to predispose depressed mood
(Metalsky et al., 1982). The practical signifi-
cance lies in the possibility of identifying, in
advance, individuals at risk for depressive re-
tection to bad life events from explanatory style.

Study 8: Prison Experiment

Does explanatory style predict depression
following imprisonment (Bukstel & Kilmann,
1980)? For most individuals, imprisonment is inarguably a bad event. Like any total institu-
tion, prisons deny individuals control over
even the most mundane aspects of their lives
(Goffman, 1961; Taylor, 1979). We would ex-
pect, then, that a common reaction to im-
prisonment is depression (Seligman, 1975).
Furthermore, we predicted that prisoners with
the depressive explanatory style are the most
likely to become depressed after interment
(Abramson et al., 1978). We report a prelim-
inary study of this (Peterson, Nutter, & Selig-
man, 1982). Upon imprisonment, individuals
completed the ASQ, and shortly before release,
these same individuals completed the BDI.
Within 1 week following imprisonment at
one of four maximum security prisons in New
York, Ohio, and Pennsylvania, 245 adult males
(ranging in age from 17 to 64 years; average
age = 27 years) completed the ASQ. Within
1 week before their release, which varied from
1 month to 1 year, they completed the BDI.
For 28 prisoners, BDI scores from the time of
initial imprisonment were available. These av-
eraged only 1.68, suggesting that subjects were
not at all depressed at the time of imprison-
ment.

At the end of their imprisonment, prisoners
scored an average of 17.7 on the BDI, placing
them in the moderately to severely depressed
range (Beck, 1967). Furthermore, their de-
pressive symptoms at the end of imprisonment
were strongly associated with their explanatory
style at the beginning of imprisonment. As
expected, explanations for bad events were
positively correlated with depressive symp-
toms: internality (r = .34, p < .001), stability
(r = .36, p < .001), and composite (r = .35, p < .001). Sur-
prisingly, explanations for good events were
similarly correlated with depression: inter-

nality \( (r = .39, p < .001) \), stability \( (r = .38, p < .001) \), globality \( (r = .37, p < .001) \), and composite \( (r = .39, p < .001) \). Unlike results in our other research, explanatory styles for bad versus good events did not have opposite effects on depression. Furthermore, unlike results in our other research, explanatory styles for bad versus good events were not independent. Instead, they were substantially intercorrelated \( (all \, rs > .60; \, all \, ps < .001) \).

Other Experiments of Nature

Several other experiments of nature deserve mention. Although not designed to test the helplessness reformulation, they are intriguing and illustrate how investigations of reactions to naturally occurring bad events might be used to test the reformulation. Tennen, Affleck, Allen, McGrade, and Ratzan (1983) studied 32 children who had diabetes. Children who explained the onset of their disease in terms of their behavior (i.e., internally, unstably, and specifically; see Peterson, Schwartz, & Seligman, 1981) were rated by their physicians as coping better with diabetes than were children who explained the onset in terms of genetic inheritance (i.e., internally, stably, and globally). Although a third variable such as parental attitude may underlie these data, they are consistent with the reformulation.

Similarly, Affleck, Allen, McGrade, and McQueeney (1982) interviewed the mothers of 43 infants with severe perinatal complications. Mothers who explained the infant’s condition in terms of some behavior during pregnancy (i.e., unstably and specifically) reported less mood disturbance and anticipated fewer caretaking problems than did mothers who explained the complications in terms of other people (i.e., stably and globally). Although a third variable such as parental attitude may underlie these data, they are consistent with the reformulation.

Timko and Janoff-Bulman (1983) interviewed 42 women who had undergone a mastectomy following a diagnosis of breast cancer, asking them to offer a causal explanation for their cancer and to complete the BDI. Also assessed were other beliefs about the success of the mastectomy and the avoidability of future cancer. The helplessness reformulation was strongly supported. Causal explanations did not directly affect depression, but they did predict expectations about future cancer, which in turn predicted BDI scores. More specifically, women who explained their cancer in terms of behavior (i.e., internal, unstable, and specific causes) believed that they were cancer free; they were likely to be nondepressed. In contrast, women who explained their cancer in terms of personality (i.e., internal, stable, and global causes) believed that they were not cancer free; they were likely to be depressed.

