

Cortical Dynamics as a Therapeutic Mechanism for Touch Healing

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ABSTRACT

Touch Healing (TH) therapies, defined here as treatments whose primary route of administration is tactile contact and/or active guiding of somatic attention, are ubiquitous across cultures. Despite increasing integration of TH into mainstream medicine through therapies such as Reiki, Therapeutic Touch,TM and somatically focused meditation practices such as Mindfulness-Based Stress Reduction, relatively little is known about potential underlying mechanisms. Here, we present a neuroscientific explanation for the prevalence and effectiveness of TH therapies for relieving chronic pain. We begin with a cross-cultural review of several different types of TH treatments and identify common characteristics, including: light tactile contact and/or a somatosensory attention directed toward the body, a behaviorally relevant context, a relaxed context and repeated treatment sessions. These cardinal features are also key elements of established mechanisms of neural plasticity in somatosensory cortical maps, suggesting that sensory reorganization is a mechanism for the healing observed. Consideration of the potential health benefits of meditation practice specifically suggests that these practices provide training in the regulation of neural and perceptual dynamics that provide ongoing resistance to the development of maladaptive somatic representations. This model provides several direct predictions for investigating ways that TH may induce cortical plasticity and dynamics in pain remediation.

A BRIEF REVIEW OF TOUCH HEALING

Touch Healing (TH) therapy, broadly defined here as medical treatments whose primary route of administration is tactile contact and/or the active guiding of somatic attention, have been observed in contexts ranging from North American Pentecostal and Charismatic Christian rituals (the “laying on of hands” or “anointing of the sick”)^{1,2} to *qigong* and similar therapies in China and throughout East Asia.^{3,4} The treatments can include Light Touch, brushing, tapping, near touch, or self-directed somatosensory attention.^{5,6} Practices associated with TH figure prominently in the recent rise of alternative and complementary health practices in the United States and throughout the developed world.^{7–9} In this first section, we identify common characteristics of TH therapies as an in-

roduction to discussing how these common features predict a somatosensory plasticity and perceptual learning mechanism in TH.

Many TH therapies are used in the American health care system, with practitioners dispensing treatments in such modalities as Therapeutic Touch (TT), Healing Touch, Reiki, Polarity Therapy, Mindfulness-Based Stress Reduction (MBSR), and *qigong*.^{7,10} One example is TT, a TH modality developed in the early 1970s by D. Kunz and D. Krieger, that is currently used by tens of thousands of nurses in the United States.¹¹ The practitioner typically begins by eliciting a calm “centered” attitude in the patient. He/she then sweeps his/her hands at a distance of 1–2 inches from the body and tells the patient he/she is receiving a powerful energetic touch. Even though the practitioners’ hands are kept at a distance, patients frequently describe a “flowing

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feeling” near where they envision the practitioner’s hands to be. Another TH therapy, Reiki, parallels TT in many respects.^{12,13}

A number of East and South Asian meditative self-healing practices, including forms of Taoist and Buddhist meditation, share similar characteristics with the TH therapies described above. One example is MBSR, a self-healing practice that originates in the Buddhist practice of mindfulness.¹⁴ This treatment is commonly prescribed for pain and anxiety.¹⁵ Practitioners use meditative procedures to enter a calm state and then follow a protocol of directing focused, mindful awareness to specific areas of the body, and to breathing-related sensations. Practitioners’ self-reports of bodily feelings as spontaneously arising and changing during practice echo patient descriptions of the spontaneous bodily feeling of “flow” commonly experienced during TT and Reiki.¹⁶

Multiple ethnographic accounts^{2,17,18} describe a similar set of factors active in ritual healing. For example, in a Thai ritual healing ceremony (described in Tambiah¹⁹), the healer first fosters a relaxed meditative calm in his patients. He or one of his assistants then lightly touches each member of the audience. In the healer’s touch, patients report feeling a surging somatosensory sensation that they associate with healing deities said to be evoked by the healer.

This brief overview of TH across cultures suggests several general traits: (1) Light Touch or “implied touch” is engaged; (2) Somatosensory attention is modulated; (3) Stimuli are administered in a behaviorally relevant context, in which the patient anticipates the possibility of healing; and (4) Treatment is delivered to a relaxed recipient. Using taxonomic methods described by Kaptchuk and Eisenberg,²⁰ we have identified three classes of therapies that, to differing extents, demonstrate the cardinal features of TH described above. In Class 1, *Light Touch* is delivered in a light or superficial manner with the goal of evoking an internal somatosensory response from patients. Class 2, *guided somatosensory attention*, includes meditative practices in which patient-practitioners use a self-healing meditative protocol to modulate their own somatosensory attention. In Class 3, *ritual healing*, the healer administers a touch stimulus with an instruction that imbues the touch with a specific ritual meaning that has high behavioral relevance to the patient (e.g., the touch of a healing “spirit” or presence).

