Commentary

Psychological Science
1–3

DOI: 10.1177/0956797615584304
pss.sagepub.com

Evidence for the Upward Spiral Stands Steady: A Response to Heathers, Brown, Coyne, and Friedman (2015)

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Received 1/27/15; Revision accepted 4/7/15

In 2013, we reported in *Psychological Science* on a longitudinal field experiment in which we randomized participants to receive positive-emotions training (or not), in order to illuminate the pathways by which positive emotions might build physical health (Kok et al., 2013). In their Commentary, Heathers, Brown, Coyne, and Friedman (2015) claim to overturn the conclusions that we and our coauthors drew in that original report. Here, we rebut their claims and illustrate that our substantive conclusions in fact stand steady.

Conceptually, Heathers and his colleagues contend that cardiac vagal tone is not a valid proxy for physical health, and that even if it were, high-frequency heart rate variability (HF-HRV) is a flawed measure of cardiac vagal tone. It is true that scientists continue to debate the proper measurement and interpretation of cardiac vagal tone. It is also true, as we noted in our original report (pp. 1130–1131), that future research should include additional objective health-related markers to complement the findings that we reported. However, other claims by Heathers et al. are based on misrepresentations of our original report and of the extant research literature. For example, our conclusions do not hinge on the unique properties of a single measurement approach because we found support for our hypothesized model using both a frequency-domain measure of cardiac vagal tone (HF-HRV) and a time-domain measure of cardiac vagal tone (respiratory sinus arrhythmia, which entails direct measurement of respiration using pneumatic bellows that encircle the rib cage). Although we clearly conveyed this dual measurement strategy in our report (p. 1126), Heathers and his colleagues fail to mention this fact and indeed imply that we did not measure respiration (p. XXX).

Heathers et al. also imply that our posttraining assessments of HF-HRV were obtained while participants meditated, which would presumably slow their breathing. This was not the case, however. As indicated in our original report (p. 1126), baseline and posttraining measures of cardiac vagal tone were carried out under identical instructions, at rest, without constraints or suggestions regarding respiration. To test whether respiratory changes were indeed confounded with experimental treatment, and thereby might offer an alternative explanation for our reported findings, here we report the results of a repeated measures analysis of variance (ANOVA) that compared baseline and posttraining values for respiratory period (seconds per breath cycle; 3 participants were not included in the analyses because of missing respiratory data). Mean respiratory period for the experimental group was 5.17 s per breath cycle, 95% confidence interval (CI) = [4.51, 5.91], at baseline and 5.45 s per breath cycle, 95% CI = [4.80, 6.19], after the workshop. The corresponding values for the waiting-list control group were 4.97, 95% CI = [4.36, 5.66], and 5.00, 95% CI = [4.42, 5.65].[1] The ANOVA revealed that main effects of treatment condition and time were not significant, $F(1, 56) = 0.564$ and $F(1, 56) = 0.676$, respectively. The interaction of treatment condition and time was also not significant, $F(1, 56) = 0.444$. Thus, contrary to the claims of Heathers et al., changes in respiration cannot account for the observed changes in HF-HRV reported in Kok et al. (2013).

Regarding the validity of cardiac vagal tone as a health-relevant biological indicator, ample evidence across 36 studies using a variety of measurement techniques, including HF-HRV, has shown cardiac vagal tone to be associated with all-cause mortality and cardiovascular

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Table 1. Results of the Mediational Parallel-Process Latent-Growth Models Using Square-Root and Logarithmic Transformations of High-Frequency Heart Rate Variability (HF-HRV)

Path in Figure 2 of Kok et al. (2013)	Square-root transformation (Kok et al., 2013)	Logarithmic transformation[a]
Condition to PE intercept (*a*)	–0.03	–0.03
Baseline HF-HRV × Condition to PE intercept (*b*)	0.12	0.17
Baseline HF-HRV to PE intercept (*c*)	0.05	0.01
Condition to PE slope (*d*)	0.05***	0.05**
Baseline HF-HRV × Condition to PE slope (*e*)	0.04**	0.03*
Baseline HF-HRV to PE slope (*f*)	–0.01	–0.01
PE intercept to SC intercept (*g*)	1.11***	1.10***
PE slope to SC slope (*h*)	1.04***	1.03***
PE intercept to posttraining HF-HRV[b]	0.07	–0.17
SC intercept to posttraining HF-HRV (*j*)	–0.22	0.04
SC slope to posttraining HF-HRV (*k*)	4.90*	3.42*

