Perceived Self-Efficacy in Coping With Cognitive Stressors and Opioid Activation

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This experiment tested the hypothesis that perceived self-inefficacy in exercising control over cognitive stressors activates endogenous opioid systems. Subjects performed mathematical operations under conditions in which they could exercise full control over the cognitive task demands or in which the cognitive demands strained or exceeded their cognitive capabilities. Subjects with induced high perceived self-efficacy exhibited little stress, whereas those with induced low perceived self-efficacy experienced a high level of stress and autonomic arousal. Subjects were then administered either an inert saline solution or naloxone, an opiate antagonist that blocks the analgesic effects of endogenous opiates, whereupon their level of pain tolerance was measured. The self-efficacious nonstressed subjects gave no evidence of opioid activation. The self-inefficacious stressed subjects were able to withstand increasing amounts of pain stimulation under saline conditions. However, when endogenous opioid mechanisms that control pain were blocked by naloxone, the subjects were unable to bear much pain stimulation. This pattern of changes suggests that the stress-induced analgesia found under the saline condition was mediated by endogenous opioid mechanisms and counteracted by the opiate antagonist.

There is a growing body of evidence that the ability to exercise control over potential stressors is a critical factor in the activation of different neurophysiological systems. Exposure to stressors without controlling efficacy activates neurotransmitters, stress-related hormones, and impairs various cellular components of the immune function (Bandura, Taylor, Williams, Mefford, & Barchas, 1985; Coe & Levine, in press; Maier, Laudenslager, & Ryan, 1985). Exposure to the same stressful events with controlling efficacy has few neurochemical effects. These findings are 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 species and stressors. Research into the neurochemical effects of inefficacious control therefore needs to be broadened and extended to events and psychological processes that have high ecological relevance to human coping. 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 ones that relate to the strain of task demands and workloads. It is the perception of demands as exceeding capabilities that becomes the stressful reality. Efforts to determine the neurochemical effects of inefficacious control in humans have relied extensively on correlational or quasi-experimental studies in which occurrences of life stressors are related to indexes of neurophysiological functioning. Although these lines of research have clarified some aspects of inefficacious control, experimental studies are needed to verify the direction of causality.

It is generally acknowledged that inefficacious control produces neurochemical changes by creating a state of stress. Psychological stress is the result of a relational condition in which perceived environmental demands strain or exceed perceived coping capabilities in domains of personal import. People's judgments of their controlling efficacy figure prominently in their relational appraisal of demands to capabilities. Perceived self-efficacy is concerned with beliefs in one's capabilities to mobilize the motivation, cognitive resources, and courses of action needed to meet given situational demands.

Findings of different lines of research underscore the influential role of perceived control in stress reactions (Averill, 1973; Lazarus & Folkman, 1984; Miller, 1980). A sense of controllability can be achieved either behaviorally or cognitively. In behavioral control, individuals take action that forestalls or attenuates environmental stressors. In cognitive control, people operate under the belief that they can manage such stressors should they arise. Although these two forms of controllability are distinguishable operationally, human coping under life circumstances rarely involves controlling action devoid of any perceptions of personal control, or perceptions of personal control devoid of any actions. Self-efficacious thought and actions are usually products of reciprocal causation (Bandura, 1986).
Being able to exercise control over potential stressors can diminish stress because the capability is used to reduce or to prevent aversive experiences. But there is much more to the process of stress reduction by behavioral control than simply the momentary curtailing of aversive events. Behavioral control instills and strengthens beliefs concerning one's coping efficacy. These cognitive changes serve as proximal determinants of anticipatory stress reactions and level of stress during encounters with stressors (Bandura, 1988). Thus, in some studies of controllability, merely the exercise of personal control over the occurrence of aversive events without curtailing their intensity reduces stress reactions (Gunnar-vonGnechten, 1978). Repeated failures create stress reactions when ascribed to personal incapability, but the same failure experiences leave people unperturbed if ascribed to situational factors (Wortman, Panciera, Shusterman, & Hibscher, 1976). In situations in which the opportunity to wield control exists but is unexercised, it is the self-knowledge that one can exercise control should one choose to do so rather than its application that reduces stress reactions (Glass, Reim, & Singer, 1971). These types of findings indicate that much of the stress reductive effects of behavioral control result anticipatorily from perceived capability to wield control over troublesome events rather than simply from attenuating them.