Not all studies that have looked at the link between causal explanations and reactions to serious disease have supported the reformulation (e.g., Bard & Dyk, 1956). Factors such as the seriousness and chronicity of the disease may affect the relationship between explanation and reaction, perhaps by creating expectations about the future course of the disease incongruent with its causal explanation.

Life Events and Depression

The large research literature on life events and psychological distress is also relevant to the helplessness reformulation, although few theorists have drawn explicit parallels. However, in a recent review of this literature, Thoits (1983) proposed that many of its consistent findings are compatible with a helplessness interpretation. The modest relationship typically observed between the experience of life events and subsequent distress is increased when (a) the psychological consequence is depression as opposed to some other psychopathology and (b) the events themselves are relatively undesirable, uncontrollable, unexpected, important, and clustered together in time.

Although life events research has typically not assessed causal explanations, the types of events most frequently linked to psychological distress lend themselves to internal, stable, and global explanations. This possibility is consistent with the helplessness reformulation. Furthermore, that life events are more apt to result in depression than in other psychological disorders adds to the evidence for the specificity of the helplessness model as an account of depression (cf. Raps, Peterson, Reinhard, Abramson, & Seligman, 1982).

Conclusions

These experiments of nature suggest that the helplessness model may be useful in explaining reactions to major life events. In par-
ticular, the studies by our research group suggest that the depressive explanatory style tends to produce depression when bad events are encountered. Other studies show that particular explanations of bad events are associated with depression and poor coping. Each of these studies is open to criticism, because experimental control over the bad events was not possible, and possible confounds might underlie the obtained correlations. However, because the results of these studies converge, our confidence increases that the conjunction of depressive explanatory style and bad events predisposes subsequent depression.

Experimental studies, unlike experiments of nature, allow the bad event to be presented to randomly selected subjects. We now turn to a set of laboratory experiments that suggest that helplessness symptoms occur as predicted when uncontrollable bad events are imposed on people who differ in explanations and explanatory style.

**Laboratory Experiments**

Does explanatory style affect laboratory-induced learned helplessness in the same way that it affects naturally occurring depressive symptoms? Laboratory studies give us what experiments of nature lack. We can impose a bad event on randomly selected subjects and look at the effects of preexisting or induced causal explanations. Several studies of this sort have been done.

**Study 9: Global Versus Specific Explanations**

Alloy et al. (1984) gave subjects one of three pretreatments as described by Hiroto and Seligman (1975): a condition in which button-pushing terminated an aversive noise delivered through headphones (escapable), a condition in which the noise could not be terminated (inescapable), and a condition in which no noise—escapable or inescapable—was experienced (no pretreatment). Then, subjects were tested at one of two tasks: a task similar to pretreatment—a solvable hand shuttle-box in which success moving the hand stopped an aversive noise, and a task dissimilar to pretreatment—a series of solvable anagrams administered in a different room by a different experimenter.

<table>
<thead>
<tr>
<th>Test task</th>
<th>Global</th>
<th>Specific</th>
</tr>
</thead>
<tbody>
<tr>
<td>Escapable noise</td>
<td>2.26&lt;sub&gt;a&lt;/sub&gt;</td>
<td>1.98&lt;sub&gt;a&lt;/sub&gt;</td>
</tr>
<tr>
<td>Inescapable noise</td>
<td>3.43&lt;sub&gt;b&lt;/sub&gt;</td>
<td>3.43&lt;sub&gt;b&lt;/sub&gt;</td>
</tr>
<tr>
<td>No noise control</td>
<td>2.54&lt;sub&gt;a&lt;/sub&gt;</td>
<td>2.58&lt;sub&gt;b&lt;/sub&gt;</td>
</tr>
<tr>
<td>Escapable noise</td>
<td>16.4&lt;sub&gt;a&lt;/sub&gt;</td>
<td>17.4&lt;sub&gt;a&lt;/sub&gt;</td>
</tr>
<tr>
<td>Inescapable noise</td>
<td>41.5&lt;sub&gt;b&lt;/sub&gt;</td>
<td>20.0&lt;sub&gt;a&lt;/sub&gt;</td>
</tr>
<tr>
<td>No noise control</td>
<td>20.7&lt;sub&gt;a&lt;/sub&gt;</td>
<td>19.0&lt;sub&gt;a&lt;/sub&gt;</td>
</tr>
</tbody>
</table>