Of the three forms of TH, Class 1 (*Light Touch*) therapies are the most easily characterized and least variable across healing modalities. Class 2 (*guided somatosensory attention*) meditative practices vary greatly, though forms of attentional guidance and relaxation, such as MBSR, have been systematized and studied in a scientific context.¹⁶ Because Class 1 therapies and specific exemplars of Class 2 therapy are more highly standardized and assimilated into current clinical practice in North America, this paper will focus primarily on these practices. By contrast, Class 3 therapies are more varied in setting, presentation, and clinical uses. However, although the remainder of our review does

not specifically address Class 3 TH treatments, we believe the processes described here should be evaluated further to determine whether they underlie any therapeutic changes seen in these forms of ritual healing.

EFFICACY OF TH THERAPIES

The efficacy of Class 1 (*Light Touch*) forms of healer-administered TH has been studied in clinical trials for a range of disorders centered around pain and anxiety states,²¹ headache,²² osteoarthritis,²³ carpal tunnel syndrome,²⁴ pain in patients with cancer,²⁵ agitation in Alzheimer patients,^{26,27} anxiety in patients receiving complex medical procedures,²⁸ and behavioral reactions in low-birth-weight babies.²⁹ Overall, these studies offer evidence for the efficacy of Class 1 therapies, although many studies are weakened by low sample sizes and poor study design.^{7,10,11} Two recent well-controlled studies of TH for defined pain conditions^{23,24} found that both “sham” and “real” Class 1 TH elicited significant pain relief. As we explain below, clinically meaningful improvement in both sham and real Class 1 TH can be explained by the neuroscientific mechanisms described in this paper. Specifically, “sham” as applied in these studies, should induce similar cortical plasticity. Evidence for the efficacy of Class 2 (*guided somatosensory attention*) can also be seen in observational and clinical trials performed in a range of pain conditions.^{15,30–33}

CORTICAL PLASTICITY IN CHRONIC PAIN

A prevalent form of chronic pain is centrally maintained chronic pain, in which pain continues despite the absence of active local tissue damage. Although centrally maintained chronic pain is not well-understood, it is implicated in common conditions such as low-back pain³⁴ and fibromyalgia.^{35,36} There is broad agreement that centrally maintained pain is associated with some form of cortical dysregulation^{37,38} and is especially difficult to treat.³⁹

Several brain areas in humans and other mammals contain “maps” of the body surface,⁴⁰ and alterations in the functionality of these maps have been implicated in the etiology of chronic pain.³⁴ The best-studied body map, the primary somatosensory cortex (SI), contains a representation popularly referred to as the “homunculus,” in which contiguous areas of the cortical sheet typically receive peripheral input from adjacent body zones.⁴¹ Studies in adult humans and primates show that somatosensory maps can change or remodel throughout adulthood.^{42–46} For example, somatotopic hand maps of musicians and Braille readers have specific differences from those of normal controls.^{43,47,48}

Cortical plasticity of this type is correlated with, and may be a causal mechanism in, centrally maintained chronic

pain.^{49–54} Several features characterize alterations in SI representations correlated with chronic pain. The zone of the SI body map that represents the painful peripheral region has an enlarged area of activation. This region also shows map fragmentation, where noncontiguous areas of the body surface come to be represented in adjacent parts of the map. Further, a shift in the relative amount of neural activity evoked by a constant amplitude stimulus, a change in the “gain” of neural responsiveness, can occur.^{49–51,54,55} These abnormalities are correlated with pain intensity. The converse has also been observed—when chronic pain intensity subsides, the somatosensory cortical body map becomes more like that of healthy controls.⁵⁶ In animal models, conditions that promote plasticity can also induce pain. In a chronic arm pain/repetitive strain injury paradigm, monkeys subjected to postural strain were found to have abnormal cortical maps of the wrist and hands and disordered somatotopic representation of individual fingers.⁴⁹

TOUCH HEALING AND CORTICAL PLASTICITY: A POTENTIAL THERAPEUTIC MECHANISM

The strong link between maladaptive map organization, map plasticity, and chronic pain suggests that the benefit of TH may occur by inducing beneficial reorganization of body maps (which we refer to as “therapeutic plasticity”). Our hypothesis is that TH modalities work to renormalize somatotopic maps. This prediction is supported by the observation that factors facilitating map reorganization are also cardinal features of TH treatments, and that these cardinal features act in opposition to the conditions that initiate and maintain chronic pain. As such, TH treatment can be seen as an optimal method for inducing changes in cortical map representation of the painful body part. We begin with a brief overview of the “cardinal features” of chronic central pain, cortical somatosensory plasticity, and Healing Touch.

