COMMENT ON "NEGATIVE EMOTIONS AND ACUTE CARDIOVASCULAR RESPONSES TO LABORATORY CHALLENGES"

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In their provocative article, Feldman et al. (1) suggest that emotion may not be the primary pathway by which stress affects cardiovascular (CV) functioning. They reanalyze data from several laboratory-based cardiovascular reactivity studies and show that individual changes in CV (systolic blood pressure [SBP] and diastolic blood pressure and heart rate) functioning have a negligible to moderate correlation (between .13 and .34) with individuals' reports of changes in negative emotion (NE). The authors conclude that negative emotions probably "play a small role in physiological responses to acute laboratory stressors" (1).

The issue addressed in the article is an important one. There is a strong tendency in behavioral medicine research to assume that the physiological effects of stress are cognitively mediated. This assumption is so strong that emotion/mood data are collected primarily as a manipulation check, to verify or document that the laboratory task is indeed stressful, and have only rarely been used to test the assumed mediating role of emotions.

Despite my sympathy with the conclusion of their article, I have reservations about the analysis which led to these conclusions. Specifically, the computation of the correlation of change in negative emotion with change in CV functioning[1] begins by subtracting out of both sets of change scores the respective mean levels of change associated with each task. Tables 2 and 4 of the article show that the tasks (i.e. stressors) were associated with substantial increases in the CV measures, while Tables 3 and 4 show the same for negative emotion. These concurrent increases in both the CV measures and negative emotion are essentially lost in the correlation of change scores. The remainder of this comment will illustrate, conceptually and empirically, the potential problem of ignoring the mean change in both measures.

Consider a hypothetical task that was associated with an average increase of 15 mm Hg in systolic blood pressure and an average increase of 1 point on a 4-point negative emotion scale. Further suppose that this was a very finely tuned task and there were no individual differences (i.e. all individuals respond identically) in both the SBP and NE response. In this case, there is no correlation between the change scores. Yet, clearly the task had a large effect on both SBP and NE, and thus SBP and NE must be related to each other. In one sense, the association is perfect: every time NE increases by 1 point, SBP increases by 15 mm Hg. What we cannot disentangle in this example, or most of the others discussed below, is whether (a) the relationship between SBP and NE is spurious, (b) NE mediates the effect of the task on SBP, (c) SBP mediates the effect of the task on NE, or (d) there is a more complicated causal relationship involving reciprocal effects and/or other (omitted) factors.

Next, consider a modification to the above scenario. This time, I allow for individual differences in the SBP response, but continue to assume a constant change in NE for all individuals. Again, there is no correlation between change in SBP and change in NE, and yet the two must be related (this time, not perfectly) because the task leads to an increase in both.

In both scenarios, the central problem is the lack of variability in the change scores of one or both outcomes. Of course, there was variability in the change scores of both the CV measures and negative emotion for each of the studies analyzed by Feldman et al. (1). Without variability in CV reactivity, there would be no rationale for the hundreds of laboratory-based studies that have investigated whether race, gender, age, personality, etc. predict individual differences in reactivity. The hallmark of this type of research is the standardization of tasks. The goal of each particular task is to expose all individuals to the same amount of stress and look for differences in their CV response. While it is desirable that the task be associated with an increase in negative emotion as a manipulation check that the task is indeed stressful, the tasks are not designed to generate individual differences in emotional response. In fact, to the extent that negative emotion is an indicator of stress, one could argue that the goal of this research is to minimize the variability of the change in negative emotion, which starts to resemble the second hypothetical scenario described above.

Is it possible that much of the observed variability in the negative emotion change scores is due to measurement error? In their discussion, Feldman et al. (1) include a thoughtful review of several issues pertaining to self-report measurements of emotion. They conclude that, "The magnitude of stressor-elicited change in negative emotion appeared large enough to examine the association between NE and CV responses to stress." However, this refers to the average change in negative emotion, which is subtracted out in the calculation of the association between the two types of response to stress and provides no indication of the amount of reliable variance in the NE change scores. In this regard, it is also important to remember that change scores are usually less reliable than raw scores.[2]

How might one ensure that there was enough reliable variability in negative emotion to allow one to detect the hypoth-

[1] While not the focus of this comment, there is a growing consensus among statisticians that residualized change scores may be more problematic than raw change scores (2). One wonders whether the use of residualized change scores for the CV measures and raw change scores for NE may have suppressed the correlation between the CV change and NE change measures.

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[2] For example, if the reliability of the pretask and posttask measures of negative emotion were each .80 and the two measures are correlated .50, then the reliability of the change scores is .60. Under such a scenario, the reported correlations between the NE and CV responses to stress would be substantially attenuated due to unreliability. The Feldman article does not contain the information required to adjust their correlations for attenuation.