**Note.** From Alloy, Peterson, Abramson, & Seligman (1984). For similar test task, maximum latency = 5.00 s. For each condition, n = 18. For dissimilar test task, maximum latency = 100.0 s. For each condition, n = 10. Means in a row or in a column of three are different (p < .05) by Newman-Keuls procedure if they have different subscripts.

According to the reformulation, all subjects should show deficits following inescapable noise when tested on the similar noise task. However, only subjects with a global explanatory style for bad events should show deficits following inescapable noise when tested on the dissimilar cognitive task. Table 11 shows these predictions to be supported. Furthermore, the differences remain even when internality and stability scores are covariates.

This study experimentally manipulated bad events and also looked at a specific role assigned to one of the explanatory dimensions. In a related investigation (Peterson & Seligman, 1981), we obtained some preliminary evidence that stability for bad events is related to the time course of laboratory-induced helplessness as predicted by the reformulation.

**Other Laboratory Experiments**

Several other laboratory studies concern both the manipulation of causal explanations and the manipulation of bad events. Pasahow (1980) manipulated the global-specific dimension and imposed bad events on subjects in the learned helplessness triadic design. Subjects instilled with a global explanation were given written instructions that told them that the concept-identification task they were about to take correlated very highly with how people
performed on all psychology experiment tasks. Specific subjects were given written instructions that described the task as a concept-formation task that had little or no correlation with other tasks used in psychology experiments.

All of the subjects were then given unsolvable concept-identification problems and then tested on anagrams. Subjects induced to make global explanations for their failure performed worse on the anagrams than did subjects encouraged to give specific explanations. This suggests that the manipulation of explanations along the global-specific dimension for bad events appropriately predicts deficits when bad events are then imposed on subjects.

Thus, we are given converging evidence about the globality dimension for bad events. In the Alloy et al. (1984) study, preexisting explanatory style was merely measured and the bad event manipulated. Preexisting global style for bad events predicted failure at new problems once the bad event occurred, whereas specific style did not. In parallel, if causal explanations are themselves manipulated along with the bad event, Pasahow (1980) showed that the same results hold.

McFarland and Ross (1982) investigated the effect of explanations on self-esteem following failure. They assigned female college students to success or failure feedback conditions following a social accuracy test. They also manipulated the causal explanation for performance at this test: internal or external. Consistent with the reformulation, subsequent self-esteem was lowest for subjects who experienced failure and were induced to explain it internally. So, a manipulation designed to produce internal causal explanations lowered self-esteem following a bad event, whereas a manipulation designed to produce external causal explanations did not.

In a laboratory experiment with depressed inpatients, Miller and Norman (1981) told subjects who were either acutely depressed or recently improved and made helpless by in-escapable noise, that they had done well at a task measuring social intelligence. Subjects were induced to explain their success in one of four ways: with an internal–global cause, an internal–specific cause, an external–global cause, or an external–specific cause. Consistent with the reformulation, both depressed and helpless subjects reported a less depressed mood when induced to explain their success internally. Similarly, both depressed and helpless subjects performed better at a subsequent anagram task when induced to explain their success globally.

