Perceived Self-Efficacy and Pain Control: Opioid and Nonopioid Mechanisms

Albert Bandura, Ann O'Leary, C. Barr Taylor, Janel Gauthier, and Denis Gossard
Stanford University

In this experiment, we tested for opioid and nonopioid mechanisms of pain control through cognitive means and the relation of opioid involvement to perceived coping efficacy. Subjects were taught cognitive methods of pain control, were administered a placebo, or received no intervention. Their pain tolerance was then measured at periodic intervals after they were administered either a saline solution or naloxone, an opiate antagonist that blocks the effects of endogenous opiates. Training in cognitive control strengthened perceived self-efficacy both to withstand and to reduce pain; placebo medication enhanced perceived efficacy to withstand pain but not reductive efficacy; and neither form of perceived self-efficacy changed without any intervention. Regardless of condition, the stronger the perceived self-efficacy to withstand pain, the longer subjects endured mounting pain stimulation. The findings provide evidence that attenuation of the impact of pain stimulation through cognitive control is mediated by both opioid and nonopioid mechanisms. Cognitive copers administered naloxone were less able to tolerate pain stimulation than were their saline counterparts. The stronger the perceived self-efficacy to reduce pain, the greater was the opioid activation. Cognitive copers were also able to achieve some increase in pain tolerance even when opioid mechanisms were blocked by naloxone, which is in keeping with a nonopioid component in cognitive pain control. We found suggestive evidence that placebo medication may also activate some opioid involvement. Because placebos do not impart pain reduction skills, it was perceived self-efficacy to endure pain that predicted degree of opioid activation.

Pain is a complex psychobiologic phenomenon influenced by psychosocial factors rather than simply a sensory experience arising directly from stimulation of pain receptors. Level of pain depends not only on the intensity of sensory stimuli but on how attention is deployed, how the experience is cognitively appraised, the coping strategies used to modulate pain, and modeled reactions to nociceptive stimulation (Craig, 1983; Turk, Meichenbaum, & Genest, 1983). The same intensity of nociceptive stimulation can thus give rise to varying conscious perceptions of pain. Evidence that psychosocial determinants play an important role in perceived pain has led to the development of psychobiologic models of pain (Melzack & Wall, 1982). A number of psychological procedures have been shown to attenuate pain and raise pain tolerance. Placebos can bring pain relief to many people. The analgesic potency of placebos mimics variations in the impact of drugs, producing additive effects, dose-level effects, and greater pain relief from a placebo injection than from a placebo pill (Evans, 1974). Cognitive coping strategies, which rely extensively on attentional diversion and cognitive restructuring of painful experiences, have also proven effective in alleviating pain (Hilgard & Hilgard, 1975; McCaul & Malott, 1984; Turk et al., 1983). Self-relaxation serves as another means of lessening pain (Sanders, 1979).

Although pain control by psychological means is well established, the mechanisms by which they enhance pain tolerance are less well understood. In this experiment, we sought to clarify the relation between psychological and physiological mechanisms of pain control. Perceived self-efficacy is one psychological mechanism that has been shown to mediate different forms of physiological activation and health-related behavior (Bandura, in press; O'Leary, 1985a). Perceived self-efficacy is concerned with people's judgments of their capabilities to execute given levels of performance and to exercise control over events. Judgments of personal efficacy affect what courses of action people choose to pursue, how much effort they will put forth in a given endeavor, how long they will persevere in the face of aversive experiences, whether their thought patterns help or hinder their endeavors, and how much stress they experience in coping with taxing environmental demands (Bandura, 1986).

There are several ways in which perceived coping efficacy can bring relief from pain. People who believe they can alleviate suffering will likely mobilize whatever ameliorative skills they have learned and will persevere in their efforts. Those who doubt their controlling efficacy are likely to give up readily in the absence of quick results. A sense of coping efficacy also re-
duce distressing anticipations that create aversive physiological arousal and bodily tension, which only exacerbate pain sensations and discomfort (Bandura, Reese, & Adams, 1982; Bandura, Taylor, Williams, Mefford, & Barchas, 1985). The attentional resources available at any given moment are severely limited (Kahneman, 1973). It is hard to attend to more than one thing at a time. Dwelling on pain sensations makes them more noticeable and thus more difficult to bear. Perceived self-efficacy can lessen experienced pain by diverting attention from pain sensations to competing engagements. To the extent that pain sensations are supplanted in consciousness, they are felt less.