Cardinal factors in persistent chronic pain

Repeated sensory input. Repetitive motion, postural strain, and peripheral lesions are common driving factors in the initiation of chronic pain.^{49,57–59} With cumulative exposure, these somatic inputs cause changes in central neural structures involved in pain perception, creating a lasting “pain memory” of the associated sensory inputs.^{34,59}

Somatosensory attentional modulation. Pain draws patients’ attention preferentially toward the pain experience⁶⁰ and imposes a constant cognitive load that interferes with the performance of other tasks⁶¹ and with the perception of alternative stimuli.⁶²

Behavioral relevance. Painful stimuli are (by definition) behaviorally relevant and salient. As sensory inputs other

than the pain decrease in their salience, patients develop the expectation that the pain will continue unabated in the future and disrupt their ability to function.⁶³ In clinical trials, the expectation of future pain predicts poor outcome.⁶⁴

Stress. Physiologic and psychologic stress are a *sine qua non* of the pain experience. For example, back pain and upper extremity pain are associated with chronic psychologic workplace stress.^{65,66} Physiologically, chronic pain has been demonstrated to increase the release of stress hormones such as glucocorticoids.⁶⁷

Cardinal factors in the optimal induction of cortical plasticity

Repeated sensory input. The induction of SI plasticity depends in large part on repeated and selective contact with a body region. In monkeys, for example, tactile stimuli administered over a period of weeks to the finger produce significant long-term reorganization of the finger region of the SI map.^{68,69} Similar observations have been made in rodent SI after the selective trimming of all but one or two of the facial whiskers,^{70,71} and in humans after repeated stimulation of the fingertips.⁴²

Somatosensory attentional modulation. Modulation of somatosensory attention contributes significantly to long-term plastic remodeling in sensory cortical maps. Systematic practice in shifting attention enhances performance on perceptual tasks.⁷² In humans and monkeys, shifts in the focus of attention between different peripheral stimuli can also reorganize primary sensory cortical maps on millisecond to second time scales.^{73,74}

Behavioral relevance. The context and expectation associated with a stimulus are central to its efficacy in changing the somatosensory cortical body map. In primate studies, if an animal receives two identical competing stimuli, greater map change is evoked by the stimulus that is more behaviorally relevant (e.g., the signal that the monkey expects will be accompanied by a reward).^{69,75,76} This favored plasticity context is likely facilitated by the release of neuromodulators such as acetylcholine.^{77–79}

Stress. High levels of physiologic stress, or the presence of high concentrations of stress-related hormones, can block long-term potentiation, a hallmark of central nervous system plasticity.⁸⁰ The association between stress and chronic pain, in settings where chronic pain emerges from behavioral maladaptive processes, suggests that stress may play a physiologic role in preventing map renormalization, thereby “locking in” maladaptive plasticity. Pain experienced in the midst of chronic stress may be imprinted on the central nervous system as a long-standing, maladaptive alteration in central processing.^{81,82} The converse is also true: relaxation

facilitates perceptual learning⁸³ and cortical remodeling processes.⁸⁴

Cardinal factors of TH treatment

The TH cortical plasticity model described here is related to Class 1 TH therapies such as TT and Reiki. It may also explain the prevalent use of Class 2 meditative practices such as MBSR as therapies for chronic pain.^{31,32}

Class 1 (Light Touch) and repeated sensory input. Class 1 TH therapies typically include repeated administration of real or implicit tactile contact applied to specific skin regions. Tactile stimuli are presented in body regions remote from the overrepresented, painful part. Such contact is usually administered with Light Touch, and tactile stimuli are repeated within a session and across multiple days. Such repeated activation of body representations through peripheral input and/or internally generated tactile imagery^{85,86} may induce therapeutic plasticity.

Class 1 (Light Touch) and somatosensory attentional modulation. Many Class 1 TH modalities described above use an “implied touch” stimulus, in which the practitioner places his/her hand near, but not directly on, the body, as some aspect of their treatment. Although some have questioned the efficacy and legitimacy of such “implied touch” procedures,^{8,87} our TH cortical plasticity model suggests that “implied touch” stimuli may serve the function of guiding subjects’ somatosensory attention, and this process may engage at least a subset of the transformative mechanisms that contribute to tactile-induced plasticity. In the case of chronic pain, the use of an “implied touch” stimulus to guide somatosensory attention away from the chronic pain site may increase treatment efficacy.