Finally, Anderson (1983) manipulated causal explanations following failure for college students preselected for explanatory style. Using his Attributional Style Assessment Test (ASAT), Anderson (1983) obtained subjects who explained failure characterologically (i.e., internally, stably, and globally) and subjects who explained failure behaviorally (i.e., internally, unstably, and specifically). These subjects then attempted to persuade other students to donate blood; explanations for failure were experimentally varied and included an ability/trait condition and a strategy/effort condition. Consistent with the reformulation, subjects who offered behavioral explanations, whether by preselection or manipulation, reported greater expectations of success, displayed higher motivation, and performed more successfully following failure than did subjects who offered characterological explanations (see also Anderson & Jennings, 1980).

Conclusions

Laboratory investigations of learned helplessness in humans have been controversial since their inception (Wortman & Brehm, 1975). In a recent review, Silver, Wortman, and Klos (1982) criticized these studies on three grounds. First, the relationship between experience with uncontrollable events and subsequent deficits may be mediated by a variety of factors, not just the expectation of response–outcome independence (e.g., Frankel & Snyder, 1978; Peterson, 1978; Tennen, 1982). Second, laboratory studies of human helplessness may be subject to demand characteristics that trivialize obtained results (Orne, 1962). Third, the laboratory setting is artificial, and generalization of results to human helplessness and depression outside of the laboratory may not be justified (e.g., Bulman & Wortman, 1977; Coyne, Aldwin, & Lazarus, 1981).

These are reasonable criticisms, and the research program described here attempts to answer them. First, as we have already stated,
the expectation of response–outcome independence is regarded as a sufficient condition for depressive deficits, not a necessary one. That other factors may also determine deficits following uncontrollability is undoubtedly true and not contrary to this theory. However, converging research shows the importance of expectations in the development of such deficits. Second, laboratory experiments may indeed be confounded by demand characteristics, but other research strategies also have inherent flaws. Thus, we favor a multimethod research strategy and a search for convergence among results (Campbell & Fiske, 1959). Finally, the only way to ascertain whether laboratory findings are artificial is to attempt studies outside the laboratory. The out-of-the-laboratory results described here are mostly consistent with the laboratory data.

These laboratory data indicate that the measurement and manipulation of explanations and explanatory style, when accompanied by the manipulation of bad events, yield what the theory predicts. The global–specific dimension, when manipulated and when measured, governs the breadth of helplessness deficits. The internal–external dimension, when manipulated, appears to govern self-esteem deficits. Finally, tentative evidence suggests that the stable–unstable dimension governs the duration of helplessness deficits.

Case Studies: Investigation of Real Lives

The final line of evidence we wish to present comes from the study of individual lives. It converges on the proposition that explanatory style is a risk factor for depressive symptoms. One major barrier in trying to study the consequences of explanatory style in the laboratory or in natural settings is the obtrusive and reactive nature of questionnaires. The development of a valid method for assessing explanatory style through the content analysis of written transcripts allows us to study, in a wholly unobtrusive and nonreactive way, the natural history of explanatory style and depression. We now present several studies that use this technique. The first two studies analyze verbatim transcripts of actual psychotherapy sessions with depressed patients. The third study analyzes verbatim interviews originally conducted decades ago with individuals for whom measures of current psychological characteristics are available.

Study 10: Causal Explanations and Psychotherapy

In the first study (Peterson & Seligman, 1981), each causal explanation for a bad event was extracted from 300-word excerpts from verbatim transcripts of individual psychotherapy sessions. Transcripts were available from the beginning, middle, and ending of successful psychotherapy with four different patients suffering depression following a loss. We blindly rated these explanations for internality, stability, and globality, and averaged these scores across explanations offered within the same excerpt. For each patient, explanatory style perfectly ordered the three sessions; the most internal, stable, and global explanations were offered in the beginning session, whereas the least internal, stable, and global explanations were offered in the ending session. These data supplement the Persons and Rao (1981) study already described in suggesting that changes in explanatory style index changes in depression during psychotherapy.

Study 11: Causal Explanations and Mood Swings

In the second study (Peterson et al., 1983), we used the symptom–context method, a content–analytic approach developed by Luborsky (1964, 1970), to investigate the explanatory antecedents of mood swings by a single patient during psychotherapy sessions. With the symptom–context method, context is assessed immediately before and after the onset of the symptoms of interest. In this case, the context was causal explanations for bad events, and the symptoms were mood swings—in and out of depression during therapy sessions.