Results of several lines of research indicate that perceived self-efficacy can mediate the analgesic potency of different psychological procedures. Reese (1983) found that cognitive techniques, self-relaxation, and placebos all increase perceived self-efficacy to cope with pain, with cognitive techniques being most effective. The more self-efficacious the people judged themselves to be, the less painful they experienced later cold pressor tests and the higher was their pain threshold and pain tolerance. Holroyd and his colleagues (Holroyd et al., 1984) demonstrated that perceived self-efficacy, created by false feedback during biofeedback training, predicted reduction in tension headaches, whereas the actual amount of control over muscular activity achieved in treatment was unrelated to the incidence of subsequent headaches. Changes in perceived self-efficacy to regulate pain, induced through bogus social comparative feedback, similarly predict magnitude of change in pain tolerance (Litt, 1987).

That perceived self-efficacy makes pain easier to control is further corroborated by studies of acute and chronic clinical pain. In a study of primiparous women who had completed childbirth classes, Manning and Wright (1983) found that the higher their perceived self-efficacy that they could exercise control over pain while giving birth, the longer they tolerated labor pain before requesting medication and the less pain medication they used. Shoor and Holman (1984) documented the influential role of perceived self-efficacy in coping with the chronic pain of arthritis. When patients are equated for degree of physical debility and other relevant factors, those who believe they can exercise some influence over their pain and arthritic condition lead more active lives and experience less pain. O'Leary (1985b) found that training in self-regulatory skills increased the perceived efficacy of patients suffering from rheumatoid arthritis to reduce pain and to pursue potentially painful activities. The stronger their perceived coping efficacy, the less pain they experienced, the less they were disabled by their arthritis, and the greater reduction they achieved in inflammation of their joints.

Research has clarified some aspects of the physiological mechanisms mediating pain reduction. Studies by Levine and his associates with postoperative dental pain indicate that endogenous opioids can be activated by a placebo (Levine, Gordon, & Fields, 1978; Levine, Gordon, Jones, & Fields, 1978). A socially administered placebo produces analgesia that is antagonizable by naloxone, whereas unsignaled mechanical infusion of the placebo has no analgesic effect (Levine & Gordon, 1984). There is some evidence to suggest that placebo-induced analgesia may involve both a nonopioid component and a stress-analgetic component that is antagonizable by naloxone (Gracey, Dubner, Walskee, & Deeter, 1983).

It might be reasoned from research with animals on stress-induced analgesia that coping efficacy may enhance pain control mainly through nonopioid mechanisms. Stress can activate endogenous opioids that block pain transmission (Maier, Laudenslager, & Ryan, 1985). Thus, the pain reactivity of animals who have controlled shock offset is unaffected by opiate antagonists, whereas yoked animals who experienced equivalent amounts of shock without being able to control it display pain analgesia that is counteracted by opiate antagonists. 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.

At first sight, helplessness theory and self-efficacy theory appear to be at odds on how controlling efficacy relates to pain tolerance and the mechanisms mediating it. Endurance of pain is associated with deficient control in helplessness theory but with controlling efficacy in self-efficacy theory. A plausible explanation for the seemingly contradictory findings is in terms of the markedly different consequences of control in the types of coping situations used.

The exercise of control produces fundamentally different conditions of pain stimulation in the common animal and human coping situations that would argue for some opioid involvement with high self-efficacy. In the typical animal study, control promptly terminates pain stimulation. By contrast, in the human situation, active exercise of personal efficacy can attenuate conscious pain, but in so doing it can promote even more taxing pursuits that heighten the level and duration of pain stimulation. Indeed, a strong sense of coping efficacy often increases engagement in pain-generating activities to the point where it can create stressful predicaments. Thus, for example, self-efficacious people suffering from arthritis generate pain and discomfort when they first take on more vigorous activities, or subjects experience more severe pain stimulation the longer they endure the cold pressor task. Indeed, in the cold pressor situation, continuing exercise of controlling efficacy through cognitive means eventually heightens pain to the point where it begins to overwhelm one's coping capabilities. The stress of failing with mounting pain in later stages of coping would activate opioid systems. According to this conception of the human coping process, both opioid and nonopioid mechanisms operate in the regulation of pain, but their relative contribution varies with degree of controlling efficacy and stages of coping. A nonopioid mechanism would subserve pain tolerance while cognitive control is effectively exercised, but an opioid mechanism would come into play in later stages of coping when control techniques are no longer sufficient to attenuate increasing pain or to block it from consciousness.

