

Impact of Perceived Self-Efficacy in Coping With Stressors on Components of the Immune System

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This experiment examined the impact of experimentally varied perceived self-efficacy in exercising control over stressors on components of the immunological system. Immunological changes while coping with phobic stressors were measured within an intrasubject control design that included a baseline phase, an efficacy-acquisition phase, and a maximal-efficacy phase. In each of these phases, perceived coping self-efficacy, level of autonomic and endocrine activation, and several components of the immunological system were measured. Development of strong perceived self-efficacy to control phobic stressors had an immunoenhancing effect. A slow growth of perceived self-efficacy, heart rate acceleration, and cortisol activation attenuated immunological system status during the efficacy-acquisition phase. Rapid growth of perceived self-efficacy also predicted maintenance of immunoenhancement during the maximal perceived self-efficacy phase.

The recent years have witnessed a major shift in the perspective on health. The biopsychosocial perspective (Engle, 1977), which focuses on the interactive influence of psychosocial and biological factors on health and illness, is replacing the traditional biomedical model, which focuses on pathogens and somatic dysfunctions. This broadened perspective has fostered diverse lines of research designed to clarify the role of personal-

ity factors in health functioning (Matarazzo, Weiss, Herd, Miller, & Weiss, 1984; Rodin & Salovey, 1989; Taylor, 1990). One can distinguish between two levels of research on the psychosocial determinants and mechanisms of health and illness (Bandura, in press). The more basic level aims to elucidate the mechanisms through which psychosocial factors affect biological systems that mediate health and illness. The second level of research concerns the exercise of behavioral control over modifiable aspects of health.

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Stress has been implicated as an important contributing factor to many physical dysfunctions (Goldberger & Breznitz, 1982; Krantz, Grunberg, & Baum, 1985). Recent investigations with animals identified controllability as a key organizing principle regarding the nature of stress effects. Exposure to stressors with a concomitant ability to control them has no adverse effects. However, exposure to the same stressors without the ability to control them activates neuroendocrine and opioid systems and impairs various components of the immune system (Coe & Levine, in press; Maier, Laudenslager, & Ryan, 1985; Shavit & Martin, 1987).

Although the impact of perceived control on human health outcomes has been the subject of much research (Langer, 1983; Peterson & Stunkard, 1989; Rodin, 1986; Schulz, 1976), the influence it exerts on the immune system has received little attention. Our understanding of the effects of uncontrollable stressors on immunocompetence is based mainly on experimentation with animals involving uncontrollable physical

stressors. Stressors take diverse forms and can produce different patterns of physiological activation. This places certain limitations on extrapolation of conclusions across different species, stressors, and patterns of controllability. Uncontrollable physical stressors are not only stressful but also inflict some physical trauma that can activate a variety of complicating physiological processes. Most of the important stressors with which humans have to cope involve psychological threats (Lazarus & Folkman, 1984). Moreover, stress reactions are governed largely by perception of coping self-efficacy rather than being triggered directly by the objective properties of threats and environmental demands (Bandura, 1988b). It is the perception of environmental threats as exceeding one's coping capabilities that becomes the stressful reality. Research into the immunological effects of inefficacious control, therefore, needs to be broadened and extended to events and psychological processes that have high ecological relevance to human coping.

Efforts to determine the immunologic effects of psychological stressors in humans have relied extensively on correlational or quasi-experimental studies in which occurrences of life stressors are related to the incidence of infectious illnesses or to indexes of immunologic functioning (Jemmott & Locke, 1984; O'Leary, 1990; Palmblad, 1981). Exposure to stressors is usually accompanied by impairment of the immune system (Kiecolt-Glaser & Glaser, 1987). Evidence also suggests that improving people's capabilities to ameliorate stress reactions may be immunoenhancing (Kiecolt-Glaser et al., 1986, 1985). Although these lines of research have clarified some aspects of inefficacious control of stressors, experimental studies are needed to verify the direction of causality.

The present research investigated the impact on human immune functioning of experimentally varied perceived self-efficacy in coping with a psychological stressor. Human coping involves an important feature that is rarely systematically examined in either animal laboratory paradigms or human field studies. In animal experimentation, controllability is usually studied as a fixed dichotomous property in which animals either exercise complete control over physical stressors or they have no control whatsoever. In contrast, human coping usually entails an ongoing process of developing and reappraising one's coping efficacy rather than unalterable self-efficacy in the face of unremitting bombardment by stressors. Most human stress is activated in the course of learning how to exercise control over recurring stressors. Stress activated in the process of acquiring coping self-efficacy may have very different effects than stress experienced in aversive situations with no prospect in sight of ever gaining any self-protective efficacy. It would not be evolutionarily advantageous if acute stressors invariably impaired immune function, because of their prevalence in everyday life. If this were the case, people would experience high vulnerability to infective agents. The present experiment was designed to provide a refined analysis of how gaining a sense of coping mastery over stressors affects immune function.

In social cognitive theory (Bandura, 1986, 1988b), perceived self-efficacy to exercise control over potentially threatening events plays a central role in stress reactions. Threat is not a fixed property of situational events. Rather, it is a relational property concerning the match between perceived coping capabilities and potentially hurtful aspects of the environment. Peo-

ple who believe they can exercise control over potential threats do not conjure up apprehensive cognitions and are not stressed by them. But those who believe they cannot manage threats experience high levels of stress.

That perceived coping efficacy operates as a cognitive mediator of stress has been tested by creating different levels of perceived coping self-efficacy and relating them at a microlevel to different manifestations of stress. Perceived inefficacy in controlling psychological stressors is accompanied by high levels of subjective stress, autonomic activation, and plasma catecholamine secretion (Bandura, Reese, & Adams, 1982; Bandura, Taylor, Williams, Mefford, & Barchas, 1985; Ozer & Bandura, 1990). The combined results from the different manifestations of stress are consistent in showing that stress reactions are low when people cope with stressors in their perceived self-efficacy range. Self-doubts in coping efficacy produce substantial increases in subjective stress and physiological activation. After perceived coping self-efficacy is strengthened to the maximal level, coping with the previously intimidating tasks no longer elicits differential physiological activation. Perceived self-inefficacy in exercising control over stressors also activates endogenous opioid systems (Bandura, Cioffi, Taylor, & Brouillard, 1988). The latter effect is of special interest in light of evidence that some of the immunosuppressive effects of inefficacy in controlling stressors, such as reduced natural killer cell cytotoxicity, are mediated by release of endogenous opioids (Shavit & Martin, 1987). When opioid mechanisms are blocked by an opiate antagonist, the stress of coping inefficacy loses its immunosuppressive power.