Our subject was a patient, Mr. Q, who demonstrated precipitous shifts in mood during psychotherapy sessions (conducted over 4 years). More than 200 sessions with Mr. Q were tape-recorded, and a sample was transcribed, allowing a thorough content analysis of his explanations before and after mood swings. Three types of sessions were analyzed: those in which Mr. Q became more depressed, those in which he became less depressed, and
those in which no shift at all occurred. Explanations for bad events were extracted from these sessions before and after the mood swings, and were rated for internality, stability, and globality, as described above.

Several criteria were used to identify Mr. Q’s swings to and from depression: (a) Mr. Q’s report of a shift in mood (e.g., “My mood just went down”), (b) the agreement of two independent judges reading the transcript of the therapy session that a mood swing had just occurred, and (c) the fact that the swing did not occur in close proximity to another swing (i.e., within 15 min of the same session). Applying these three criteria resulted in a final set of four swings in which the patient became more depressed and five swings in which he became less depressed. These mood swings were validated by both the agreement of two independent judges listening to the taped therapy sessions that Mr. Q’s voice showed a change in affective quality and the scoring by an independent judge using the depression component of the Gottschalk and Gleser (1969) hostility-inward scale.

Explanations for bad events were extracted from the 400 words (spoken by Mr. Q) before the swing and from the 400 words following the swing. For comparison purposes, causal explanations were also extracted from randomly chosen 800-word segments of three sessions in which no swing occurred. In all, 63 bad events linked to causal explanations were rated for internality, stability, and globality by four independent judges blind to their source. Ratings by the judges were combined, and composites were formed. Means for the different types of sessions are shown in Figure 2.

The differences predicted by the reformulation were present before swings in mood. Highly internal, stable, and global causal explanations preceded increased depression, whereas much more external, unstable, and specific statements preceded decreased depression. There was no overlap between the ratings of causal explanations before swings to more versus less depression.

This is a fine-grained case study, but because it is correlational, it is necessary to rule out some plausible third-variable arguments to conclude that explanations, and not some factor correlated with them, produced mood swings. Various content characteristics of these same transcripts, including Mr. Q’s statements about anxiety, hopelessness, guilt, hostility to self, loss of self-esteem, and Oedipal conflict, were examined. These were not redundant with the causal explanation scores, rising and falling with level of mood in a given session. In contrast, explanations were different before swings.

This study shows that the reformulation may be applied powerfully to specific individuals with depressive symptoms. Explanation ratings for sessions in which swings to increased depression occurred did not at all overlap with explanation ratings for sessions in which swings to decreased depression occurred, allowing perfect prediction.

Study 12: Individuals of the Berkeley–Oakland Study

We now describe a preliminary study using what we have dubbed the *time machine* method. While conducting content analysis of causal explanations provided in essays and therapy transcripts, we realized that there was
no reason to limit ourselves to contemporary data. Content analysis can be used with any written material, including newspaper stories, autobiographies, transcribed interviews, letters, diaries, and so on (Simonton, 1981). So, we decided to use content analysis to extract causal explanations from open-ended material residing in already-existing data archives. The explanatory style of individuals years ago can be assessed through content analysis, and the relationship between causal explanations and later characteristics of these individuals can be ascertained. Prospective longitudinal research spanning decades is possible retrospectively.

Our first attempt to study individual lives over decades analyzed material in the Berkeley–Oakland Growth Study (Elder, Bettes, & Seligman, 1982), an ongoing investigation of the effects of the Great Depression on family and life patterns (Elder, 1974). Although economic hardships of the Great Depression hit both the lower class and the middle class, the lower class was hit harder. Indeed, middle-class individuals who suffered financial hardships during the Great Depression years later were happier, healthier, and more efficacious than middle-class individuals who had not been economically deprived (Elder, 1974). Does explanatory style mediate this interaction between social class and economic hardship?