This study was designed to test for opioid and nonopioid mechanisms of pain control and the relation of opioid involvement to perceived coping efficacy when perceived self-efficacy is enhanced by placebos or by cognitive control strategies. Subjects were taught cognitive methods of pain control, were administered a placebo presented as a medicinal analgesic, or received no intervention. Following the treatment phase, their
perceived efficacy to control and reduce pain and their tolerance of cold pressor pain were measured. In all conditions, subjects were then administered either naloxone or a saline solution, and thereafter their perceived self-efficacy and their pain tolerance were measured at periodic intervals.

We predicted that because instruction in cognitive strategies imparts skills for controlling pain, it would prove more effective than placebos in enhancing perceived coping efficacy and pain tolerance. The stronger the subjects’ perceived coping efficacy, the longer they would be able to endure mounting pain. On the assumption that pain tolerance under the treatment conditions reflects some opioid involvement that is antagonizable by naloxone, we predicted that naloxone subjects would display lower pain tolerance than would their saline counterparts after the time needed for the drug impact had elapsed. The stronger the perceived efficacy, the greater should be the naloxone counteraction of analgesia. Subjects taught cognitive means for attenuating the impact of pain stimulation were also expected to be able to achieve some increase in pain tolerance independent of opioid mechanisms. The control condition was not expected to produce any opioid involvement because subjects would terminate the pain task quickly. Hence, naloxone and saline controls should not differ from each other.

**Method**

**Subjects**

The subjects were 36 men and 36 women drawn from an introductory psychology course. They were randomly assigned to conditions, balanced for sex, with 12 subjects in each of the four treatment conditions and two control groups. Two subjects in the cognitive control condition who later received saline and 1 placebo subject who later received naloxone achieved maximal pain tolerance in the posttreatment assessment. Inclusion of these subjects is appropriate for evaluating the power of treatments, but it could preclude changes for the subsequent phase of the study because their tolerance was already at the highest level and could not increase any further. A maximum posttreatment level is not as problematic for the naloxone subject because it provides the greatest leeway for reductive change in pain tolerance, and naloxone was expected to decrease rather than raise pain tolerance. Nevertheless, 3 additional subjects were added from the same subject pool and substituted in the test of opioid involvement. The experiment was presented as part of a program of research investigating psychological and physiological mediators of pain.

**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 warm water kept at 37 °C was used before 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 bilge pump and maintained at a constant temperature of 0 °C.

Subjects were instructed to place their dominant hand in the warm water for 3 min to equalize initial hand temperatures. They were then asked to immerse their hand in the ice water for as long as they could. The test had a 5 min ceiling. The pain tolerance score was the number of seconds subjects were able to keep their hands in the ice water. Those whose pain tolerance exceeded 2.5 min in this screening assessment were excluded from the study. The mean pretest pain tolerance was 56 s. The study was confined to subjects who had difficulty coping with pain to provide a stringent test of the treatments and to allow wide latitude for change. Forty percent of all the subjects who had been pretested met this criterion.

**Perceived Self-Efficacy**

Self-efficacy scales were devised to measure perceived self-efficacy to withstand pain and perceived self-efficacy to reduce its intensity. To familiarize subjects with the format for recording their self-efficacy judgments, they first completed a practice scale measuring their self-judged capability to lift objects of varying weights. In judging their perceived efficacy to tolerate pain, subjects were presented with 20 items representing increasing lengths of cold pressor stimulation, ranging from 15 s to 5 min. The items in the scale measuring pain reduction efficacy described four severities of pain ranging from dull to excruciating and for each severity three degrees of pain reduction, namely small, moderate, or large reductions. On each scale, subjects checked the items they judged they could perform. For each item so designated, they rated the strength of their perceived self-efficacy on a 100-point scale, ranging in 10-unit intervals from high uncertainty through intermediate values of certainty to complete certitude.

Prior research has shown that recording self-percepts of efficacy to control pain has no reactive effects on pain tolerance (Reese, 1983). The measures of strength of perceived self-efficacy to cope with pain were obtained by dividing the summed magnitude scores by the total number of items.

Subjects who met the selection criterion on the initial cold pressor test were scheduled for participation in the formal experiment several days later at a research facility of the Stanford Medical Center. They were administered a pretreatment cold pressor test and judged their efficacy to withstand and to reduce pain, both before and after the test of pain tolerance.