Because physiological systems are highly interdependent, the types of biological reactions that have been shown to accompany perceived coping inefficacy are involved in the regulation of immune systems. Hormonal mediation of immune function has received some study (Borysenko & Borysenko, 1982; Coe & Levine, in press). Uncontrollable stressors increase the release of corticosteroids and catecholamines. Elevated levels of corticosteroids inhibit lymphocyte metabolism, interfere with the process of lymphocyte proliferation (Borysenko & Borysenko, 1982), and inhibit production of interferon and Interleukin-1 (Besedovsky, del Rey, Sorkin, & Dinarello, 1986; Munck, Guyre, & Holbrook, 1984). Increased levels of cortisol have been associated with decreased lymphocyte response to mitogen stimulation and diminished ability to destroy foreign cells (Claman, 1972). However, corticosteroids may have differential effects on immune function depending on their level. These and other findings support a mediating role of cortisol in immunologic response to stressors, although the form of the relation requires further specification.

Results also yield some support for autonomic regulation of the immune system. Several mechanisms of operation have been proposed: They include release of catecholamines resulting in redistribution of lymphocytes into the bloodstream and reduced lymphocyte response to mitogens (Crary, Borysenko, et al., 1983; Crary, Hauser, et al., 1983; Landmann et al., 1984), changes in splenic activity related to levels of norepinephrine (Besedovsky, del Rey, & Sorkin, 1985), autonomic secretion of neuroregulatory peptides that alter the cellular environment (Maclean & Reichlin, 1981), and direct autonomic innervation of lymphoid organs (Williams et al., 1981). To help clarify possi-

ble mediating linkages under changing controlling self-efficacy, this experiment included measures of endocrine and autonomic activity.

In studying the immunologic effects of differential levels of perceived coping self-efficacy, we used an experimental paradigm combining a strong phobic stressor with a powerful efficacy-induction procedure that has shown promise for clarifying other psychobiological linkages (Bandura et al., 1982; Bandura et al., 1985). This paradigm permits examination of causal relationships under laboratory conditions with a high degree of control. Because a high sense of controlling self-efficacy can be quickly instilled, it is possible to create conditions incorporating a chronic stressor with differential levels of perceived controlling self-efficacy. In the mastery phase of the study, the phobia is eradicated, so that all participants gain lasting relief from a chronic stressor while contributing to knowledge.

In the present experiment, immunologic changes in snake phobics with a low sense of coping efficacy were measured under three conditions within an intrasubject control design. These conditions included a baseline control phase involving no exposure to the phobic stressor, a perceived self-efficacy-acquisition phase in which subjects were exposed to the phobic stressor as they attempted to gain a sense of coping efficacy, and a perceived maximal self-efficacy phase in which they coped with the same phobic stressor for the same duration after they had developed a complete sense of coping efficacy. In each of these phases, we measured strength of perceived coping self-efficacy, heart rate acceleration, cortisol activation, and several components of the immunologic system.

On the basis of evidence that uncontrollable stress operates as an immunosuppressant, it would be predicted that immune functions should be attenuated during perceived coping inefficacy but restored during maximal perceived self-efficacy. However, in this experiment, perceived coping self-efficacy is a changing, rather than a fixed, property. Evidence that acquisition of skills to control stress can be immunoenhancing (Kiecolt-Glaser et al., 1986, 1985) suggests that the direction and magnitude of immunological change is related to the rate of growth of perceived coping self-efficacy. Attenuation of immune function would be associated with slow growth of perceived coping self-efficacy and high levels of autonomic and endocrine activity.

Method

Subjects

The subjects were 20 severe snake phobics, 19 women and 1 man, recruited through radio, television, and newspaper announcements. They ranged in age from 25 to 48 years, with a mean age of 37 years. The experiment was conducted in two laboratories using identical procedures. Of the total sample, 15 subjects were studied at Stanford University, 5 were studied at Rutgers University.

Subjects were screened by telephone to exclude those 49 years or older because of immunological changes in later years (Schleifer, Keller, Bond, Cohen, & Stein, 1989). In addition, those who took medication likely to affect immune function—such as antihypertensives, antihistamines, anti-inflammatory drugs, and antidepressants—were also excluded. Forty-six people were excluded because they exceeded the age level or were on medication.

The subjects' phobic dread of snakes seriously impaired and constricted their lives and created continual distress for them. They avoided social, recreational, and vocational activities that might have brought them into contact with a snake, however remote the possibility, such as hiking, camping, gardening, bicycling, swimming in lakes, or traveling to rustic areas. The most pervasive aversive consequences of the phobia were thought-produced distress. They were plagued by recurrent intrusive thoughts of dreadful encounters with snakes. Even a picture or the mere mention of a snake would trigger perturbing ruminative thoughts over which they could exercise little control. They all suffered recurrent nightmares in which they were threatened and pursued by menacing snakes. The phobic threat was clearly a strong chronic stressor.

Monitoring and Control of Health-Related Behavior

Physical and health factors known to affect immune function were closely monitored and, where possible, controlled. Prior to each session, subjects recorded on a standard form any recent illnesses, medications they had taken, changes in their weight and quantity and quality of sleep, amount of exercise, consumption of caffeinated coffee, alcoholic beverages, and cigarettes, use of birth control pills, and the menstrual cycle phase. Subjects exhibited no significant differences in these health-related factors across the three phases of the experiment. To standardize controllable factors that could affect immune function, for 24 hours before the baseline session and each of the subsequent sessions, subjects were instructed to restrict their consumption of caffeine and alcohol and to limit their level of exercise. Their recorded reports indicate they followed these instructions. Because menstruation is accompanied by hormonal changes, we assessed the menstrual cycle phase. There was no confounding of menstrual cycle phase across the three phases of the experiment.

Pretest Assessment

Behavioral test. Subjects were tested for the severity of their phobic behavior with a series of 21 coping tasks requiring increasingly more threatening interactions with a corn snake. The set of tasks required subjects to approach the caged snake, to place their hands in the cage, to touch and hold the snake with gloved and bare hands, to let it loose in the room and return it to the cage, to hold it within 12 cm of their face, and, finally, to tolerate the snake crawling in their lap while they held their hands at their sides.

The behavioral test was discontinued when the subject could not complete a performance task in the hierarchical series. Those who could place a bare hand in the cage were considered insufficiently phobic and were not included in the experiment. All subjects who met this criterion participated in the study. The sample of subjects included in the study were severely phobic. Seventy-five percent of them could not even come close to the glass cage; the remainder could approach the cage but were not about to place their hands in it.

Perceived coping self-efficacy. Subjects were provided with the list of coping tasks included in the behavioral test and instructed to rate the strength of their capability to perform the different coping activities. They rated the strength of their perceived self-efficacy on a 100-point scale, ranging in 10-unit intervals from high uncertainty (0), through intermediate values of certainty, to complete certitude (100). The mean strength of perceived self-efficacy was computed by summing the magnitude scores across coping tasks and dividing the sum by the total number of tasks. The test-retest reliability for this measure was found to be high ($r = .86$) in previous studies (Bandura, Adams, & Beyer, 1977).

Subjects' perceived self-efficacy was measured before and after the test of phobic behavior. They exhibited a uniformly weak sense of

coping efficacy before the behavior test ($M = 11$), and they remained highly inefficacious after the test ($M = 13$).