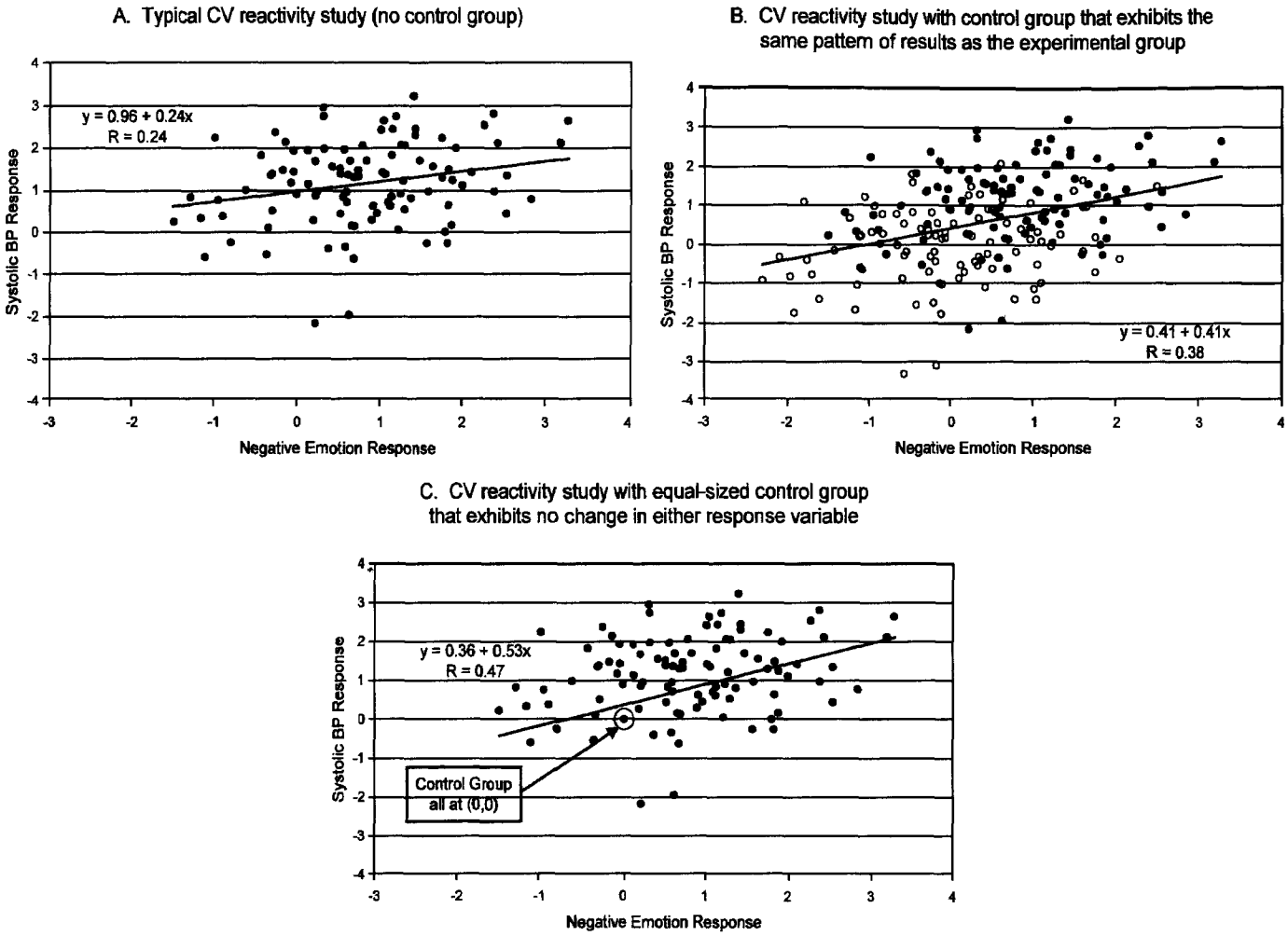

FIGURE 1: Systolic Blood Pressure and Negative Emotion Responses to a Handgrip Task—3 Scenarios.

esized associations? One possibility would be to include a control group that was not exposed to the task. To demonstrate how inclusion of a control group could alter the interpretation, I have constructed two scenarios based upon an approximation of one part of the Feldman et al. (1) data. The scenarios make different assumptions about the control group data, and I expect that the truth lies somewhere between these two extremes. Panel A shows a plot of a hypothetical dataset, constructed to simulate the meta analysis results for the relationship of NE change and SBP change for the three studies that used the handgrip task. The correlation is .24 (the median correlation in Table 5 of Feldman et al. [1]), the mean change in NE is .79 standard deviations, and the mean change in SBP is 1.17 standard deviations (calculated from the effect sizes reported in Table 4). Under the first scenario, shown in Panel B, the control group exhibits just as much variability in NE change and SBP change and the same association between them, but the mean change is zero on both outcomes. Under this condition, the correlation across the two groups between the two change measures increases to .38, and the R^2 is 2.5 times that for Panel A. In the second scenario, no one in the control group exhibits any change in NE or SBP, as represented by the exaggerated point at (0,0) in Panel C. Under this scenario, the correlation between the two change measures is .47, and the R^2 is nearly 4 times that for Panel A. By introducing a hypothetical control group that is not exposed to the task/stressor (and therefore does not exhibit an average increase in either NE or SBP), we build into the variability of both sets of change scores the variability that is associated with the task.

In summary, it is anticipated that in typical laboratory-based CV reactivity studies the correlation between NE and CV responses will be attenuated due to (a) restricted variability of NE (and perhaps CV) change scores and (b) the unreliability of change scores. Thus, while Feldman et al. (1) are correct to state that "the correlation between change in emotion and change in CV response is an estimate of the maximal [causal] role of emotion in the association between stress and CV response" (1, p. 218), their estimates of this correlation are probably minimal estimates and the true correlations are likely to be substantially greater. This said, it remains to be determined how much of this association is spurious versus causal.

REFERENCES

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