**Treatment Conditions**

Subjects were randomly assigned to one of three conditions, each of which lasted 30 min.

**Cognitive coping.** Subjects assigned to the cognitive coping condition received instruction and practice in using different cognitive strategies for alleviating pain. These strategies included attention diversion from pain sensations to other matters, vivification of engrossing imagery, dissociation of the limb in pain from the rest of the body, cognitive transformation of pain as nonpain sensations, and self-encouragement of coping efforts.

Subjects were provided with several examples of each strategy. They selected those they believed would be most effective and practiced them. This individualized approach was used to ensure that subjects could choose and switch strategies quickly when they later had to manage cold pressor pain.

**Placebo medication.** Subjects in the placebo condition were given a placebo pill and asked to wait for 30 min for the drug to take effect. The placebo was administered by a male physician who described it as a widely used medicinal analgesic. To enhance further the credibility of the placebo administration, the physician conducted a brief screening interview as to whether subjects were currently taking any medication or were allergic to any kind of medication.

**Control condition.** Subjects assigned to the control condition received the same orienting instructions, but they simply waited for 30 min. This group provided a control for the effects of the common instructional component and repeated measurements.
Posttreatment Assessment

At the end of the 30-min period, all subjects were administered the self-efficacy scales, then the cold pressor test, and then readministered the self-efficacy scales. All the treatments were conducted by two female experimenters, and all the measurement procedures were administered by a separate female tester at all points of assessment. To control for any possible bias, the assessor had no knowledge of the treatment conditions to which any subject had been assigned.

Naloxone Intervention

To test whether changes in pain tolerance are mediated by activation of the endorphin system, half the subjects in each condition received an injection of 10 mg of naloxone, an opiate antagonist. The other half were given a placebo injection of saline solution. The subjects were informed at the outset of the experiment that the injection would contain a drug that may affect the physical mechanism controlling pain but that its individual effects on the experience of pain were not yet fully known. The physician administered the injections under a double-blind procedure. Neither the physician nor the tester knew whether the subjects received naloxone or saline.

Postinjection Tests

The self-efficacy scales were administered before and after each of three cold pressor tests during the 60-min period following the injection. The assessments were performed at 5, 20, and 60 min after the injection. The lengthier times were chosen because Levine, Gordon, and Fields (1978) found that naloxone antagonist effect does not become evident until after about 20 min.

Results

Analysis of variance on pretest scores showed that the groups did not differ initially in their perceived self-efficacy either to withstand or reduce pain or in their actual ability to tolerate pain. Nor were there any significant sex differences or interactions between sex and treatment effects in percentage change as a function of treatment on any of the measures.

Perceived Coping Self-Efficacy

The mean percentage change in perceived self-efficacy compared with the pretest level is plotted in Figure 1 for the different treatment conditions. Treatment had a highly significant effect on perceived self-efficacy both to withstand pain, F(2, 67) = 5.31, p < .01, and to reduce pain, F(2, 67) = 9.17, p < .001.

In intragroup comparisons, evaluated by the t test for correlated means, control subjects displayed no significant changes in either form of perceived self-efficacy. Administration of the placebo raised subjects' perceived self-efficacy to withstand pain, t(24) = 4.20, p < .001, but did not alter their perceived efficacy to effect reductions in pain. Training in cognitive coping strategies heightened perceived self-efficacy both to withstand pain, t(25) = 4.01, p < .001, and to reduce it, t(25) = 3.32, p < .005.

In analysis of intergroup differences, subjects in the cognitive control condition judged themselves considerably more efficacious at withstanding pain than did subjects in the placebo condition, t(49) = 2.30, p < .025, or in the control group, t(47) = 3.14, p < .01. The cognitive copers also judged themselves much more efficacious at reducing pain than did the placebo subjects, t(48) = 3.61, p < .01, or the control subjects, t(48) = 3.98, p < .001. The differences between the placebo and control conditions in perceived self-efficacy to withstand or to reduce pain were not statistically significant.

Posttreatment Pain Tolerance

Treatment effects were evaluated in terms of percentage change in pain tolerance because this measure controls for individual differences in initial ability to withstand pain and has been shown to be more sensitive to treatment influences than are simple difference scores (Hilgard et al., 1974). Figure 1 presents the mean percentage change from the pretest level in tolerance of cold pressor pain as a function of treatment conditions.