Perceived control without the actuality has been shown to reduce stress reactions. People who are led to believe they can exercise some control over aversive events display lower autonomic arousal and less impairment in performance than do those who believe they lack personal control, even though they are equally subjected to the painful stimulus (Geer, Davison, & Gatchel, 1970; Glass, Singer, Leonard, Krantz, & Cummings, 1973). The foregoing studies have relied on plausible presumptive mediation inferred from the manipulations rather than on direct assessment of perceived self-efficacy and its linkage to level of stress reactions.

As already alluded to, in social cognitive theory perceived self-efficacy operates as a cognitive mechanism through which controllability affects stress reactions (Bandura, 1986). If people believe they can deal effectively with potential stressors, they are not perturbed by them. But if they believe they cannot control aversive circumstances, they have much cause for distress. They tend to dwell on their coping deficiencies and see the environment as fraught with threats. In so doing, they distress themselves and constrain and impair their level of functioning (Beck, Emery, & Greenberg, 1985; Lazarus & Folkman, 1984; Meichenbaum, 1977; Sarason, 1975). Perceived self-inefficacy to control perturbing cognitions further augments stress reactions (Kent & Gibbons, 1987).

The microrelation of perceived inefficacy to stress reactions has been examined most extensively in coping with phobic stressors (Bandura, O'Leary, Taylor, Gauthier, & Gossard, 1987; Bandura, Reese, & Adams, 1982). Phobics display little stress on tasks for which they judge themselves fully efficacious. As they cope with tasks for which they distrust their coping efficacy, however, their subjective distress mounts, their heart rate accelerates, their blood pressure rises, and they display elevated catecholamine secretion. After their perceived coping efficacy is fully strengthened, they manage the same stressors with little stress or physiological activation. Thus, the combined results from different manifestations of stress are consistent in showing that stress reactions to coping tasks differ when perceived self-efficacy differs, but reactions to the identical tasks are the same when perceived self-efficacy is raised to the same maximal level.

Studies with animals subjected to painful stimulation show that stress can activate endogenous opioids that block pain transmission (Kelley, 1986). Opioid involvement is indicated by evidence that stress-induced analgesia is blocked by opioid antagonists, such as naloxone. It is not the physically painful stimulation per se but the psychological stress over its uncontrollability that seems to be a key factor in opioid activation (Maier, 1986). Animals who can turn off shock stimulation show no opioid activation, whereas yoked animals who experience the same shock stimulation without being able to control its offset give evidence of stress-activated opioids. Of the different functions of endogenous opioids, their pain-relieving effects have received greatest attention. However, endogenous opioids have a broader adaptive function. By blunting the aversive impact of stressors, they enable individuals to deal more effectively with distressing environmental events.

The purpose of the present study was to determine whether perceived inefficacy in exercising control over cognitive stressors activates endogenous opioid systems. Subjects performed mathematical operations under conditions in which they could exercise full control over the cognitive task demands or in which the cognitive demands strained or exceeded their cognitive capabilities. Changes in subjects' perceived mathematical self-efficacy, their level of autonomic arousal during the cognitive stressor task, and their subjective distress, mental strain, and perceived performance impairment were measured. Following induction of high and low levels of perceived mathematical self-efficacy, subjects at each level of self-efficacy were administered either an inert saline solution or an opiate antagonist, naloxone. Their level of pain tolerance was then measured at periodic intervals.

Perceived controlling inefficacy was expected to be accompanied by high stress reactions. We hypothesized that self-inefficacious stressed subjects would be able to withstand increased amounts of pain stimulation because of the analgesic effects of opioid activation. However, they would be unable to bear much pain stimulation when the endogenous opioid mechanisms that control pain were blocked by naloxone. In contrast, we predicted that the self-efficacious nonstressed subjects would display no significant changes in pain tolerance under either saline or naloxone conditions.