Intragroup Control Design

The experiment included three phases. In the initial baseline phase, subjects were tested without any exposure to the phobic stressor to provide a control period against which to evaluate the effects of differential levels of perceived coping self-efficacy on immune function. In the self-efficacy-growth phase, subjects coped with the phobic stressor in the context of acquiring a sense of efficacy. In the final maximal-self-efficacy phase, subjects coped with the same stressor after their perceived self-efficacy had been raised to the maximum level. An intergroup control design was not used because it would be unrealistic to expect subjects to commit large blocks of time during working hours, either unoccupied or filled with irrelevant activities, restrict exercise and consumption of coffee and alcohol preceding the sessions, and repeatedly undergo multifaceted physical and psychological assessments over lengthy sessions without any alleviation of their phobic dysfunction.

Note, however, that the present experiment is founded on a large body of prior research using the same mode of efficacy induction and the same measures of perceived self-efficacy with the same phobic threat (Bandura, 1988a). The links between the efficacy-induction procedure; changes in perceived self-efficacy; and subsequent changes in subjective distress, coping behavior, autonomic activation, and catecholamine secretion have already been verified in these prior controlled studies. Moreover, matched phobics in control conditions who do not receive the efficacy-induction intervention achieve little or no change in perceived self-efficacy, anxiety arousal, or coping behavior (Bandura et al., 1977; Bandura, Blanchard, & Ritter, 1969; Blanchard, 1970).

The sessions were conducted in the mornings at identical time periods to control for any circadian effects on immune and endocrine function. Moreover, to control for any possible weekday effects, all subjects except one also completed all phases of the experiment on the same weekday for the 3 successive weeks. Each phase of the study was separated by 1 week to eliminate any possible carryover effects on immune function from one phase to another. Female experimenters conducted the sessions in the two laboratories. Except for the higher cortisol levels in the Rutgers subsample, there were no significant differences between the subgroups from the two laboratories on any of the measures at any phase of the experiment.

Baseline Phase With No Exposure to Stressors

At least 1 week after subjects had completed the screening procedures, they participated in the control session to provide a baseline of immune function and other aspects of psychobiological functioning in the absence of any exposure to the stressor. The following baseline measurements were conducted.

Autonomic function. To provide an index of autonomic activation, subjects' heart rate was measured throughout the 1-hour baseline session following a 5-min adaptation period. A cardiac holter monitor with a tape readout provided the mean heart rate for every 1-min interval.

Immunological indexes. Thirty cc's of blood were drawn for the immunological assays at the end of the baseline session. Total number of lymphocytes and helper and suppressor T cell numbers were used to provide a general measure of immune integrity. T cells play a prominent regulatory role in the immune system. They mediate hypersensitivity reactions, destroy cancerous cells and viruses, and regulate the activity of the humoral immune system. The total number of lymphocytes and subsets of T4 helper and T8 suppressor cells were measured

through the process of cellular immunofluorescence at the Stanford University Blood Bank using a Fluorescence Activated Cell Sorter (FACS) machine (R. A. Hoffman, Kung, Hansen, & Goldstein, 1980). The ratio of T4 cells to T8 cells, a clinically significant index of the modulation of immunity, was derived from these values. Expression of the Interleukin-2 receptor was measured during the cellular immunofluorescence process for enumerating lymphocytes and T cell subsets. Expression of Interleukin-2 receptor is an indication of activation, generally assumed to reflect exposure to antigenic stimulation. HLA-DR is a receptor found on B cells and on activated T cells. Because only about 1% of cells with HLA-DR are T cells, HLA-DR can be taken to be a measure of B cell numbers.

Endocrine function. Saliva cortisol was used to measure activation of the pituitary-adrenal cortical system, a highly sensitive marker of psychological stress (Baum, Grunberg, & Singer, 1982; Levine, 1983). The saliva samples were obtained after the adaptation period and at the end of the session. This hormone is a stable molecule in saliva that is highly correlated (.89) with plasma cortisol levels (Umeda et al., 1981). Cortisol concentrations in saliva have been found to be independent of flow rate (Riad-Fahmy, Read, Walker, & Griffiths, 1982). Because stress can reduce salivation, subjects were given a lemon drop to stimulate the flow of saliva. The same quantity of saliva was collected across subjects and phases of the experiment. The saliva was spun, and the supernatant was frozen at -5°C . Saliva cortisol was measured in triplicate by radioimmunoassay (Klemm & Gupta, 1975).

Perceived self-efficacy. At the end of the baseline session, subjects' strength of perceived self-efficacy was measured with the efficacy scale described earlier.

Psychosocial measures. Several measures of psychosocial factors that might affect immune function were also administered at the end of the baseline session. They included a perceived stress scale that measured the degree to which people perceive situations in their life as stressful (Cohen, Kamarck, & Mermelstein, 1983). The University of California, Los Angeles Loneliness Scale measured degree of satisfaction with social activities and relationships (Russell, Peplau, & Cutrona, 1980). An anger expression scale that assessed the degree to which people suppress or express angry feelings (Speilberger et al., 1985) and the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) were also administered. The subjects did not exhibit high levels of psychosocial dysfunction on these inventories, which attenuates correlations by the restricted range of scores. Indeed, these measures showed little consistent relationship to degree of change in immune status.

Anticipatory stress. A major component of stress over potentially threatening events arises from anticipatory self-arousal and thoughts of one's coping inefficacy rather than only by actual encounters with threats (Bandura, 1986; Bandura et al., 1988). Therefore, to measure the level of anticipated activation, subjects recorded their level of anticipatory stress on rating scales on each of the 2 days immediately preceding the second phase, involving exposure to the stressor while acquiring perceived coping self-efficacy. These scales measured level of anxiety and intrusive thoughts about reptiles, apprehensiveness over the forthcoming exposure to a snake, and general physical tension. On each of the two days, subjects rated their stress reactions in the morning, afternoon, and evening. In addition, they recorded any sleep disturbance and any dreams about reptiles. These measures were designed to evaluate the level of anticipatory stress subjects experienced as they were about to confront their phobic threat.

Exposure to Stressor While Acquiring Perceived Coping Self-Efficacy

In the self-efficacy induction phase of the experiment, all subjects participated in a 2-hr session of structured activities designed to enable

them to confront and cope with the snake. The experimenter first modeled effective coping strategies and then provided guided mastery experiences by enlisting mastery performance aids to enable subjects to engage in progressively more threatening interactions with the phobic object (Bandura, Jeffery, & Wright, 1974). The coping activities were reduced to graduated tasks of easily mastered steps; joint performance with the experimenter enabled the participants to perform tasks they would not considering doing on their own; graduated times of performance emboldened the participants to risk threatening tasks they would have refused if they had to perform them for a long time at the outset. As they increased their sense of efficacy, the duration of coping involvement was extended; physically protective aids were also introduced, if necessary, to promote self-efficacy-enhancing experiences. With increasing growth of perceived self-efficacy, the mastery aids were discontinued to verify that the coping attainments stemmed from the exercise of personal agency rather than from mastery aids. Self-directed mastery experiences were then arranged to strengthen and generalize perceived self-efficacy.