The obtained significant treatment effect, F(2, 67) = 8.15, p < .001, was due mainly to the noteworthy power of cognitive control. Cognitive copers substantially increased their ability to tolerate pain stimulation, t(25) = 4.02, p < .001, and surpassed both the placebo, t(49) = 3.49, p < .001, and control, t(48) = 3.66, p < .001, subjects in this regard. The latter two groups did not alter their pain tolerance and were similar in this respect in the immediate posttreatment assessment. However, at the 60-min posttreatment assessment, placebo subjects who had received saline significantly increased their pain tolerance (23%) compared with their pretest level, t(11) = 2.01, p < .05. In contrast, control subjects who had received saline showed no significant (12%) change in pain tolerance, t(11) = 1.20.

Relation of Perceived Self-Efficacy to Pain Tolerance

Product-moment correlations between perceived self-efficacy at the end of treatment and pain tolerance were computed separately for the different treatment conditions. When the obtained correlations were of comparable magnitude, they were averaged across treatments by means of an r-to-z transformation.

Self-efficacy to withstand pain. Subjects' perceptions of their efficacy to withstand and reduce pain were correlated with their level of pain tolerance at the end of treatment. Perceived self-efficacy to withstand pain and to reduce pain were related at a moderate positive level in all three conditions. The average correlation was r(70) = .30, p < .01.

Perceived self-efficacy to withstand pain was uniformly highly related to pain tolerance regardless of whether subjects were in the cognitive (r = .64), placebo (r = .61), or control (r = .90) condition. The average correlation between perceived self-efficacy and pain tolerance was r(71) = .75, p < .0001. The average correlation between level of pain tolerance in the pretest and perceived self-efficacy to endure pain in the posttreatment assessment was r(71) = .84, p < .0001. Pretest pain tolerance also correlated with posttreatment pain tolerance, r(71) = .82, p < .0001. Perceived self-efficacy retained its significant relation to pain tolerance even when pretest pain tolerance was partialled out, r(71) = .20, p < .05. The more strongly subjects believed they could endure pain, the longer they tolerated painful stimulation.

People's perceptions of their efficacy to cope with pain un-
doubtedly influenced how long they tolerated pain in the pretest. It is not as though perceived self-efficacy affected future pain tolerance but had no effect whatsoever on pretest pain tolerance. Because pretest tolerance partly reflects the influence of perceived self-efficacy, partialing out prior tolerance also removes the perceived efficacy component from the effect of subsequent perceived efficacy on pain tolerance. Hence, partial coefficients most likely underestimate the magnitude of the actual relation between perceived self-efficacy and endurance of pain.

**Self-efficacy to reduce pain.** Perceived self-efficacy to reduce pain represents a more active exercise of personal control. How long subjects could tolerate pain in the pretest was related across conditions to the strength of their belief that they could alleviate pain, $r(72) = .21, p < .05$. In the placebo condition, in which the presumed medicinal aid would be regarded as the main source of enhanced control, perceived reductive efficacy did not correlate with pain tolerance in the posttreatment phase ($r = -.13$). However, for the cognitive copers and control subjects, who had to draw on their own coping resources to ameliorate pain, the stronger their posttreatment belief that they could reduce pain, the longer they tolerated the mounting pain stimulation, $r(48) = .34, p < .01$. However, the partial coefficient controlling for pretest tolerance ($r = .08$) is nonsignificant.

**Test for Opioid Mechanism**

In Figure 2, the percentage change in pain tolerance from the posttreatment level is plotted as a function of whether subjects received saline or naloxone. The critical test of opioid activation involves intratreatment comparison between naloxone and saline conditions. If endogenous opiates have been activated, naloxone subjects should exhibit lower pain endurance than should their saline counterparts. If there is no opioid involvement, whether subjects receive naloxone or saline should make no difference. The differences between these two conditions in percentage change in pain tolerance were tested at each of the three postinjection periods for each of the treatment conditions. However, the comparisons of primary interest concerned the predicted naloxone antagonistic effects in the placebo and cognitive control conditions at the 20-min and 60-min periods.

Naloxone and saline control subjects did not differ significantly at any point. The pain tolerance of subjects in the placebo condition rose over time for those administered saline but declined over time for those given naloxone. Indeed, at the end of the 60-min period, 83% of the saline subjects had raised their pain endurance, whereas only 42% of the naloxone subjects had done so. The difference between these two proportions, which is highly significant ($z = 2.07, p < .02$), assumes added import from evidence that naloxone had no effect in the control condition. Subjects were just as likely to raise their pain tolerance with naloxone (58%) as with saline (42%) ($z = .78, ns$).