Note: The tables presents unstandardized coefficients. The root-mean-square error of approximation was 0.078 for both models, and the comparative fit index was .95 for both models. PE = positive emotions; SC = social connections. [a]When we refit the original model with the logged transformation of HF-HRV, the first-order derivative product matrix was nonpositive definite. We resolved this problem by utilizing posttraining HF-HRV, rather than the HF-HRV residualized change score (used in Kok et al., 2013), as the final dependent variable and including an additional path from baseline HF-HRV to posttraining HF-HRV. [b]For clarity of presentation, the path from PE intercept to change in HF-HRV was not shown in Figure 2 of Kok et al. (2013) and thus does not have an alphabetic path designation.
*$p < .05$ (two-tailed). **$p < .01$ (two-tailed). ***$p < .0001$ (two-tailed).

disease (for a review, see Thayer & Lane, 2007). In prospective studies, decreased vagal function precedes the development of hypertension (Schroeder et al., 2003) and diabetes (Carnethon, Golden, Folsom, Haskell, & Liao, 2003). Thus, cardiac vagal tone is a useful index of physical health and mortality risk.

Statistically, Heathers and his colleagues make two primary claims. First, they claim that a significant main effect of experimental condition on cardiac vagal tone is required for demonstration of an upward spiral. We note here that modern mediation analyses are not predicated on tests of direct effects being significant (MacKinnon & Fairchild, 2009; Zhao, Lynch, & Chen, 2010). Indeed, a direct effect of loving-kindness meditation (LKM) on HF-HRV is not required to test our primary hypothesis (depicted in Fig. 1 in our original report), which stated that the well-known link between positive emotions and health (Pressman & Cohen, 2005) might be explained by related changes in social closeness. To test our hypothesis, we randomly assigned participants to a 6-week meditation workshop centered on LKM as a training technique, in order to experimentally manipulate positive emotions over time; such randomization is necessary for making causal assertions about subsequently observed physiological changes relative to any changes observed in the control group. As noted in the online Supplemental Material for our original report (p. 2), we observed a linear increase in positive emotions among participants in the LKM group. This result indicates that the experimental manipulation was successful.

Moreover, our complete latent-growth model (pp. 1127–1128) clearly demonstrated that change in cardiac vagal tone differed between the LKM and control groups: Experimental condition and baseline HF-HRV combined to predict the slope of change in positive emotions, which in turn predicted the slope of change in social connections, which in turn predicted change in HF-HRV over the 9-week span of the study. Our focus was to demonstrate an upward spiral through these interrelationships. Our findings allow the conclusion that, relative to participants in a waiting-list control group, those who practice LKM may increase their HF-HRV if, and only if, LKM increases their positive emotions and, in turn, their perceived positive social connections.

The second statistical claim of Heathers et al. is that a logarithmic transformation is preferable to a square-root transformation as a means to normalize the skew in the raw HF-HRV data. What these authors fail to mention is that when their preferred transformation is used to test our hypothesized model (Fig. 2 in our original report), the same pattern of significance emerges. Table 1 compares the model results with a square-root transformation (as reported in our original report, pp. 1127–1128) and the model results with a logarithmic transformation. In reanalyzing our data for this rebuttal, we also considered Heathers and his colleagues' concerns about "biologically impossible" and "implausibly high" values for HF-HRV (pp. XXX, XXX). Specifically, for each of the 6 participants whose HF-HRV values these authors called into question, we shared the corresponding raw data files

with James Long, an outside expert in the measurement and interpretation of autonomic physiological data, who was unaware of the participants' experimental condition. After a detailed examination of these data files, he concluded that data from 2 of the identified 6 participants should indeed be excluded from analyses (557004 and 557027 at posttraining). The results in the logarithmic-transformation column of Table 1 reflect the omission of these 2 participants. Regardless of whether these participants are included or not, and whether raw data are transformed by square root or by logarithm, substantively identical results emerge (cf. paths *d, e, g, h*, and *k* for the two models in Table 1).

In sum, none of the critiques offered by Heathers et al. change the fact that the data we reported in Kok et al. (2013) support the conclusion that there is an upward spiral between positive emotions and cardiac vagal tone.

Author Contributions

B. E. Kok drafted the manuscript, and B. L. Fredrickson provided critical revisions. Both authors approved the final version of the manuscript for submission.

Acknowledgments

We wish to thank Aaron Boulton, Ann Firestine, and James Long for their assistance with preparing this rebuttal. We also thank the coauthors of our original report for helpful feedback on an earlier draft.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Funding

National Institute of Mental Health Grant MH59615, awarded to B. L. Fredrickson, was used to fund data collection for the original study (Kok et al., 2013).

Note

1. Raw values were log-transformed to correct for skewness, then descriptive statistics were computed and back-transformed to the original scale.

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