Causal explanations were extracted from the open-ended responses of 28 women who described the worst events during the preceding year in both 1943 and 1970. The focus of this study was on hopelessness—the use of stable and global causes to explain bad events—so these explanations were rated for stability and globality, but not internality. A hopeless outlook in 1943 was significantly correlated with a hopeless outlook in 1970 (r = .44, p < .05), through which it affected overall psychological health (assessed by a Q-sort procedure) in 1970.

More detailed analyses suggested that hardships in 1929 predicted hopelessness in 1943. Hardships per se had no direct effect on psychological health in 1970. Only when hardships gave rise to a hopeless outlook was psychological health affected for the worse decades later. Furthermore, these patterns seem more pronounced among the lower class than among the middle class. The small sample size renders this conclusion highly tentative, but it appears as if middle-class individuals were not so prone to react to economic difficulties with hopelessness, perhaps because they had available to them means for coping with difficulties. The helplessness model proposes that early mastery experience immunizes one against the bad effects of uncontrollability (Maier & Seligman, 1976); these data from the Berkeley–Oakland Study may illustrate immunization.

In an even more preliminary investigation using the time-machine methodology, we analyzed interviews from the Grant Study, a longitudinal investigation of the Harvard classes of 1939–1942. Over the 40 years of this research, the material success, social success, mental health, and physical health of these men have been recorded, with special interest in whether such psychological characteristics as defense mechanisms predict future life course (Vaillant, 1977). A great amount of open-ended interview material is available for each individual studied. We extracted causal explanations for bad events from a 1946 interview about war experiences conducted with 18 men. Explanations were rated for internality, stability, and globality, as in our other studies.

These ratings were correlated with a global rating of physical health in 1980: 1 = healthy; 2 = minor health problems; 3 = chronic illness; 4 = disabled; and 5 = dead. Because of the small sample size, we report only the correlation with the composite explanatory style. It was positive (r = .40, p < .10). Depressive explanatory style in 1946 predicted poor physical health 34 years later. The tentative nature of this study must be emphasized. Sample size is small, and the obtained correlation is of borderline statistical significance. The dependent measure assessed overall physical health, not level of depression, and we do not know what the mechanism linking explanatory style and health might be.

Both of these investigations are preliminary, and we are currently analyzing the causal explanations in a greater number of interviews. Nevertheless, we have described these still-in-progress studies because they suggest that the helplessness reformulation can be applied to real people over the life span.

General Conclusions

We have described five interlocking types of research investigating the attributional re-
formulation of the helplessness model of depression. The cross-sectional studies showed that a characteristic way of explaining bad events with internal, stable, and global causes co-occurs with depressive symptoms. The longitudinal studies showed that this explanatory style preceded the development of depressive symptoms. The experiments of nature indicated that this style resulted in depression once bad events were encountered. The laboratory experiments showed that imposing uncontrollable bad events on individuals making particular explanations had the predicted effects on helplessness deficits. Finally, the case studies illustrated that the reformulation applies predictively to the depressive symptoms of specific individuals.

Taken together, these five lines of research support the predictions of the attributional reformulation of the learned helplessness model of depression. Each type of research is open to criticism on several grounds, but the convergence of results across different strategies of investigation, different operationalizations, and different populations argues strongly for the validity of the reformulation (Campbell & Fiske, 1959).

There are aspects of the reformulation still in need of theoretical and empirical scrutiny, and we briefly discuss these now.

1. **The role in depression of causal explanations of good events.** The helplessness reformulation does not explicitly address such causal beliefs, but they seem to have at least a weak empirical relationship with depressive symptoms. We have assessed such beliefs and generally found them to have effects on depression opposite to those of causal explanations about bad events, but at less robust levels. However, some of the findings do not fit these generalizations. In our longitudinal investigation of children, explanatory style for bad events predicted subsequent depression, whereas style for bad events did not. In our study of prisoners, the explanation of good events with internal, stable, and global causes was positively, not negatively, correlated with later depression. A sound theoretical treatment is awaited.