Because of a highly divergent subject, the difference between the saline and naloxone conditions in percentage change in pain tolerance at the 60-min period was not statistically significant. One of the placebo subjects in the naloxone condition had been replaced with another subject because he had reached the maximum level of tolerance in the posttreatment test. Naloxone had a marked effect on this subject's ability to endure pain, producing a 68% decrease in pain tolerance. A Mann-Whitney $U$ test with this subject included yielded a marginally significant naloxone antagonistic effect at the 60-min test for magnitude of change in pain tolerance ($z = 1.52, p = .06$).

The subjects whose coping efficacy was enhanced by cognitive pain control techniques show evidence of opioid activation after sufficient time had elapsed for naloxone to exert its antagonistic effect. When tested at 20 min, cognitive copers who were given naloxone were much less able to endure pain than were those who were administered the saline solution, $t(22) = 1.96, p <$
Naloxone cognitive copers also displayed lower pain tolerance than did saline cognitive copers at the 60-min test, although at a slightly lower level of significance, t(22) = 1.56, p < .06.

The perceived self-efficacy of cognitive copers at the end of treatment to withstand pain did not predict the extent to which naloxone affected pain tolerance. However, perceived self-efficacy to reduce pain, which involves a more active exercise of personal control, predicts the magnitude of the naloxone effect at 20 min, the point at which naloxone had the greatest impact. The higher the posttreatment perceived self-efficacy to reduce pain, the more naloxone reduced subjects' pain endurance, r(10) = .48, p = .058. At the 60-min test, where there seemed to be somewhat less opioid involvement, perceived self-efficacy to reduce pain was still moderately related to the extent of the naloxone effect, r(10) = .37, but with the small number of subjects the correlation fell short of significance.

Initial level of pain tolerance correlated negatively, though nonsignificantly, with degree of naloxone effect at 20 min (r = -.18) and 60 min (r = -.33) and at .12 with posttreatment reductive self-efficacy. The strength of the relation between perceived self-efficacy to reduce pain and degree of opioid activation is further increased at both the 20-min (r = .51, p < .05) and 60-min (r = .44, p < .08) periods when initial level of pain tolerance is controlled by partial correlation.

Placebo medication strengthened perceived self-efficacy to endure pain but did not alter perceived capability to alleviate it. For placebo subjects, it is the former aspect of perceived self-efficacy that predicts the extent to which naloxone reduced pain tolerance in the 60-min test, where there appeared to be some opioid involvement. The stronger the perceived self-efficacy to withstand pain at the end of treatment, the greater the naloxone antagonistic effect (r = .54, p < .03). The partial coefficient (r = .56, p < .025) was also significant.

As might be expected, no significant correlations were found between perceived self-efficacy and analgesia in conditions and test periods in which naloxone had no opioid antagonistic effect.

**Test for Nonopioid Mechanism**

In the preceding tests for opioid involvement, differences between saline and naloxone subgroups within each treatment constituted the relevant comparison. Although subjects preassigned to the saline and naloxone subgroups were comparable in the posttreatment assessment before receiving the injection, percentage change in pain tolerance was calculated from the posttreatment level to control for even any minor variations within treatments. Tests for attenuation of pain stimulation through a nonopioid mechanism of operation are concerned with differences between treatments under conditions of opioid blockage. Therefore, for this analysis, percentage change in pain tolerance at each of the three postinjection tests was calculated from the pretest baseline level. A nonopioid mechanism is indicated if treatment subjects surpass control subjects in pain tolerance in the subgroups in which opioid mechanisms are blocked by naloxone. If pain tolerance is regulated solely by opioid mechanisms, cognitive control should produce no increases in pain tolerance under naloxone.

The mean changes in pain tolerance achieved by the three naloxone subgroups are plotted in Figure 3 for each test period. Unlike naloxone blockage, which takes time to produce its effects, cognitive control can be exercised fully from the outset.
Indeed, cognitive copers significantly increased their pain endurance at every test: at 5 min, t(10) = 3.49, p < .005; at 20 min, t(11) = 3.33, p < .005; and at 60 min, t(11) = 2.63, p < .025. Neither the modest increases by the placebo subjects nor the minimal changes by the controls reached statistical significance at any of the time points. In intergroup comparisons, the changes achieved by the cognitive copers surpassed those of the controls at 5 min, t(20) = 2.42, p < .025; at 20 min, t(22) = 2.36, p < .025; and at 60 min, t(22) = 1.97, p < .05. The intermediate changes displayed by the placebo subjects did not differ significantly from those in the cognitive or control conditions.