As in the baseline session, heart rate was monitored throughout the session. Saliva samples were obtained and perceived coping self-efficacy was measured before, at the midpoint, and at the end of the two-hr session. The blood sample was drawn for immunologic assays at the end of the session.

Maximizing the Strength of Perceived Coping Self-Efficacy

All subjects also participated in an additional 2-hr session, during which they received further guided mastery to raise their perceived coping self-efficacy to maximal strength of 100 for each coping task in the efficacy scale. Their perceived efficacy was measured before, at the midpoint, and at the end of this session. Thus, all subjects received two sessions of structured mastery experiences, which provided a uniform period for measuring the rate of growth of perceived coping efficacy. Although subjects began at baseline with a weak sense of efficacy, they differed in the rate with which they acquired a strong sense of coping efficacy. Subjects who had not achieved the maximal level of perceived coping self-efficacy by the end of the second session received additional guided mastery with periodic efficacy assessments until they judged themselves maximally self-efficacious for each coping task.

Exposure to Stressor With Maximal Perceived Coping Self-Efficacy

After subjects had achieved a maximal sense of coping self-efficacy, they participated in a 2-hr session at the same time on the same weekday of the following week. During this session, subjects performed the different coping activities with the snake but with a maximal sense of perceived coping self-efficacy. Saliva samples were obtained, and subjects' perceived coping self-efficacy was measured at the same three points in the session as in the earlier phases. Their heart rate was monitored throughout, and the blood sample was drawn at the end of the session.

Results

Anticipatory Stress

Subjects experienced moderate anticipatory stress 2 days before and even more elevated stress the day immediately preceding the efficacy-acquisition phase of the experiment. The rise in level of stress over the 2 days was significant for each manifestation of stress and for the aggregated measure, $t(19) = 3.75$,

$p < .001$. These data corroborate a relatively high level of anticipatory stressful self-arousal. Some sleep disturbance was also reported by half the subjects, but it did not worsen over the 2 days.

Perceived Coping Self-Efficacy

The mean strength of perceived coping self-efficacy at each of the three phases of the experiment is presented in Figure 1. A one-way analysis of variance revealed the changes to be highly significant, $F(8, 144) = 259.34$, $p < .0001$. Analyses of variance were also computed for changes in perceived self-efficacy within each of the three phases. Subjects exhibited an extremely weak sense of coping efficacy at the outset of the baseline phase and did not change in this regard at the midpoint and final assessment of the session.

Subjects began the stressful inefficacy phase at the same low-perceived-efficacy level as at the end of the baseline period. However, their perceived self-efficacy progressively increased in strength as they continued to gain experience in coping with the phobic stressor, $F(2, 38) = 88.45$, $p < .0001$. In pairwise comparisons, they judged themselves more efficacious at the midpoint, $t(19) = 8.35$, $p < .001$, and at the end of the phase, $t(19) = 10.96$, $p < .001$, than when they first began the coping session. The rise in perceived self-efficacy strength between the latter two points was also significant, $t(19) = 7.02$, $p < .0001$.

Subjects substantially increased in perceived strength of coping self-efficacy from the end of the self-efficacy-acquisition phase to the beginning of the maximal self-efficacy phase of the experiment, $t(19) = 6.44$, $p < .0001$. As shown in Figure 1, subjects displayed essentially maximal perceived self-efficacy throughout the third phase. However, at the outset of the session, 7 subjects rated their coping self-efficacy slightly less than the maximal level ($M = 97$), which yielded small but significant differences at the $p < .05$ level in comparison with the midpoint and endpoint assessments. The striking increase in perceived coping self-efficacy was reflected in marked changes in activities and thought patterns as recorded by subjects in the postexperiment questionnaire. All subjects (100%) reported complete relief from perturbing intrusive thoughts and nightmares, and all but 1 subject reported complete freedom of action in settings and activities they had phobically avoided. These qualitative data corroborate the results of the formal self-efficacy assessment that the requisite condition of perceived controlling efficacy was, indeed, achieved.

Changes in Level of Heart Rate and Cortisol Activation

In the baseline period, subjects had no exposure to the phobic stressor. Although they had equivalent exposure to the phobic stressor during the high- and low-efficacy phases of the study, note that they performed different sets of coping activities in the latter two phases. During much of the self-efficacy-acquisition phase, many of the subjects were able to deal with only the weakest threats, whereas in the maximal perceived self-efficacy phase, they performed the most menacing activities, such as having the snake crawl on them and bringing it to their face. The effects on heart rate and cortisol activation of more taxing coping activities in the final phase would offset

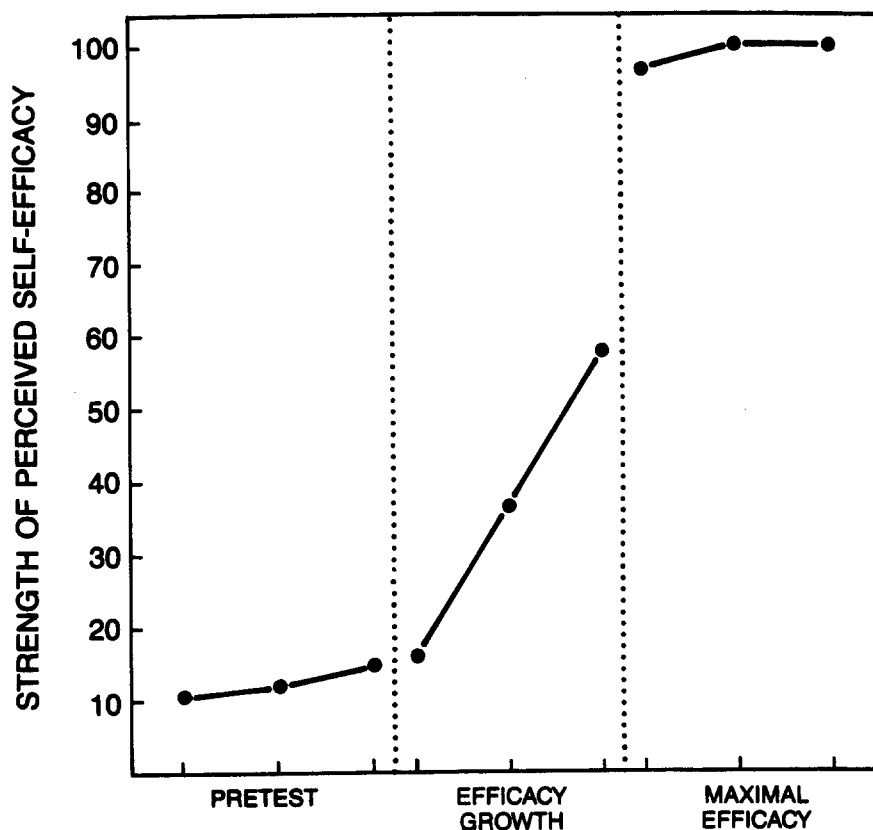


Figure 1. Strength of perceived coping self-efficacy at three points at each of the three phases of the experiment.

differences arising from divergence in perceived coping self-efficacy. In addition, subjects expressed some excitement over demonstrating their spectacular coping triumph in the maximal self-efficacy phase of the experiment.