Method

Subjects

Twelve male and 28 female paid volunteers from a college population participated in the study. They were randomly assigned to conditions, balanced for sex, with 10 subjects in each of four conditions. Subjects were selected whose pain tolerance on a cold-pressor test fell within the tolerance range, above 20 s at the lower limit and below 180 s at the upper limit. These screening criteria were used to avoid ceiling and basement effects. The mean pretest pain tolerance was 69 s. Subjects
judging their perceived efficacy to tolerate pain, subjects were presented each test of pain tolerance. Obtained by dividing the summed magnitude scores by the total number measures of strength of perceived self-efficacy to cope with pain were scale, ranging in 10-unit intervals from high uncertainty, through intermediate levels of certainty, to complete certitude. The measure of strength of perceived self-efficacy to cope with pain were obtained by dividing the summed magnitude scores by the total number of items. The self-efficacy scales were administered before and after each test of pain tolerance.

Pain Tolerance Test

Pain was induced with the cold-pressor procedure. Two insulated containers were used in the tests of pain tolerance. One container filled with water kept at 20 °C was used prior to each cold-pressor test. The other container was divided into two compartments by a wire screen, with ice in one side and ice-free water in the other. The water was circulated by a submerged pump and maintained at a constant temperature of 0 °C.

Subjects were instructed to place their dominantly preferred hand in the 20 °C water for 2 min to equalize initial hand temperatures. They were then asked to immerse their hand in the ice water up to the wrist for as long as they could. The pain tolerance score was the number of seconds subjects were able to keep their hands in the ice water. In the drug phase of the study, the maximum time allowed for each cold-pressor test was 5 min. All the cold-pressor pain tests were administered by a female experimenter.

Efficacy Induction Procedure

In the next step of the experiment, subjects performed the mathematical problem-solving task under conditions designed to create high and low perceived controlling efficacy. All the instructions and the cognitive task demands were presented on a computer monitor following a programmed procedure. To eliminate any possible social bias, the female experimenter initiated the appropriate computer program for each subject by entering into the subjects’ keyboard a preassigned code, and then promptly left the room. The code determined which efficacy induction condition subjects would receive and to whom they would be yoked. For subjects in the low controlling efficacy condition, the program presented the same number of mathematical problems as had been attempted by their counterparts in the high controlling efficacy condition to whom they had been randomly yoked. Thus, each pair of yoked subjects was presented with the same number of mathematical problems in the identical order, and the conditions differed only in the degree to which subjects could exercise control over the cognitive task demands. The codes were predetermined by a coder who was not associated with the conduct of the experiment. This coding procedure ensured that the experimenter had no knowledge of the conditions to which subjects had been assigned, as the computer automatically branched to the appropriate stimulus presentation on the basis of the coded information.

The mathematical problems required subjects to perform sequentially a series of cognitive operations on three integers to arrive at a solution (e.g., 73 - 15 x 3). The task was described as a test of basic cognitive-processing capabilities. Subjects were presented with the mathematical problems continuously over a period of 18 min. They were told that their performance would be assessed as a function of both speed and accuracy. To enhance personal involvement in the task, they were further informed that, at the end of the session, they would compare their cognitive processing attainments with those of others who had performed the computational activity.

Subjects in the high self-efficacy condition could exercise full control over the cognitive task demands because they regulated the pace of the task. Problems appeared on the monitor one at a time and remained there until subjects pressed the key for the next mathematical problem. The yoked subjects in the low self-efficacy condition were presented the same problems in the same order as their high efficacy counterparts, but at a pace that exceeded their cognitive capabilities. The pace was set on the basis of pretest evidence of the minimal time required to perform the necessary cognitive operations. To equate for total length of the problem-solving task, several time gaps were programmed between the rapidly paced subset of problems.

Perceived Mathematical Self-Efficacy

The instructional part of the computer program instructed subjects to complete the mathematical self-efficacy scale before and after the mathematical problem-solving session. The self-efficacy scale included 10 levels of performance attainments that ranged from solving 10% to 100% of the problems. Subjects rated the strength of their self-judged efficacy that they could achieve each of the levels of mathematical attainment by using a 100-point scale ranging in 10-unit intervals from high uncertainty, through intermediate levels of certainty, to complete certitude. The measure of perceived mathematical self-efficacy was obtained by dividing the summed magnitude scores by the total number of items.