Class 2 (MBSR) and somatosensory attentional modulation. The cardinal difference between Class 1 and Class 2 therapies is that, in the case of the latter, the somatosensory attentional practices used in many meditative traditions such as mindfulness meditation or *qigong* do not involve an actual TH stimulus. Instead, Class 2 therapies typically teach a daily self-healing regimen that involves systematic shifting of somatosensory attention across the body. In MBSR, practitioners are taught, iteratively, to cultivate meditative awareness of bodily sensations and specific body processes (e.g., breathing). This practice of MBSR’s focused awareness of bodily sensations follows directly from early forms of Buddhist mindfulness meditation (e.g., the Body Contemplation practices described in the Four Foundations of Mindfulness⁸⁸). By repeatedly practicing somatosensory attentional focus, we hypothesize that practitioners learn to more optimally control cortical, and more generally, neural dynamics, allowing them to regulate the throughput of information in these representations. This

learned control may provide a self-regulatory mechanism for preventing the formation of, and/or renormalizing maladaptive cortical maps that emerge in chronic pain. Learning control of attention could allow practitioners to better “gate” sensory signals in contexts that might lead to a negative alteration of the map, such as repetitive and possibly painful behaviors. Although it is an open question as to how and whether shifts in focused somatosensory attention to distinct body regions can serve as a surrogate for actual tactile contact, and how control over these dynamics may gate sensory information, preliminary findings that short-term attentional shifts effect changes in cortical plasticity^{73,74} suggest that attentional shifts could initiate changes in cortical dynamics that bring about long-term remodeling in cortical maps.

Class 1 (Light Touch) and behavioral relevance. Class 1 practitioners frequently claim that touch stimuli are imbued with a potentiating “energy” or vibrational power. Healers and recipients within this community also frequently hold that the positive expectancy elicited by a belief in a therapy’s healing power contributes to positive outcomes.⁸⁹ According to our TH cortical plasticity model, patients who believe such claims may endow TH with greater behavioral relevance (e.g., through enhanced neuromodulator release). This increased behavioral relevance may facilitate therapeutic cortical remodeling of body maps and, through this mechanism, elicit pain relief.

Class 2 (MBSR) and behavioral relevance. Like the other TH examples described above, MBSR provides practitioners with a nonspecific positive therapeutic expectancy. In addition, by increasing the relevance of neutral sensations (e.g., sensations related to breathing), MBSR may facilitate a behavioral context in which negative chronic pain-related sensations are accorded decreased salience. As this decreased salience is repeatedly reinforced through practice, mindful patients report being able to disengage from negative schemas that may be actively contributing to chronic pain.^{14,90}

Class 1 (Light Touch) and relaxation. Class 1 therapies are nearly always described as relaxing and often include an explicit relaxation instruction prior to onset. Patients are typically placed supine, in a quiet room, and asked to lie in as calm and relaxed a state as possible. According to our proposed framework, relaxation may be critical to the efficacy of TH therapies because it may relieve the blockade that psychologic stress and stress hormones create against plasticity.^{13,91,92}

Class 2 (MBSR) and relaxation. MBSR uses basic Buddhist philosophic principles that prescribe a “calm mind” as a necessary precondition for attaining benefit from meditation.⁹³ The stress reduction training that is an essential com-

ponent of this practice may also be a form of training in the self-regulation of plasticity, allowing subjects to persistently maintain “healthy” map structure.

The alignment of optimal cortical remodeling of somatotopic maps with factors common to Class 1 and Class 2 TH therapies suggests a mechanism by which TH therapies elicit relief of chronic pain. Below we consider the broader significance of our therapeutic plasticity model and its relevance for further research in TH and other complementary and alternative medicine modalities.

APPLICATIONS OF THE MODEL: AVENUES FOR FUTURE RESEARCH

This model provides a framework for investigating ways that TH might induce cortical plasticity and pain remediation. We propose that TH optimizes cortical plasticity by enhancing and guiding attentional focus to nonpainful representations, reducing stress and providing high behavioral relevance in the context of repeated tactile stimulation. Further, these practices may provide training in the self-regulation of neural dynamics and perception, helping to prevent the establishment of maladaptive patterns. Future investigations will allow explicit tests of whether varying specific components of the model produces hypothesized differences in outcome.