Subjects' baseline heart rate was 78 bpm. During the efficacy-acquisition phase, their heart rate rose by 11% in relation to the baseline level, $t(18) = 4.30$, $p < .001$, and declined by 3% from the efficacy-acquisition to the maximal self-efficacy phase.¹ The same pattern of results is obtained for mean level of heart rate at the three phases as for relative change in heart rate. Analyses of variance of these data reveals a significant phase effect, $F(2, 36) = 9.75$, $p < .001$. In paired comparisons, the heart rate acceleration in the efficacy-acquisition phase was highly significant, $F(1, 18) = 18.22$, $p < .001$, but the decline in the maximal-efficacy phase was not.

Changes in cortisol activation followed a similar recurrent pattern across phases. Cortisol is highly responsive to novel elements and situational changes. The cortisol level subjects displayed after gaining familiarity with situational and coping demands provided the most informative measure of level of endocrine activity. If subjects continued to display an elevated level of cortisol after they had been thoroughly familiarized with the range of coping tasks introduced in the session, it would reflect a high level of phobic stress. Therefore, subjects' level of cortisol at the end of each session served as the main measure in the analyses. Subjects displayed elevated cortisol

levels at the beginning of each phase but as the session progressed, cortisol levels declined. As subjects gained familiarity with the experimental setting, their initially high cortisol activation ($M = 0.60$) diminished across sessions, $F(2, 36) = 41.76$, $p < .001$. The decrease in average cortisol level of 32% from the baseline value to the efficacy-acquisition phase was significant, $t(19) = 3.86$, $p < .001$, but the 5% decline from the efficacy-acquisition to the maximal efficacy phase was not.

We saw earlier that when phobics performed the same coping tasks under differential strength of perceived self-efficacy, heart rate, blood pressure, and catecholamine secretion were elevated under weak perceived self-efficacy and dropped to a low level under maximal strength of perceived self-efficacy

¹ During the maximal-self-efficacy phase, 1 subject exhibited an extreme heart rate throughout the session that diverged markedly from her heart rate levels in previous sessions and from all other measures signifying absence of stress. This highly anomalous heart rate suggested sustained periods of supraventricular tachycardia. The tremendous fluctuation in her heart rate along with periods of sustained extreme heart rate was consistent with this condition (Rosen & Bauernfiend, 1983). The pattern was too aberrant to reflect sympathetic stimulation nor did it fit a technical malfunction. Although this subject displayed the anomalous heart rate only in the maximal self-efficacy phase, this subject was not included in any analyses involving heart rate.

Table 1
*The Means and Standard Deviations for Each of Several Components
of the Immune System at Each of Three Phases of the Experiment*

Immune component	Experimental phase					
	Baseline		Efficacy growth		Maximal efficacy	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Lymphocytes	1,572	400	1,867	607	1,813	591
Total T cells	1,124	295	1,364	473	1,256	414
Helper T cells	721	196	873	326	819	290
Suppressor T cells	370	143	427	174	408	163
Helper/suppressor	2.22	1.07	2.21	0.81	2.11	0.62
Interleukin-2	41	34	46	85	22	19
HLA-DR	216	93	283	139	263	180

(Bandura et al., 1982; Bandura et al., 1985). The pattern of cortisol secretion in the present study is in accord with the findings of Nesse and his colleagues (Nesse et al., 1985) that cortisol elevation is highest in anticipation of treatment for phobic dysfunction.

Cortisol and heart rate activation were unrelated in the baseline and maximal self-efficacy phases. However, in the self-efficacy-acquisition phase, in which there was significant autonomic activation, heart rate acceleration was significantly associated with elevated cortisol level, $r(17) = .44, p < .05$. Growth of perceived self-efficacy was unrelated to level of cortisol or heart rate. However, the more rapid the acquisition of perceived self-efficacy, the lower was the subjects' heart rate in the maximal efficacy phase, compared with their baseline level, $r(17) = -.51, p < .025$.

Immune Changes as a Function of Perceived Coping Self-Efficacy

The immunological system status at each of the three phases of the experiment is summarized in Table 1. Percentage changes in immunological system components during exposure to the phobic stressor in the acquisition and maximal self-efficacy phases were computed in relation to level of immunocompetence in the baseline phase. Figure 2 presents the pattern of immune change as a function of perceived coping self-efficacy. The effects were analyzed in terms of percentage of change, to control for individual differences in baseline levels. The significance of the differences across phases are provided in Table 2.

For purposes of expository convenience, the terms immunoenhancement and immunosuppression are used simply as descriptors for quantitative increases and decreases, respectively, in the various components of the immune system. The predominant changes were increases in the immunological system status during the phase in which subjects initially confronted the stressor inefficaciously but continued to develop their sense of coping efficacy. The immunoenhancement was significant for all lymphocyte functions. Coping with growing perceived self-efficacy raised the total number of lymphocytes and T lymphocytes and increased helper and suppressor T cells without disrupting the balance between them. HLA-DR also increased

substantially during the efficacy-acquisition phase. Expression of the Interleukin-2 did not change significantly.

During the subsequent phase, when subjects coped with the stressor with a maximal sense of efficacy, immunological system status changed toward the baseline level. However, the change was not complete, in that subjects continued to display significantly higher lymphocyte and helper T cell function, marginally higher suppressor T cell function, and higher HLA-DR than they did at the baseline phase (Table 2).

Divergent Patterns of Immune Changes

Exposure to the stressor during the perceived self-efficacy-acquisition phase produced two markedly divergent patterns of immune responses. For most of the indexes of immunological system status, approximately three quarters of the subjects exhibited immunoenhancement, and the remaining subjects experienced a decline in immunological system status. For both immunoenhancers and immunoattenuators, the level of immunological system components changed toward their baseline level during the phase in which subjects coped with the stressor with maximal perceived self-efficacy (Figure 3).

Predictors of Changes in Immunological System Status

The hypothesized predictor variables were correlated with percentage change from baseline values in each immunological component for each of the two coping phases of the study. The first variable was growth of perceived coping self-efficacy as indexed by the percentage change in self-efficacy strength from the beginning of the guided mastery session to the end of the supplemental session received by all subjects. As previously noted, both cortisol and autonomic activity have been found to exert immunosuppressive effects. Subjects' level of cortisol at the end of the efficacy-acquisition phase was used in the analysis. Mean heart rate during the efficacy-acquisition phase was the third variable in the analysis.