The treatment conditions were highly effective in instating high and low levels of perceived self-efficacy. In only two instances did subjects’ perceived self-efficacy diverge from the induction conditions. One subject in the low controllability condition registered high perceived self-efficacy (83 strength), and one in the high controllability condition registered very low perceived self-efficacy (39 strength), despite having solved 92% of the mathematical problems. As this study is concerned with the opioid activation effects of perceived self-efficacy, the latter subjects were assigned to the high and low perceived self-efficacy groups, respectively.

The mean strength of perceived mathematical self-efficacy was 23 for subjects in the low efficacy group, and 87 in the high efficacy group. This marked difference in perceived self-efficacy is highly significant, t(38) = 14.23, p < .0001.
Naloxone Intervention

Because of its capacity to counteract the analgesic effect of opiates by blocking opiate receptors, naloxone is widely used to detect endogenous opioid activity. To test for opioid activation, half the subjects in each perceived self-efficacy group received an intravenous injection of 10 mg of naloxone, an opiate antagonist. Previous studies have shown that this high dosage of naloxone produces complete opioid blockade (Levine & Gordon, 1984). The other half of the subjects were given an injection of saline solution.

The subjects were informed at the outset of the experiment that they would receive an injection of either an inactive medication or a substance that may affect the physical mechanism controlling pain but that the individual effects on the experience of pain were not yet fully known. A nurse administered the injections under a double-blind procedure. Neither the nurse nor the tester knew whether the subjects received naloxone or saline.

Postinjection Tests

The experimenter administered pain tolerance tests at 5, 15, and 30 min after the injection. Levine and his associates (Levine, Gordon, Jones, & Fields, 1978) have found that naloxone's antagonistic effects do not become evident until after 5 min. This temporal effect is corroborated by previous research on cold-pressor pain (Bandura et al., 1987).

Postexperiment Questionnaire

At the conclusion of the experiment, subjects rated the level of stress and time pressure they experienced during the mathematical task, and the percent of their errors they judged were due to the pressure of the cognitive demands. These ratings were made on 10-point scales. At the end of the session, subjects were given a full explanation of the nature and purpose of the study.

Results

Analyses of variance (ANOVAS) performed on the pretest scores revealed that the groups did not differ initially on any of the measures of perceived self-efficacy, pain tolerance, or level of heart rate. Nor were there any significant sex differences in percentage change as a function of treatment on any of the measures.

Changes in Perceived Mathematical Self-Efficacy

At the outset, subjects registered a moderately strong sense of mathematical self-efficacy (67 strength). However, after coping with the task demands, subjects in the low self-efficacy group experienced a marked decline in perceived self-efficacy, t(19) = 9.91, p < .0001, whereas those in the high self-efficacy group heightened their sense of mathematical self-efficacy, t(19) = 3.67, p < .001. These differential changes in self-judged efficacy, which are plotted in Figure 1, are highly significant, t(38) = 8.49, p < .0001.

Level of Subjective Stress

Figure 2 summarizes the mean levels in subjective stress and stress-induced performance impairment reported by subjects in the high and low perceived self-efficacy groups.

Compared with the self-efficacious subjects, the self-ineffi-
revealed a significant interaction, $F(10, 360) = 1.92, p < .04$.
The perceived self-in efficacious subjects exhibited a higher
heart rate than their perceived self-efficacious counterparts during
the first 2-min interval, $t(36) = 1.87, p < .04$, and the third
2-min period, $t(36) = 1.51, p < .07$. Although the perceived self-
inefficacious subjects also continued to exhibit a higher heart
rate at all the subsequent time points, the differences fell short
of statistical significance.

The differences in heart rate between the two groups again
became highly pronounced at the end of the task when subjects
were rating their perceived mathematical self-efficacy. Perceived
self-in efficacious subjects showed a heightened heart rate,
whereas the perceived self-efficacious ones displayed a marked
decline in heart rate. These opposite directional changes are
highly significant, $t(36) = 1.97, p < .03$.