We speculate that our pattern of findings with children may be related to the sorts of events that adults take for granted that children do not. When do we engage in explanatory analysis, and when do we forego it? Peirce (1955) suggested that the function of thought is to allay doubt (see also Wong & Weiner, 1981). It seems likely that we ask **why** more often when we are in doubt—when our path is suddenly obstructed—than when things are going smoothly. Indeed, consciousness itself may emerge only when our routine actions fail (cf. Langer, 1978). This implies that the explanatory analysis of failure among adults would be much more articulate than would the analysis of success. Children, on the other hand, newer at the game, may analyze the causes of both failure and success closely. Such a developmental process would render children’s explanatory style for good events more useful than that of adults in predicting depression.

2. **The origins of explanatory style.** The helplessness reformulation is silent about antecedents. We are turning our research attention to this issue, and it promises to be exciting. Several preliminary findings are worth mentioning. First, in our study of explanatory style and depressive symptoms among children (Seligman et al., 1984), we obtained corresponding scores from the mothers of 47 children in our sample and from the fathers of 36. We found that explanatory style for bad events and depressive symptoms were correlated for mothers and their children: explanatory style \(r = .39, p < .01\) and depression \(r = .37, p < .01\). Mothers’ explanatory style also correlated with children’s depression \(r = .42, p < .005\), whereas children’s explanatory style weakly correlated with mothers’ depression \(r = .27, p < .10\). Scores for fathers were unrelated to those of their children and to those of their mates. In our content analysis of the Berkeley–Oakland Study, explanatory style of mothers and sons (but not of mothers and daughters) also converged.

There are two other important hints about the origins of explanatory style. In addition to learning it from one’s mother, one may learn from the type of criticisms leveled by teachers. Dweck and Licht (1980) looked at the explanations that teachers gave in the third grade when boys and girls failed in the classroom. They found that teachers criticized girls with internal, stable, and global statements. Boys, on the other hand, were criticized with more unstable and specific explanations, such as “You’re being rowdy”, “You’re not concentrating.”
In other research, Dweck and Licht (1980) gave fourth grade boys and girls unsolvable problems and showed that boys were less susceptible to helplessness than were girls. Boys also tended to give less internal, less stable, and less global explanations for their failures on these tasks than did girls. Boys said: “I wasn’t trying hard”, “I wasn’t paying attention”, and “I don’t care about your test.” Girls attributed their failure to incompetence and stupidity. Dweck and Licht (1980) argued that the negative explanatory style of the girls relative to the boys may originate from learning their teachers’ criticisms. That women show more depressive symptoms as adults than do men (Radloff, 1975) may indicate that they have learned an explanatory style as girls that makes them vulnerable to depression as women.

Finally, the reality of one’s first trauma may play an important role in setting explanatory style. Brown and Harris (1978) interviewed lower- and middle-class women in South London and found that almost 20% of the lower-class women showed severe symptoms of depression. An unusually high percentage of these depressed women had lost their mothers by death before they were 11 years old. The death of mother for a young girl, as opposed to the death of mother for a teenager, has more stable consequences. In addition, for a young girl, as opposed to a teenager, a large part of her repertoire (global) is wiped out when her mother dies. It may be the case, then, that because a mother dying represents a stable and global loss for a young girl, and because her personal helplessness is thereby underscored, that as an adult she imposes internal, stable, and global explanations on other bad events, rendering her vulnerable to depression.

In summary, then, there are at least three means by which explanatory style may be acquired. First, one may learn it by imitating parents, particularly the primary care giver, in our society the mother. Second, the criticisms that are launched at one by teachers following failure may teach an explanatory style. Finally, the reality of one’s first traumatic loss—the extent to which it is actually internally, stably, and globally caused—may set one’s explanatory style for life.

