

Loving-Kindness Meditation Findings Not Related to Baseline Differences

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Recently, this journal included a report on the positive effects of a loving-kindness meditation intervention for persistent low back pain patients (Carson et al., 2005), along with commentary on this article (Bowman, 2005). Although Bowman's comments were mostly complimentary of the merits of this pilot study, I believe it is important to clarify several key issues raised by the commentary, to wit: (a) Differences between the treatment and control group outcomes could be attributable to baseline between-group differences in anger variables, (b) the etiology of back pain among treatment responders possibly was psychological rather than physical, and (c) a shift from positive to negative emotions may reduce pain that results from psychological trauma but may not affect pain that is due to physical trauma.

First, with regard to the effect of baseline differences in anger variables (i.e., the intervention group was lower on Trait Anger and Anger Expression Out, and higher on Anger Control In and Anger Control Out), as stated in our article, we did perform post-hoc analyses to determine whether improvements in the intervention group might be partially attributable to baseline differences in these variables. For the sake of brevity, we did not include these results in our findings. However, the analyses and results were as follows: We reasoned that if the intervention was differentially effective for patients depending on baseline anger differences, then change scores in pain and psychological distress variables should be correlated with such baseline anger differences—that is, greater improvements should be associated with either less Trait Anger, less Anger Expression Out, more Anger Control In, or more Anger Control Out at baseline. We therefore conducted bivariate correlational analyses (Pearson's r) between change scores (calculated such that higher scores indicated greater improvements) on outcomes that were significantly improved at posttest in the intervention group (i.e., Pain Intensity, Usual Pain, Overall Psychological Distress, and Anxiety; note that no significant changes were observed in the

control group) and baseline scores on these anger variables. Results suggested that baseline Trait Anger and Anger Expression Out scores were unrelated to participants' improvements. Moreover, Anger Control In at baseline was negatively correlated with several improvements. Higher baseline Anger Control In was actually associated with less rather than more improvement in Pain Intensity ($p \leq .05$) and Anxiety ($p \leq .01$). Similarly, Anger Control Out at baseline was negatively correlated with improvement in Pain Intensity ($p \leq .01$). Thus, rather than more baseline control over anger being associated with greater improvement, the reverse was the case—patients who had greater baseline anger control were likely to show less improvement in pain or psychological distress after the intervention. So it is highly unlikely that baseline differences in anger scores can account for the differential results we obtained in the intervention versus control group.

Second, with regard to whether the etiology of back pain among treatment responders was psychological rather than physical, this was not the case. Nearly all the treatment responders had undergone clearly identified physical trauma events leading to back pain, such as industrial accidents, highway accidents, and so on.

Last, with respect to whether a positive emotional shift may affect pain that comes from psychological trauma but is unlikely to decrease pain resulting from physical trauma, this postulate runs contrary to current evidence-based theories of pain, such as the gate control and neuromatrix models (Melzack, 1991). A good deal of data has been amassed to support these theories, which hold that pain perception always integrates a complex set of variables, including substantive input from affective and cognitive factors—regardless of the extent or absence of physical trauma. Research has demonstrated, for instance, that induction of negative affect consistently and significantly decreases tolerance for laboratory pain stimuli, whereas the induction of positive affect increases such tolerance (Zelman, Howland, Nichols, & Cleeland, 1991).

References

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