Heart rate was significantly related to perceived mathematical
self-efficacy. For the perceived self-in efficacious subjects, the
weaker their perceived self-efficacy following the computational
task, the more elevated was their heart rate. This was true for
the first 2 min of the computational task, $r(18) = .47, p < .02$;
the first third of the task when the elevation was most evident,
$r(18) = .47, p < .02$; and for the entire duration of the computa-
tional activity, $r(18) = .45, p < .03$. The greater the decline in
perceived self-efficacy, the more elevated was their heart rate
during the first 2 min, $r(18) = .33, p < .08$, and the first third
of the computational task, $r(18) = .30, p < .10$.

The perceived self-efficacious subjects exhibited a uniformly
high sense of efficacy following the computational task. Even
within the limited variance at this upper level of efficacy, the
weaker the perceived self-efficacy, the higher the heart rate dur-
ing the first third of the task, $r(16) = .38, p < .06$. The more
subjects increased their perceived self-efficacy, the greater was
the decline in their heart rate at 2 min, $r(16) = -.35, p < .08$,
and the first third of the task, $r(16) = -.37, p < .07$. Moreover,
the stronger the subjects' perceived self-efficacy, the greater the
reduction they experienced in heart rate when they judged their
self-efficacy after the computational task had been completed,
$r(16) = .52, p < .02$.

**Test for Opioid Activation**

The changes exhibited under different drug conditions by
perceived self-efficacious and self-in efficacious subjects were
evaluated in terms of percentage change in pain tolerance from
the preinjection baseline level. This measure was used because
it controls for individual differences in initial ability to with-
stand pain and has been shown to be more sensitive to treatment
influences than are simple difference scores (Hilgard et al.,
1974).

Variances in pain tolerance were significantly much larger
under saline than under naloxone conditions at each of the tests
of pain tolerance. Several extreme responders produced highly
skewed distributions. Hence, nonparametric tests were used to
test for naloxone antagonistic effects. Figure 4 presents the me-
dian changes in pain tolerance at the three postinjection tests
as a function of perceived self-efficacy and drug administration.
Opioid activity is indicated if subjects who had been given nal-
oxone are less able to endure pain stimulation than those given
saline.

**High perceived self-efficacy.** To evaluate intragroup changes,
Friedman two-way ANOVAs were performed separately on the scores for the saline and naloxone subgroups across the four tests of pain tolerance. For the highly self-efficacious subjects, neither the fluctuations in pain tolerance under naloxone, \( \chi^2(3) = 3.00 \), nor the rise under saline, \( \chi^2(3) = 4.11 \), was significant.

Evaluation of intergroup differences by the Mann–Whitney test showed that highly self-efficacious subjects administered naloxone did not differ significantly in pain tolerance from those given saline at any of the three postinjection tests. Thus, subjects who judged themselves highly efficacious in coping with cognitive task demands showed no evidence of opioid activation.

Low perceived self-efficacy. Perceived self-inefficacious subjects, who were stressed by their inability to fulfill cognitive demands, displayed a uniformly low level of pain tolerance under naloxone (Figure 4). In marked contrast, self-inefficacious subjects administered saline achieved a substantial rise in pain tolerance, \( \chi^2(3) = 9.60, p < .03 \). Pairwise comparisons were performed with the Wilcoxon test between the levels of pain tolerance at the four data points to identify the significant changes. Compared with their baseline level, self-inefficacious saline subjects were better able to tolerate painful stimulation at both the 15-min test, \( z = 2.50, p < .02 \), and the 30-min test, \( z = 2.09, p < .04 \). They were also able to tolerate much more pain stimulation both at the 15-min test, \( z = 2.09, p < .04 \), and the 30-min test, \( z = 1.99, p < .05 \), than at the 5-min point.

The divergent tolerances of painful stimulation by self-infficacious subjects under drug and saline conditions are significant at the points at which the opioid antagonistic effects of naloxone should become most evident. The groups did not differ at the 5-min test. However, perceived self-inefficacious subjects administered naloxone were much less tolerant of pain than their saline counterparts at the 15-min test, \( U = 25, p < .03 \), and at the 30-min test, \( U = 24, p < .03 \).