3. Explanatory style as a trait. We view explanatory style as a trait, analogous to liberalism in politics or vanity in interpersonal relations. We believe this for three reasons. First, the ASQ is designed to look at explanations given across situations: half bad, half good. An explanatory style is derived from cross-situational consistency, and this is part of what is meant by trait. Second, some people have more or less consistency across situations. For example, some individuals average 5.0 on stability for bad events because they answer 5 to each of the six bad situations for stability, whereas others average 5.0 because they chose three 7s and three 3s. These latter individuals can hardly be said to have a trait with respect to stability. When we calculate the standard deviation of ratings for each dimension and discard those individuals who have large standard deviations, we find that correlations with depression and other outcomes go up. Third, explanatory style shows fairly high stability across time.

Although we believe explanatory style is traitlike, we do not believe it is invariant—nor, incidentally, are traits like liberalism and vanity. We have two reasons for taking the plasticity of explanatory style seriously. First, although we have argued that explanatory style affects depression, we also suspect that depression affects explanatory style. Persons and Rao (1981) found that explanatory style changed for the better among patients as their depression lifted. So also did Castellon et al. (1982) and Hamilton and Abramson (1983). Therapy and other good or bad life events may change explanatory style (Seligman, 1981). We do after all acquire it at some time in our lives. Second, some individuals may display consistent explanations of a given type at one time and consistent explanations of another type at another time. Mr. Q’s explanations, for example, predicted his depressive mood shifts, but they did not cohere into a style. On some days, he explained bad events internally, stably, and globally, whereas on other days, he explained them externally, unstably, and specifically.

Future investigations should adopt a more sophisticated view of explanatory style. It should be treated as a dependent variable that can be modified by life events, as well as an independent variable that modifies future events. Also, the likelihood that there is a bidirectional influence between depression and explanatory style should be explored. And fi-
nally, the term *style* should be reserved for individuals whose causal explanations show low variability across time and situations.

4. *Seldonics.* There is one implication of our research program that we believe to have uncommon promise. We call it Seldonics, after Hari Seldon (11,988–12,069 G.E.), the fictional psychohistorian who developed a science “that deals with the overall reactions of large groups of human beings to given stimuli under given conditions” (Asimov, 1982, p. 12). By assuming a large enough group of people (and a set of behavioral laws and mathematical relations left to the reader’s imagination), Seldon founded a powerful psychohistory that predicts, for human conglomerates, crises and their resolutions far into the future. No such claims are made here.

However, the technique of verbatim analysis has intriguing possibilities. First, it can be reliably done. Second, it converges with a questionnaire that seems to predict which human conglomerates will react to failure with passivity and depression and which will react with active coping attempts and good cheer. Third, it may predict when a group reacts to failure with helplessness (Berkeley–Oakland Study), and it did predict with accuracy when one individual (Mr. Q) became depressed and when he became nondepressed. Finally, it is wholly unobtrusive and nonreactive, able to be used with individuals quick or dead or otherwise impractical to test with questionnaires.

Let us imagine that one wants to predict (or postdict) whether a terrorist will react to the bombing of his headquarters with redoubled terrorist acts or with passivity. Similarly, imagine that one wants to predict (or postdict) how a human conglomerate like a given salesforce will react to a bad event like an unsuccessful product line. Will this salesforce sell more versus less of the established product lines than will a second salesforce?

All that is needed to attempt these predictions is (a) prior verbatim material (if the ASQ has not been administered) such as diaries, letters, high school compositions, therapy transcripts, and quotes from the newspaper, in the case of individuals, or a composite of such material, in the case of conglomerates; (b) coding and analysis of the explanatory profile for failure contained in this material; and (c) reasonable operationalizations of failure and of coping. Our speculations are testable, and if true, they would be consistent with Hari Seldon’s program.

To conclude, we have described the empirical support so far obtained for the helplessness reformulation. Results to date support the model. We have briefly sketched some areas in need of further theoretical and empirical scrutiny. The attributional reformulation promises to be a useful account of depression. Explanatory style, in conjunction with actual bad events, precede the development of depressive symptoms. Attention to them may be a practical means of predicting who is at risk for depression. Assaying them may help diagnose depression with specificity and sensitivity. And interventions directed at changing them may be an effective means of combating depression.

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