The model also explains randomized trial outcomes in which both sham and real Class 1 TH appeared to provide meaningful pain relief.^{23,24} Sham TH that does not control for the potentially positive effects of somatosensory attentional modulation, or a behaviorally relevant context, may elicit “active” processes that engage mechanisms that are operative in real TH. Our model thus suggests the importance in future trials of rigorously controlling for factors (e.g., behavioral relevance and somatosensory attention) traditionally regarded as epiphenomenal.

Additionally, although current research is limited, several studies suggest that this model may also be applicable, in a modified form, to emotional disorders. Classic accounts of emotional psychology (e.g., the James-Lange theory) suggest that emotions are rooted in the interoceptive perception of ongoing body states.^{94,95} Building on this earlier theory, recent studies have found that somatosensory cortical body maps may be an important component of the neural architecture of emotional processing.⁹⁶ Use of TH treatments such as Reiki or TT is increasing for affective disorders,^{7,13,97} whereas recent randomized controlled trials support the use of a Class 2 mindfulness-based therapy for prevention of depression relapse.^{98,99} Taken together with the prevalence of comorbidity of depression with chronic pain,^{100,101} these studies suggest that further investigation of emotional processing as it relates to our model of therapeutic somatosensory cortical plasticity is warranted.

CONCLUSION

Our model has the potential to bring order to what has previously been a poorly defined group of therapies. Using a theory-driven taxonomy, we describe characteristics of TH therapies common to a range of modalities across cultures. These shared characteristics, which include repeated sensory input, somatosensory attentional modulation, behavioral relevance, and relaxation, suggest a single class of mechanisms may underlie the diverse modes of TH.

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REFERENCES

1. Simpson M, King M. “God brought all these churches together”: Issues in developing religion-health partnerships in an Appalachian community. *Public Health Nurs* 1999; 16:41–49.
2. Csordas T. *Sacred Self: A Cultural Phenomenology of Charismatic Healing*. Berkeley: University of California, 1994.
3. Chang S. The nature of touch therapy related to Ki: Practitioners’ perspective. *Nurs Health Sci* 2003;5:103–114.
4. Ots T. The silenced body—the expressive leib. In: Csordas T, ed. *Embodiment and Experience: The Existential Ground of Culture and Self*, Cambridge: Cambridge University Press, 1994:161–132.
5. Wendle M. Effects of Tellington Touch in adults awaiting venipuncture. *Res Nurs Health* 2003;26:40–52.
6. Adams G. Shiatsu in Britain and Japan: personhood, holism and embodied aesthetics. *Anthropol Med* 2002;9:245–265.
7. Astin J, Harkness E, Ernst E. The efficacy of “distant healing”: a systematic review of randomized trials. *Ann Intern Med* 2000;132:903–910.
8. Rosa L, Rosa E, Sarner L, Barrett S. A close look at therapeutic touch. *JAMA* 1998;279:1005–1010.
9. Shumay D, Maskarinec G, Gotay CC, Heiby EM, Kakai H. Determinants of the degree of complementary and alternative medicine use among patients with cancer. *J Altern Complement Med* 2002;8:661–671.
10. Meehan T. Therapeutic Touch as a nursing intervention. *J Adv Nurs* 1998;28:117–125.
11. Peters R. The effectiveness of therapeutic touch: a meta-analytic review. *Nurs Sci Q* 1999;12:52–61.
12. Engebretson J, Wardell DW. Experience of a Reiki session. *Altern Ther Health Med* 2002;8:48–53.
13. Miles P, True G, Reiki—review of a biofield therapy history, therapy, practice, and research. *Altern Ther Health Med* 2003;9:62–72.