Discussion

Results of this experiment attest to the effectiveness of cognitive control of pain and to the influential role that perceived self-efficacy plays in this process. Training in cognitive control strategies heightened perceived self-efficacy to endure and to alleviate pain. The enhanced perceived self-efficacy was accompanied by a substantial increase in pain tolerance. These changes, achieved by cognitive control in both perceived self-efficacy and pain tolerance, far surpassed those by the placebo and control conditions.

Placebo medication had a differential impact on perceived endurance self-efficacy and reductive self-efficacy. Subjects believed they were better able to withstand pain with the aid of a supposedly pain-relieving medication. However, success in reducing experienced pain depends on effective exercise of pain-ameliorating skills, which medication alone does not provide. Placebo medication did not persuade subjects that they became more capable of exercising reductive control over pain. These findings underscore the value of measuring different aspects of perceived self-efficacy in research designed to elucidate the exercise of control over pain and other complex affective states.

In accord with previous findings (Evans, 1974), about one third of the subjects displayed a positive placebo response as reflected in a notable increase in pain tolerance. However, another common finding that generally receives little attention in the literature is that about a quarter of the subjects show a negative placebo response. The net effect is a lack of an overall change in the immediate posttreatment test. But as placebo subjects continued to cope with pain, they eventually increased their pain tolerance. The nature of the placebo response is predictable from how the placebo affected subjects' perceived self-efficacy to withstand pain. Subjects who judged themselves efficacious in bearing pain given the supposed medicinal aid were good pain endurers, whereas those who continued to doubt their efficacy to manage pain despite receiving the placebo medication were less tolerant of pain. To persons with low perceived self-efficacy, the evident failure to achieve relief from pain even with the help of a medicinal analgesic gives further testimony for their coping inefficacy.

The variable effects of placebos on perceived self-efficacy most likely reflect past correlated experiences with medication. If acting on self-percepts of efficacy in conjunction with medication usually brought them substantial pain relief, people would come to judge themselves more efficacious to reduce pain with a medicinal aid. The regulatory function of perceived self-efficacy would be enlisted as well by placebo medication presented as a pain killer. The enhanced perceived self-efficacy produced by placebo medication would activate pain-relieving processes. In contrast, people who had often experienced no relief or even heightened pain despite medication would not be at all persuaded that placebo medication has enhanced their capability to relieve pain. Indeed, a low sense of efficacy to exercise control over pain may diminish the potency even of genuine analgesics. That correlated experiences affect how people respond to placebo medication was demonstrated by Voudouris, Peck, and Coleman (1985). They produced both positive and reverse placebo effects by having people experience decreases or increases in nociceptive stimulation after taking placebo medication. Whether the effects of such correlated experiences are mediated by changes in perceived coping self-efficacy remains to be determined.

Placebos have been shown to exert a more potent analgesic effect, as measured by ratings of felt intensity of pain, in studies of severe clinical pain (Gracely et al., 1983; Levine & Gordon, 1984). Because such pain is prolonged and cannot be promptly terminated when it becomes unbearable, it is much more stressful. As previously noted, heightened stress can activate opioid-mediated analgesia.

Perceived self-efficacy predicted how well subjects managed pain. The stronger their beliefs in their ability to withstand pain, the longer they endured mounting pain, regardless of whether
their perceived self-efficacy was enhanced by cognitive means or placebo medication or varied preexistently without any intervention. Perceived self-efficacy to withstand pain retains its relation to pain tolerance when initial differences in pain tolerance are controlled. Converging lines of evidence from investigations of both laboratory and clinical pain indicate that perceived self-efficacy operates as an important cognitive factor in the control of pain (Holroyd et al., 1984; Litt, 1987; O'Leary, 1985b; Reese, 1983; Shoer & Holman, 1984).

Perceived self-efficacy to reduce pain related positively to pain tolerance in the cognitive and control conditions but negatively in the placebo condition. Cognitive copers and controls had to rely on their own personal skills to effect reductions in pain. The stronger they believed in their capabilities to do so, the longer they endured mounting pain. However, this relation stems in part from preexisting capability to tolerate pain, which may itself partly reflect the exercise of perceived coping self-efficacy. Placebo medication seemed to undermine the active exercise of personal efficacy. To the extent that subjects expected analgesic medication to see them through the painful experience, they would be less inclined to mobilize and sustain coping efforts.