Table 3 contains the pattern of zero-order correlations with changes in immunological indexes. In accord with prediction, the findings generally showed that slow growth of perceived

Table 2
Significance of the Changes in Immune Status From the Baseline Level to Exposure to the Stressor During Acquisition and Maximal Perceived Coping Self-Efficacy

Measure	Baseline vs. efficacy acquisition		Baseline vs. maximal efficacy		Efficacy acquisition vs. maximal efficacy
	<i>t</i>	<i>p</i>	<i>t</i>	<i>p</i>	<i>t</i>
Lymphocytes	2.67	.02	2.42	.05	0.78
Total T cells	2.80	.02	1.87	.08	1.48
Helper T cells	2.87	.01	2.17	.05	1.16
Suppressor T cells	2.16	.05	1.73	.10	0.33
Helper/suppressor T cells	0.81		0.27		0.52
Interleukin-2	0.90		1.10		1.61
HLA-DR	2.80	.02	2.02	.06	0.31

self-efficacy, high cortisol activation, and heart rate acceleration were associated with lower immunological status during the efficacy-acquisition phase. The immunological change from the efficacy-acquisition phase to the maximal efficacy phase represents mainly a decline toward the baseline level.

Rate of growth of perceived self-efficacy emerged again as a consistent predictor of immunological change during the perceived maximal-efficacy phase. The slower the subjects' growth of perceived efficacy, the more their immunological status declined toward their baseline level. High cortisol activation was related to lower lymphocyte count and helper:suppressor ratio. Heart rate predicted only lymphocyte count during this phase.

Regression Analysis

Hierarchical regression analyses were performed to test the multivariate relation of the three stress-related variables to changes in immunological system components. Growth of perceived self-efficacy was considered the first factor because of its demonstrated paramount role in stress reactions (Bandura, 1988b). Cortisol, which can produce immunosuppressive effects at high levels, was entered as the second factor, followed by Heart Rate. The results of the regression analysis are summarized in Table 4.

During the efficacy-acquisition phase, the rises in lymphocytes and helper and suppressor T cells and HLA-DR were related to rapid growth of perceived coping self-efficacy, to low cortisol levels, and to a lesser extent to a low level of heart rate. Rate of growth of perceived coping self-efficacy also emerged as a strong predictor of the degree of decline in these immune components toward the baseline level in the maximal self-efficacy phase. Subjects who had rapidly acquired a strong sense of efficacy were less likely to exhibit declines in immune status. Cortisol level accounted marginally for declines in lymphocytes and in the ratio of helper to suppressor cells. Level of heart rate did not attenuate immunological status in the maximal-efficacy phase when the influence of the other variables was controlled.

Discussion

The findings of the present experiment are generally supportive of the hypothesis that perceived self-efficacy to exercise control over stressors is a modulator of immunological system status. The powerful guided mastery procedure made it possible to create differential levels of perceived coping self-efficacy under high experimental control and to examine the immunological effects of these intrasubject changes in perceived coping self-efficacy. The especially noteworthy finding is that stress activated in the process of gaining coping mastery is immunoenhancing rather than immunosuppressing. This is reflected in a higher level of lymphocyte and T cell function. Acquisition of perceived self-efficacy to control stressors produced more than simply transient changes in immunity. The increase in immunological competence was generally sustained over time as evi-

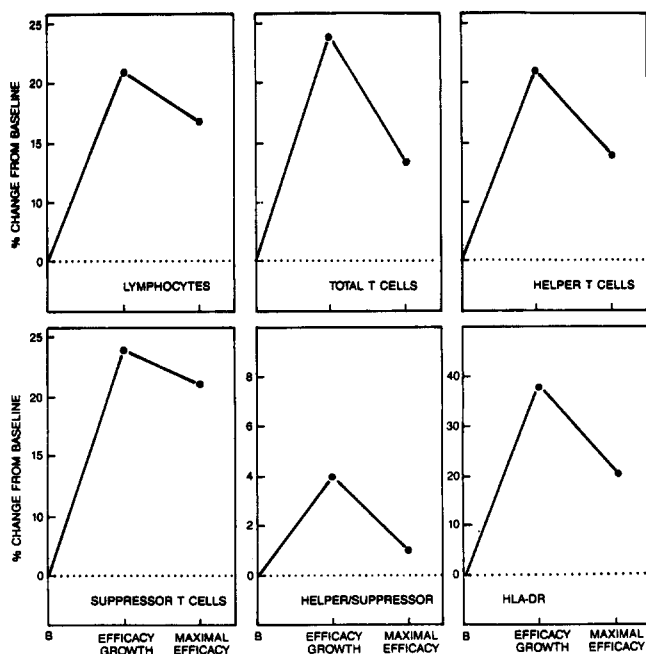


Figure 2. Changes in components of the immune system experienced as percentage of baseline values during exposure to the phobic stressor while acquiring perceived coping self-efficacy (*efficacy growth*) and after perceived coping self-efficacy develops to the maximal level (*maximal efficacy*).