Heightening pain sensitivity by opioid blockage would attenuate pain tolerance under all conditions and, indeed, the two naloxone groups did not differ in this respect. Self-inefficacious
saline subjects increasingly surpassed their self-efficacious saline counterparts on successive cold-pressor tests. However, several subjects in the latter group displayed sizable increases in pain tolerance so that the intergroup differences fell short of significance. The predictors of pain tolerance under different conditions, which are considered next, reveal interesting relations that help to explain these increases.

Pain Control Self-Efficacy and Pain Tolerance

Pain endurance self-efficacy. Subjects' perceived self-efficacy to withstand pain measured before their baseline assessment predicted their level of pain tolerance in both the subsequent baseline test, r(38) = .47, p < .001, and the postinjection tests. As the correlations at the different postinjection tests were of comparable magnitude, they were averaged by means of an r to z transformation. The stronger the subjects' perceived self-efficacy to withstand pain, the longer they tolerated the painful stimulation in the succeeding cold-pressor tests. However, perceived self-efficacy was more strongly related to pain tolerance under saline conditions, r(18) = .65, p < .001, than under naloxone antagonistic conditions, r(18) = .35, p < .07.

Pain reduction self-efficacy. Subjects' initial baseline perceived self-efficacy to reduce experienced pain did not predict pain tolerance for the total sample. However, the mathematically self-in efficacious subjects given saline, whose pain sensitivity was apparently blunted by opioid activity, were able to translate their perceived pain-reductive capabilities into pain-tolerant behavior. The stronger their perceived self-efficacy at the outset to reduce pain, the longer they endured painful stimulation in the postinjection tests, r(8) = .55, p < .05. Their low mathematical self-efficacious counterparts, who received the opiate antagonist, exhibited a negative, although nonsignificant, relation between perceived pain-reductive efficacy and level of pain tolerance, r(8) = - .27.

For the mathematical self-efficacious subgroups, the correlations between pain reduction self-efficacy at baseline and pain tolerance in the postinjection tests were r(8) = .46, p < .10, under saline treatment, and r(8) = -.10, ns, under naloxone treatment.

Impairment of Mathematical Self-Efficacy and Pain Tolerance

The powerful efficacy induction produced subgroups that differed markedly in mathematical self-perceived efficacy and stress, with minimal variance within the subgroups. The severely curtailed range of scores for these variables precluded any meaningful correlational analysis. However, one of the variables that has bearing on stressfulness of the cognitive task provided a wider variation of scores for correlational analysis. This variable is the percentage impairment in subjects' perceived mathematical self-efficacy as calculated from their self-judged efficacy before and after the computational task.

The subgroup combining perceived self-inefficacy with the saline treatment yielded a moderately high correlation between
magnitude of self-efficacy impairment and pain tolerance. The greater the loss subjects suffered in perceived mathematical self-efficacy, the longer they were able to endure pain in the postinjection tests, $r(8) = .57, p < .05$. In sharp contrast, self-efficacy impairment had a strong opposite effect under conditions in which endogenous opioid mechanisms were controlled by naloxone. The more subjects suffered impairment in their perceived mathematical self-efficacy, the less able they were to bear pain, $r(8) = -.59, p < .04$.

Although self-efficacious subjects did not significantly alter their pain tolerance under saline treatment, the degree of change in their perceived mathematical self-efficacy accounted for a large portion of the variance in pain tolerance exhibited by this subgroup. The more they raised their perceived mathematical self-efficacy, the better they tolerated painful stimulation, $r(8) = .84, p < .01$ (two-tailed test). However, for the self-efficacious subgroup given naloxone, gains in perceived mathematical self-efficacy were unrelated to pain tolerance.