14. Kabat-Zinn J. *Full Catastrophe Living*. New York: Delta Publishing, 1990.
15. Baer RA. Mindfulness training as a clinical intervention: A conceptual and empirical review. *Clin Psychol Sci Practice* 2003;10:125–143.
16. Cohen M. Healing at the borderland of medicine and religion: regulating potential abuse of authority by spiritual healers. *J Law Religion* 2004;18:373–426.
17. Adams JD, Garcia C. Palliative care among Chumash people. *eCAM* 2005;2:143–147.
18. Warber S, Cornelio D, Straughn J, Kile G. Biofield energy healing from the inside. *J Altern Complement Med* 2004;10:1107–1113.
19. Tambiah SJ. *A Thai Cult of Healing through Meditation, in Culture, Thought, and Social Action*. 1985, Harvard University Press: Cambridge, 87–122.
20. Kaptchuk T, Eisenberg D. Varieties of healing. 2: a taxonomy of unconventional healing practices. *Ann Intern Med* 2001;135:196–204.
21. Denison B. Touch the pain away: new research on therapeutic touch and persons with fibromyalgia syndrome. *Holist Nurs Pract* 2004;18:142–151.
22. Keller E, Bzdek VM. Effects of therapeutic touch on tension headache pain. *Nurs Res* 1986;35:101–106.
23. Gordon A, Merenstein JH, D'Amico F, Hudgens D. The effects of therapeutic touch on patients with osteoarthritis of the knee. *J Fam Pract* 1998;47:271–277.
24. Blankfield R, Sulzmann C, Fradley LG, et al. Therapeutic Touch in the treatment of carpal tunnel syndrome. *J Am Board Fam Pract* 2001;14:335–342.
25. Olson K, Hanson J, Michaud M. A phase II trial of Reiki for the management of pain in advanced cancer patients. *J Pain Symptom Manage* 2003;26:990–997.
26. Woods D, Dimond M. The effect of therapeutic touch on agitated behavior and cortisol in persons with Alzheimer's disease. *Biol Res Nurs* 2002;4:104–114.
27. Woods D, Craven RF, Whitney J. The effect of therapeutic touch on behavioral symptoms of persons with dementia. *Altern Ther Health Med* 2005;11:66–74.
28. Smith M, Reeder F, Daniel L, et al. Outcomes of touch therapies during bone marrow transplant. *Altern Ther Health Med* 2003;9:40–49.
29. de Roiste A. TAC-TIC therapy with premature infants: A series of investigative studies. *Neuro Endocrinol Lett* 2004;25(suppl 1):67–77.
30. Kabat-Zinn J, Lipworth L, Burney R. The clinical use of mindfulness meditation for the self-regulation of chronic pain. *J Behav Med* 1985;8:163–190.
31. Kabat-Zinn J. An outpatient program in behavioral medicine for chronic pain patients based on the practice of mindfulness meditation: theoretical considerations and preliminary results. *Gen Hosp Psychiatry* 1982;4:33–47.
32. Kabat-Zinn J, et al. Four-year follow-up of a meditation-based program for the self-regulation of chronic pain: Treatment outcomes and compliance. *Clin J Pain* 1987;2:159–173.
33. Randolph P, Caldera YM, Tacone AM, Greak ML. The long-term combined effects of medical treatment and a mindfulness-based behavioral program for the multidisciplinary management of chronic pain in west Texas. *Pain Digest* 1999;9:103–112.
34. Flor H. Cortical reorganization and chronic pain: Implications of rehabilitation. *J Rehabil Med* 2003;41(suppl):66–72.
35. Clauw D, Crofford LJ. Chronic widespread pain and fibromyalgia: What we know, and what we need to know. *Best Pract Res Clin Rheumatol* 2003;17:685–701.
36. Staud R, Vierck CJ, Robinson ME, Price DD. Spatial summation of heat pain within and across dermatomes in fibromyalgia. *Pain* 2004;111:342–350.
37. Treede R, Kenshalo DR, Gracely RH, Jones AK. The cortical representation of pain. *Pain* 1999;79:105–111.
38. Price D. Central neural mechanisms that interrelate sensory and affective dimensions of pain. *Mol Interventions* 2002;2:392–403.
39. Clauw D, Williams DA. Relationship between stress and pain in work-related upper extremity disorders: the hidden role of chronic multisymptom illnesses. *Am J Indust Med* 2002;41:370–382.
40. Penfield W, Boldrey E. Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. *Brain* 1937;60:389–443.
41. Kaas J. What, if anything, is SI? Organization of first somatosensory area of cortex. *Physiol Rev* 1983;63:206–231.
42. Hodzic A, Veit R, Karim AA, et al. Improvement and decline in tactile discrimination behavior after cortical plasticity induced by passive tactile coactivation. *J Neurosci* 2004;24:442–446.
43. Pascual-Leone A, Torres F. Plasticity of the sensorimotor cortex representation of the reading finger in Braille readers. *Brain* 1993;116:39–52.
44. Wall J, Kass JH, Sur M, et al. Functional reorganization in somatosensory cortical areas 3b and 1 of adult monkeys after median nerve repair: possible relationships to sensory recovery in humans. *J Neurosci* 1986;6:218–233.
45. Merzenich M, Nelson RJ, Stryker MP, et al. Somatosensory cortical map changes following digit amputation in adult monkeys. *J Compar Neurol* 1984;224:591–605.
46. Merzenich M, Kaas JH, Wall J, et al. Topographic reorganization of somatosensory cortical areas 3b and 1 in adult monkeys following restricted deafferentation. *Neuroscience* 1983;8:33–55.
47. Sterr A, Muller MM, Elbert T, et al. Perceptual correlates of changes in cortical representation of fingers in blind multi-finger Braille readers. *J Neurosci* 1998;18:4417–4423.
48. Elbert T, Pantev C, Weinbruch C, et al. Increased cortical representation of the fingers of the left hand in string players. *Science* 1995;270:305–307.
49. Byl N, Merzenich MM, Jenkins WM. A primate genesis model of focal dystonia and repetitive strain injury: Learning-induced dedifferentiation of the representation of the hand in the primary somatosensory cortex in adult monkeys. *Neurology* 1996;47:508–520.
50. Flor H, Braun C, Elbert T, et al. Extensive reorganization of primary somatosensory cortex in chronic back pain patients. *Neurosci Lett* 1997;224:5–8.
51. Flor H, Elbert T, Knecht S, et al. Phantom-limb pain as a perceptual correlate of cortical reorganization following arm amputation. *Nature* 1995;375:482–484.
52. Flor H, Knost B, Birbaumer N. Processing of pain and body-related verbal material in chronic pain patients: Central and peripheral correlates. *Pain* 1997;73:413–421.