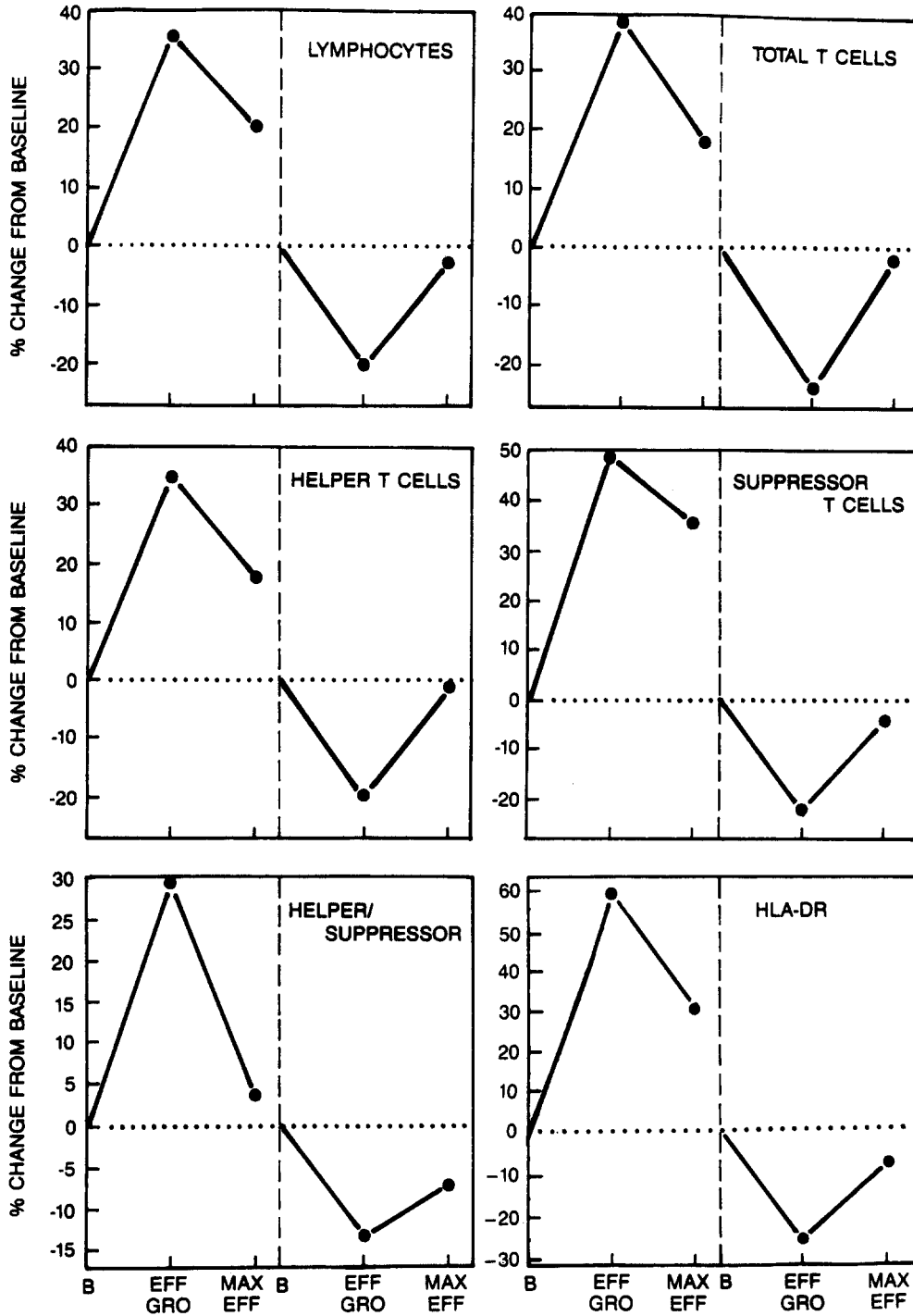


Figure 3. Divergent patterns of changes in immune states exhibited by two subgroups of subjects during acquisition of perceived self-efficacy to control stressors (Eff Gro) and after subjects developed a maximal sense of coping efficacy (Max Eff). B = baseline. The panels on the left portray the enhanced immune status displayed by about three quarters of subjects; the panels on the right portray the immunosuppressant reactions of the remaining subgroups of subjects.

dent in the significantly higher system status in the maximal perceived-self-efficacy phase than in the baseline phase.

Because HLA-DR is primarily expressed on B cells, the results of this study suggest that B cells, in addition to T cells, are

responsive to the stress of low perceived coping efficacy. That is, enhanced perceived self-efficacy affected the arm of the immune system governing humoral immunity as well as the cellular arm of the immune system. Interleukin-2 is a measure of T

Table 3
Correlations Between Stress-Related Variables and Percentage Change in Immune Status

Measure	Lymphocytes		Total T cells		Helper T cells		Suppressor T cells		Helper/suppressor		Interleukin-2		HLA-DR	
	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>
Change from baseline to efficacy-acquisition phase														
Growth of efficacy	.46	<.03	.41	<.04	.39	<.05	.38	<.05	-.24	<i>ns</i>	.09	<i>ns</i>	-.51	<.02
Cortisol	-.43	<.03	-.38	<.06	-.40	<.05	-.43	<.03	.32	<.09	.29	<i>ns</i>	.43	<.06
Heart rate	-.40	<.05	-.35	<.07	-.28	<i>ns</i>	-.43	<.03	.37	<.06	-.26	<i>ns</i>	.36	<i>ns</i>
Change from efficacy-acquisition to maximal efficacy phase														
Growth of efficacy	-.42	<.04	-.48	<.02	-.44	<.03	-.50	<.02	.21	<i>ns</i>	-.46	<.03	-.30	<i>ns</i>
Cortisol	.40	<.04	.24	<i>ns</i>	.02	<i>ns</i>	.30	<i>ns</i>	-.40	<.04	.06	<i>ns</i>	.03	<i>ns</i>
Heart rate	.42	<.04	.24	<i>ns</i>	.06	<i>ns</i>	.33	<.08	-.36	<.06	.16	<i>ns</i>	.19	<i>ns</i>

Note. For growth of efficacy and cortisol, $n = 20$; for heart rate, $n = 19$.

cell activation; exposure to any pathogen activates T cells. The general lack of significant findings for Interleukin-2 can be best understood as consistent with the expectation that not all subjects would have been exposed to a pathogen during the course of this study. While the numbers of T cell subsets changed during the study, this is distinct from the activation of T cells, which is pathogen related, and would not be expected to occur in any systematic way.

Previous research has shown that the effects of stress on the immune system vary depending on the timing, intensity, and chronicity of stressors (Coe & Levine, in press; Keller, Weiss, Schleifer, Miller, & Stein, 1981). This study indicates that growth of perceived controlling efficacy over stressors is also an influential factor governing the direction and magnitude of stress effects on immunological status. Much human stress is

generated in coping transactions while competencies are being developed and expanded. Because the nature of the challenges to competence changes across the lifespan, the process of coping with stressors and new mastery demands is a continuing one. There are substantial evolutionary benefits to experiencing enhanced immunocompetence during development of coping capabilities vital for effective adaptation. The field of health psychology has focused heavily on the physiologically debilitating effects of stressors. The findings of this study are in accord with evidence from different lines of research presented by Dienstbier (1989) on the physiologically toughening effects of mastery over stressors.

Explaining variance in immunological status within experimental phases constitutes a more stringent test of hypothesized mechanisms than does accounting for the variance across

Table 4
Regression Analysis of Stress-Related Predictors of Change in Immune Status

Immune function	Growth of efficacy				Cortisol				Heart rate			
	<i>r</i>	R^2 inc	<i>F</i>	<i>p</i>	<i>r</i>	R^2 inc	<i>F</i>	<i>p</i>	<i>r</i>	R^2 inc	<i>F</i>	<i>p</i>
Change from baseline to efficacy-acquisition phase												
Lymphocytes	.46	.21	4.70	.04	-.41	.17	4.70	.04	-.36	.13	4.04	.06
Total T cells	.41	.17	3.64	.07	-.37	.13	3.24	.09	-.32	.10	2.59	<i>ns</i>
Helper T cells	.39	.15	3.16	.09	-.38	.15	3.57	.08	-.25	.06	1.48	<i>ns</i>
Suppressor T cells	.38	.15	3.09	.10	-.42	.18	4.45	.05	-.39	.15	4.62	.05
Helper/suppressor T cells	-.24	.06	1.12	<i>ns</i>	.31	.10	1.96	<i>ns</i>	.34	.11	2.46	<i>ns</i>
Interleukin-2	.09	.008	0.14	<i>ns</i>	.29	.09	1.61	<i>ns</i>	.48	.23	5.42	.03
HLA-DR	.51	.26	6.40	.02	.42	.17	5.19	.04	.32	.10	3.32	.09
Change from efficacy-acquisition to maximal efficacy phase												
Lymphocytes	-.42	.17	3.76	.07	.39	.15	3.88	.06	.39	.15	4.28	.06
Total T cells	-.48	.23	5.24	.03	.23	.05	1.23	<i>ns</i>	.22	.05	1.04	<i>ns</i>
Helper T cells	-.44	.20	4.36	.05	.004	.00	0.0003	<i>ns</i>	.06	.003	0.06	<i>ns</i>
Suppressor T cells	-.50	.24	5.76	.03	.28	.08	1.98	<i>ns</i>	.31	.09	2.37	<i>ns</i>
Helper/suppressor T cells	.21	.05	0.84	<i>ns</i>	-.40	.16	3.38	.08	-.32	.11	2.25	<i>ns</i>
Interleukin-2	-.45	.21	4.36	.04	.03	.001	0.00	<i>ns</i>	.13	.02	0.32	<i>ns</i>
HLA-DR	-.30	.09	1.82	<i>ns</i>	.03	.0007	0.13	<i>ns</i>	.19	.035	0.59	<i>ns</i>