**Changes in Heart Rate and Pain Tolerance**

Under conditions of opioid blockage by naloxone, no relations were expected between changes in autonomic arousal and pain tolerance, and none were found. However, some positive relations were obtained between these variables under saline conditions. The higher the heart rate elevation relative to baseline level, the greater was the increase in pain tolerance at the 30-min test. These relations are $r(16) = .36, p < .07$, for elevated arousal during the first 2 min of the computational task, and $r(16) = .36, p < .07$, for arousal in the first third of the task.

**Discussion**

The results of the present experiment provide evidence that perceived self-efficacy in coping with cognitive stressors activates endogenous opioid systems. Perceived coping inefficacy was highly stressful and autonomically arousing. Subjects who perceived themselves as unable to exercise control over cognitive demands experienced a high level of stress, mental strain, and perceived impairment in cognitive functioning. In sharp contrast, subjects who had developed a strong sense of controlling efficacy were relatively unperturbed by the cognitive task.

Perceived coping inefficacy not only activated higher autonomic arousal during the problem-solving, but left subjects in a sensitized inefficacious state that persisted beyond the task. Thus, when simply asked to judge their capabilities after the cognitive task was over, the perceived self-infficacious subjects displayed a heightened autonomic arousal, whereas the perceived self-efficacious ones exhibited a marked drop in autonomic arousal. The autonomic reductive effects of perceived self-efficacy are especially striking. Stress reactions can be cognitively activated by self-referent thought (Bandura, 1986). Because of this capacity for cognitive self-activation, arousal does not simply dissipate with the termination of the stressor. The empty intervals between cold-pressor tests provided ample opportunities for cognitive reactivation of divergent levels of autonomic arousal.

Naloxone and saline treatments had substantially different effects on the self-infficacious stressed groups and the self-efficacious nonstressed groups. Subjects who had a strong sense of controlling efficacy were not sufficiently stressed to activate endogenous opioids to block pain transmission. Their pain tolerance did not change significantly across repeated cold-pressor tests under either saline or naloxone treatments. The lack of any differences in tolerance of pain stimulation between saline and naloxone subgroups of self-efficacious subjects indicates an absence of opioid activity in self-efficacious nonstressed subjects.

In contrast, the self-infficacious stressed subjects were able to withstand increasing amounts of pain stimulation under saline conditions. However, when the endogenous opioid mechanisms that control pain were blocked by naloxone, the subjects were unable to bear much pain stimulation. This pattern of changes suggests that the stress-induced analgesia found under the saline condition was mediated by endogenous opioid mechanisms and counteracted by the opiate antagonist. In accord with previous findings, the naloxone antagonistic effect became evident after sufficient time had elapsed for the drug to exert its effects (Bandura et al., 1987; Levine et al., 1978).

Experience of controllability produced substantial cognitive changes in perceived self-efficacy. Thereafter, mere self-appraisal of coping capabilities autonomically aroused the perceived self-infficacious subjects, but calmed the perceived self-efficacious ones. These different stress reactions suggest that, after self-efficacy beliefs are instilled, simply approaching environmental demands in a self-infficacious frame of mind may produce some opioid activation anticipatorily. Any reductions in pain sensitivity could make coping with aversive situations easier. Preparatory opioid activation by self-infficacious thought remains a significant problem to be investigated.

Initial strength of perceived self-efficacy to endure pain predicted how well subjects managed subsequent pain stimulation. The stronger their beliefs in their ability to withstand pain, the longer they endured mounting pain under all treatment conditions. These findings are in accord with several lines of evidence supporting the positive role of perceived self- regulatory efficacy in coping with acute pain (Bandura, in press; Litt, 1988; Manning & Wright, 1983; Vallis & Bucher, 1986) and with chronic clinical pain (Holroyd et al., 1984; O'Leary, Shoer, Lorig, & Holman, in press; Phillips, 1987; Shoer & Holman, 1984). As might be expected, perceived self-efficacy exercised weaker pain control under conditions in which sensitivity to painful stimulation was increased by blockage of endogenous opioid mechanisms. It is much more difficult to translate self-efficacy belief into pain-tolerating behavior in a physical state of heightened pain sensitivity.