53. Maihofner C, Handwerker HO, Neundorfer B, Birklein F. Cortical reorganization during recovery from complex regional pain syndrome. *Neurology* 2004;63:696–701.
54. Maihofner C, Handwerker HO, Neundorfer B, Birklein F. Patterns of cortical reorganization in complex regional pain syndrome. *Neurology* 2003;61:1707–1715.
55. Lotze M, Flor H, Wolfgang G, et al. Phantom movements and pain: An fMRI study in upper limb amputees. *Brain* 2001;124(Part 11):2268–2277.
56. Flor H, Denke C, Schafer M, et al. Effect of sensory discrimination training on phantom limb pain. *Lancet* 2001;357:1763–1764.
57. Evcik D, Yucel A. Lumbar lordosis in acute and chronic low back pain patients. *Rheumatol Int* 2003;23:163–165.
58. Greening J, Lynn B. Vibration sense in the upper limb in patients with repetitive strain injury and a group of at-risk office workers. *Int Arch Int Envir Health* 1998;71:29–34.
59. Melzack R,Coderre TJ, Katz J, Vaccarino AL. Central neuroplasticity and pathological pain. *Ann NY Acad Sci* 2001;933:157–174.
60. Eccleston C. Chronic pain and distraction: An experimental investigation into the role of sustained and shifting attention in the processing of chronic persistent pain. *Behav Res Ther* 1995;33:391–405.
61. Dehghani M, Sharpe L, Nicholas MK. Modification of attentional biases in chronic pain patients: A preliminary study. *Eur J Pain* 2004;8:585–594.
62. Moriwaki K, Yuge O. Topographic features of cutaneous tactile hypoesthetic and hyperesthetic abnormalities in chronic pain. *Pain* 1999;81:1–6.
63. Lackner J, Carosella A. The relative influence of perceived pain control, anxiety, and functional self-efficacy on spinal function among patients with chronic low back pain. *Spine* 1999;24:2254–2260.
64. Jensen M, Ehde D. Cognitions, coping and social environment predict adjustment to phantom limb pain. *Pain* 2002;95:133–142.
65. Soucy I, Truchon M, Cote D. Work-related factors contributing to chronic disability in low back pain. *Work* 2006;26:313–326.
66. Buckle P. Upper limb disorders and work: The importance of psychosocial factors. *J Psychosom Res* 1997;43:17–25.
67. Blackburn-Munro G, Blackburn-Munro RE. Chronic pain, chronic stress and depression: Coincidence or consequence? *J Neuroendocrinol* 2001;13:1009–1023.
68. Jenkins WM, Merzenich M, Ochs M, et al. Functional reorganization of primary somatosensory cortex in adult owl monkeys after behaviorally controlled tactile stimulation. *J Neurophysiol* 1990;63:82–104.
69. Recanzone G, Merzenich MM, Jenkins WM, et al. Topographic reorganization of the hand representation in cortical area 3b owl monkeys trained in a frequency discrimination task. *J Neurophysiol* 1992;67:1031–1056.
70. Armstrong-James M, Diamond ME, Ebner FF. An innocuous bias in whisker use in adult rats modifies receptive fields of barrel cortex neurons. *J Neurosci* 1994;14(11 Pt 2):6978–6991.
71. Polley DB, Chen-Bee CH, Frostig RD. Two directions of plasticity in the sensory-deprived adult cortex. *Neuron* 1999;24:623–637.
72. Fahle M. Perceptual learning: a case for early selection. *J Vision* 2004;26:879–890.
73. Buchner H, Reinartz U, Waberski TD, et al. Sustained attention modulates the immediate effect of de-afferentiation on the cortical representation of the digits: source localization of somatosensory evoked potentials in humans. *Neurosci Lett* 1999;260:57–60.
74. Noppeney U, Waberski TD, Gobelé R, et al. Spatial attention modulates the cortical somatosensory representation of the digits in humans. *Neuroreport* 1999;10:3137–3141.
75. Merzenich M, de Charms RC. Neural Representations, Experience, and Change. In: Churchland LR, ed. *Mind-Brain Continuum: Sensory Processes*. Cambridge, Massachusetts: MIT Press, 1998.
76. Staines W, Graham SJ, Black SE, McIlroy WE. Task-relevant modulation of contralateral and ipsilateral primary somatosensory cortex and the role of a prefrontal-cortical sensory gating system. *Neuroimage* 2002;15:190–199.
77. Bao S, Chan VT, Merzenich MM. Cortical remodeling induced by activity of ventral tegmental dopamine neurons. *Nature* 2001;412:79–83.
78. Bjordahl T, Dimyam M, Weinberger N. Induction of long-term receptive field plasticity in the auditory cortex of the waking guinea pig by stimulation of the nucleus basalis. *Behav Neurosci* 1998;1998:467–479.
79. Kilgard M, Merzenich M. Cortical map reorganization enabled by nucleus basalis activity. *Science* 1998;279:1714–1718.
80. Maroun M, Richter-Levin G. Exposure to acute stress blocks the induction of long-term potentiation of the amygdala-prefrontal cortex pathway in vivo. *J Neurosci* 2003;23:4406–4409.
81. Flor H. Functional organization of the brain in chronic pain. *Progr Brain Res* 2000;129:313–322.
82. Flor H. Can we train chronic pain patients to “forget” their pain. *EMBO Rep* 2002;3:288–291.
83. Facchini S, Aglioti SM. Short term light deprivation increases tactile spatial acuity in humans. *Neurology* 2003;60:1998–1999.
84. Gottselig J, Hofer-Tinguely G, Borbely AA, et al. Sleep and rest facilitate auditory learning. *Neuroscience* 2004;127:557–561.
85. Porro C, Francescato MP, Cettolo V, et al. Primary motor and sensory cortex activation during motor performance and motor imagery: A functional magnetic resonance imaging study. *J Neurosci* 1996;16:7688–7698.
86. Yoo S, Freeman DK, McCarthy JJ 3rd, Jolesz FA. Neural substrates of tactile imagery: a functional MRI study. *Neuroreport* 2003;14:581–585.
87. O’Mathuna D, Prymachuk S, Spencer W, et al. A critical evaluation of the theory and practice of therapeutic touch. *Nurs Philosophy* 2002;3:163–176.
88. Silananda U. *The Four Foundations of Mindfulness*. Boston: Wisdom Publications, 1990.
89. Kaptchuk T. The placebo effect in alternative medicine: Can the performance of a healing ritual have clinical significance? *Ann Intern Med* 2002;136:817–825.
90. Mason O, Hargreaves I. A qualitative study of mindfulness-based cognitive therapy for depression. *Br J Med Psychol* 2001;74(pt 2):197–212.