Note. For growth of efficacy and cortisol, $n = 20$; for heart rate, $n = 19$.

phases. Subjects varied considerably in the rate with which they acquired self-percepts of coping efficacy. Perceived coping inefficacy during the efficacy-acquisition phase attenuated immunological status. Perceived coping inefficacy also predicted the magnitude and direction of change in immunological status during the subsequent maximal self-efficacy phase. The slower the growth of perceived self-efficacy, the greater was the recovery from immunoattenuation after maximal perceived self-efficacy had been achieved. Rapid acquisition of perceived coping self-efficacy was thus accompanied not only by enhanced immune response, but by retention of increased level of immunological functioning during the maximal perceived-self-efficacy phase. Evidence that changes in immunological system components covary with variations in growth of perceived self-efficacy lends further credence to the contributory role of perceived controlling efficacy.

The findings also provide some support for cortisol regulation of immune function independent of the effects of perceived coping self-efficacy. The higher the endocrine activation, the more likely were subjects to experience attenuation in immune function as they were striving to gain coping mastery. Level of cortisol activation in the acquisition phase also predicted changes in lymphocyte function in the maximal self-efficacy phase, but cortisol level in the maximal phase did not. After subjects had become fully efficacious, their cortisol levels were low and varied little in this regard. Limited variance attenuates correlations. Although heart rate during efficacy acquisition correlated with changes in lymphocyte numbers in both the acquisition and maximal self-efficacy phases, it did not account for additional variance when the effects of perceived self-efficacy and cortisol activation were controlled. These findings concerning autonomic system involvement should be interpreted with caution because only one index of autonomic arousal was used. However, previous findings have shown that the pattern of changes in other autonomic indexes (i.e., systolic and diastolic blood pressure) accompanying self-efficacy enhancement are similar to those of heart rate (Bandura et al., 1982).

Evidence that rapid acquisition of perceived coping self-efficacy predicted enhanced immunocompetence after a strong sense of efficacy had been restored raises an interesting issue that warrants comment. These findings indicate that vigorous mastery of chronic stressors not only instills a strong sense of self-efficacy but leaves lasting changes that can serve as protective factors against adverse immunological effects of psychological stressors. High perceived self-efficacy gained commandingly may convey a more generalized sense of coping capability than if it is gained laboriously with prolonged stress. H. S. Hoffman (1969) showed that uncontrollable physical stressors can create vulnerabilities that leave some sensitivity to aversive events even after acquired fears have been eliminated through repeated nonreinforced exposure. Mastery modeling, of course, does much more in that it equips people with coping strategies and a resilient sense of efficacy to exercise control over potential threats. Results of the present study suggest that rapid development of perceived self-efficacy to exercise control over psychological stressors can instill a durable protection against aversive events. These findings add to other lines of evidence showing that development of stress-reduction capabili-

ties can decrease immunological vulnerability to recurrent stressors (Kiecolt-Glaser et al., 1986, 1985).

The results of this study can be understood in terms of two biochemical mediators of psychosocial influences on immunity: catecholamines, which are released with activation of the sympathetic-adrenal medullary system, and cortisol, the hormone associated with the pituitary-adrenal cortical system. In psychological terms, these two systems have been described as the *effort* and *distress* systems, respectively (Frankenhaeser, 1983) or as the *fight-flight* and *conservation-withdrawal* systems (Henry & Stephens, 1977). Although the sympathetic-adrenal cortical system is engaged primarily during fear and anger, activation of the pituitary-adrenal cortical system is thought to reflect perception that the threat is overwhelming. Activation of this latter system tends to be associated with more prolonged stressors, presumably because the organism is more likely to relinquish coping efforts in the face of unremitting threat.

Cortisol appears to be associated with suppression of immune function in both enumeration and functional measures (Cupps & Fauci, 1982), whereas catecholamines have mixed effects. Injections of epinephrine have been shown to increase numbers of blood lymphocytes while reducing their functional efficacy (Crary, Borysenko, et al., 1983; Crary, Hauser, et al., 1983; Eriksson & Hedfors, 1977).

The major finding of this study—that lymphocyte numbers increase during efforts to master the phobic threat—suggests that catecholamine release may have been the predominant determinant of the immunological changes. Indeed, studies of the microrelation between strength of perceived self-efficacy and plasma catecholamine secretion have shown that catecholamines are released while phobics cope with threatening tasks for which they doubt their coping capabilities (Bandura et al., 1985).² The fact that subjects who displayed a decrease in lymphocytes evidenced slow acquisition of perceived efficacy and prolonged elevations in salivary cortisol is consistent with the research indicating immunosuppressant effects of cortisol.

The results concerning the relation of growth of perceived self-efficacy to immune changes may be of general relevance for the impact of perceived coping efficacy on these two important stress-physiological systems during active coping. That is, rapid acquisition of perceived coping efficacy may be associated with activation of the sympathetic nervous system—*effort* in Frankenhaeser's terms—and slow efficacy acquisition with adrenal-cortical activation, or *distress*. Further investigation of this issue might add to our understanding of the biochemical effects of perceived coping efficacy.

The immune system includes multiple interacting subprocesses with intricate interconnections to other biological systems, all of which complicate evaluation of level of immunity. The clinical significance of the alterations in immune function accompanying changes in perceived coping self-efficacy re-

² According to this interpretation, it might be argued that heart rate, being a measure of autonomic arousal, should have been correlated positively with increased lymphocyte numbers. In fact, heart rate, which is determined by both sympathetic and parasympathetic stimulation, has not been found consistently to be correlated with serum catecholamines (Conrada et al., 1982; Esler, Hasking, Willett, Leonard, & Jennings, 1985).

mains to be determined. Nevertheless, knowledge of how efficacious control over stressors affects different aspects of the immune system is important to an eventual full understanding of the dual linkage between psychosocial influences, immune function, and disease processes.

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