The effects of pharmaceuticals on perceived self-efficacy have received scant attention thus far but raise issues with important implications for treatments based solely or partly on medication. A perceived self-efficacy that rests entirely on medicinal aid will not survive withdrawal of medication (Chambiss & Murray, 1979a, 1979b). Programs that combine medication with development of coping skills may have diverse effects on perceived self-efficacy, depending on how the relative contribution of these two factors is cognitively appraised and weighted. If medication helps to create conditions that enable people to acquire generalizable skills they might otherwise fail to develop, it can enhance perceived self-efficacy. If medication facilitates skill development, and the contribution of the skill component is emphasized and medication is given little weight, it will have no additive effect. And finally, medication can undermine the efficacy-enhancing value of skill development if coping successes are ascribed to medicinal aids rather than to improved capability (Craighead, Stunkard, & O'Brien, 1981).

Efficacious exercise of cognitive control over pain sensations enables people to tolerate high levels of painful stimulation. However, the more protracted their efficacy-sustained endurance, the more pain and stress they eventually create for themselves. Indeed, subjects who had prolonged their endurance substantially struggled with increasing stress as they approached the limit of their capabilities and began to experience the pain as unbearable. Thus, for cognitive copers, a nonopioid mechanism would contribute to pain tolerance during the coping phase, when the exercise of cognitive control contravenes pain sensations, but an opioid mechanism would be enlisted in later stages of coping as people experience the stressful predicament of mounting pain with failing cognitive control. An appropriate next step in this line of research is to compare the level of opioid activation during phases of successful and failing cognitive control.

The findings of this study provide some evidence for both an opioid-mediated component and a nonopioid component for attenuating the impact of pain stimulation by cognitive means. For cognitive copers administered saline, the combined action of both mechanisms contributed to their ability to endure painful stimulation. They displayed a sizable increase in pain tolerance. In contrast, cognitive copers who were administered naloxone, an opiate antagonist, found it more difficult to manage pain. However, evidence that the cognitive copers were able to increase their pain tolerance even under opioid blockage lends support for a nonopioid component in the exercise of cognitive pain control. It could be argued that naloxone did not produce complete opioid blockage. However, the use of a high dosage of naloxone, which in other studies has been shown to block opioid activity fully, would argue against such an interpretation.

The correlational findings provide new information on how different forms of self-efficacy relate to opioid activation. Coping with heightened pain accompanying naloxone requires active exercise of strategies for alleviating pain rather than mere forbearance. People who judge themselves good pain copers would be especially distressed by their eventual ineffectiveness to manage their pain. It is perhaps for these reasons that the degree of opioid activation is best predicted by perceived capability to reduce pain. The stronger the subjects' perceived self-efficacy to reduce pain, the greater was the opioid activation. An alternative possibility to mediation through stress is that self-efficacy expectations directly activate the central nervous system to release pain-blocking opioids independent of stress. Evidence that animals can learn to activate their endogenous opiate systems in the presence of cues formerly predictive of painful experiences (Watkins & Mayer, 1982) adds some credence to the possibility of direct central activation.

Our findings also provide suggestive evidence that placebo medication may activate some opioid involvement. After the full time had elapsed for naloxone to exert its antagonistic effect, subjects in the naloxone condition were less able to tolerate pain than were those who had been given saline. Because placebo medication had its major impact on perceived self-efficacy to withstand pain, it was this expression of efficacy that predicted degree of opioid involvement.

Studies of acute pain arising from dental surgery provide strong evidence of placebo-induced analgesia that is blocked by naloxone (Gracey et al., 1983; Levine & Gordon, 1984). The weaker placebo response and opioid involvement obtained in this experiment may be due to the fact that cold pressor pain is much less stressful than acute clinical pain, which is more intense, prolonged, and cannot be terminated at will. However, evidence is conflicting on whether acute clinical pain involves a stress-activated opioid component. Surgery is not only stressful but also involves some physical trauma that can activate a variety of complicating physiological processes. The role of stress in placebo analgesia can perhaps be best clarified by examining naloxone hyperalgesia accompanying placebo medication with and without psychologically induced stress. How perceived self-efficacy arising from placebo medication might activate release of pain-relieving opiates independent of stress is also an intriguing issue that awaits further research.

References

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