91. McEwen B. Protection and damage from acute and chronic stress: Allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Ann NY Acad Sci* 2004;1032:1–7.
92. McEwen B. Corticosteroids and hippocampal plasticity. *Ann NY Acad Sci* 1994;746:132–142.
93. Hanh T. *Transformation and Healing: The Sutra on the Four Establishments of Mindfulness*. Berkeley: Parallax Press, 1990.
94. Morris J. How do you feel? *Trends Cogn Sci* 2002;6:317–319.
95. James W. What is an emotion. *Mind* 1884;9:188–205.
96. Damasio AR. The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Phil Trans R Soc Lond* 1996;351:1413–1420.
97. Geccedi R, Decker G. Incorporating alternative therapies into pain management: More patients are considering complementary approaches. *Am J Nurs* 2001;101:(suppl):35–39, 49–50.
98. Teasdale J, Segal ZV, Williams JMG, et al. Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *J Consult Clin Psychol* 2000;68:615–623.
99. Ma S, Teasdale J. Mindfulness-based cognitive therapy for depression: Replication and exploration of differential relapse prevention effects. *J Consult Clin Psychol* 2004;72:31–40.
100. Bair M, Robinson RL, Katon W, Kroenke K. Depression and pain comorbidity: A literature review. *Arch Intern Med* 2003;163:2433–2445.
101. Schatzberg A. The relationship of chronic pain and depression. *J Clin Psychiatry* 2004;65:3–4.

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