Perceived self-efficacy to effect reductions in experienced pain reflects a more active exercise of personal agency than does stoic endurance. Consideration of the contributions of pain control self-efficacy and cognitive self-efficacy to variance in pain tolerance under different perceived controllability and drug conditions reveals interesting patterns of results. As will be recalled, the perceived self-efficacious nonstressed subjects exhibited no significant changes in pain tolerance over the series of postinjection tests. However, the predictors of variance in pain tolerance for these subjects differs depending on whether
they received the saline or naloxone treatment. Variation in pain tolerance for those administered naloxone seemed to be due, in part, to force of effort as reflected in the positive relation between belief in ability to endure pain and level of pain tolerance. For the subjects administered the saline solution, the predictors of variance in pain tolerance were perceived self-efficacy to withstand pain as well as enhancement of cognitive self-efficacy. Evidently, a boost in perceived self-efficacy in the cognitive domain was transferred to a more perseverant effort in the pain coping domain.

The perceived self-inefficacious subjects who were administered the opiate antagonist were also unable to endure much pain. But the predictors of their variance in pain tolerance differ in an important respect from their self-efficacious naloxone counterparts. Belief in efficacy to withstand pain and low impairment of perceived cognitive self-efficacy presaged pain tolerance. These correlates indicate reliance on forbearance and cognitive resilience in efforts to cope with pain sensations under the state of heightened pain sensitivity.

The substantial increase in pain tolerance achieved by self-inefficacious subjects administered saline seemed to rest on the stress of perceived cognitive impairment and active exercise of self-regulatory efficacy. With the blunting of pain sensitivity by opiate activity, they could readily act on their perceived self-efficacy to withstand and reduce pain sensations.

As noted, increased pain tolerance was predicted by perceived enhancement of cognitive self-efficacy in self-efficacious saline subjects, but by perceived impairment of cognitive self-efficacy in self-inefficacious saline subjects. These differential predictors may help to explain why these two groups did not show even greater divergence in pain tolerance. Among subjects in the self-efficacious saline group, those who experienced a sizable boost in cognitive self-efficacy achieved notable increases in pain tolerance. This reduced intergroup differences. Measurement of different possible determinants helps to clarify the sources of variance and magnitude of change in pain tolerance under different efficacy and drug conditions.

It is interesting to speculate on how the capacity of stressors to activate endogenous opioids evolved. In physically threatening situations entailing fear and pain, it would be highly adaptive to possess an opioid system to alleviate pain so that vigorous defense or speedy flight can be successfully executed. Stress-induced analgesia would clearly be advantageous in the struggle for physical survival. Indeed, Fanselow (1986) has shown that through paired experience with painful stimulation, danger signals acquire the ability to activate endogenous opioids that attenuate the aversiveness of painful stimuli and facilitate defensive behavior.

Modern-day struggles involve strenuous activities and prolonged exposure to psychological stressors on athletic fields, in classrooms, in social circles, and in occupational settings. These stressors produce their own set of aches, strains, pains, headaches, and other physical discomforts. Success in difficult undertakings requires perseverant effort in the face of many stressful and aversive elements. To abort one's efforts prematurely because of accompanying discomfort is self-limiting. People are better able to deal with stressful environmental demands if not beset by pain. Thus, for example, opioid reduction of pain sensitivity in exercise stress helps athletes to achieve high performances that they might otherwise forsake (Janal, Colt, Clark, & Glusman, 1984). An endogenous mechanism that enables people to handle stressful situations with some relief from physical aversiveness has some advantageous functions.

Benefits rarely come devoid of costs. A growing body of evidence reveals that the stress of coping inefficacy not only activates endogenous opioids, but also impairs cellular components of the immune system (Kiecolt-Glaser & Glaser, 1988; Maier et al., 1985; Shavit & Martin, 1987). Prolonged impairment of the immune function increases vulnerability to infection. Physiological systems are highly interdependent. There is evidence that some of the immunosuppressive effects of inefficacy in controlling stressors are mediated by release of endogenous opioids (Shavit & Martin, 1987). When opioid mechanisms are blocked by naloxone, the stress of coping inefficacy loses its immunosuppressive capabilities. Thus, the benefits of reduced pain sensitivity may be gained at the cost of immunocompetence.

References


