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

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Feldman et al. (1) analyzed data from several studies and found only small associations between cardiovascular responses and self-reported emotional responses to laboratory stressors. They concluded that "negative emotions play a small role in physiological responses to acute laboratory stressors." We believe this conclusion is premature.

The connection between emotionality and cardiovascular reactivity (CVR) can be examined at the level of tasks (Do tasks that produce strong emotions also produce large cardiovascular reactions?) and at the level of individual differences (Are people who show large emotional responses to a given task the same ones who show large cardiovascular reactions?). Feldman et al. (1) consider only the latter issue. We leave the discussion of the former to the accompanying commentary by Schwartz (2) (though we note that the difference between a resting baseline and, for example, an anger–recall or shock–threat task is the level of emotionality, and this has profound consequences for CVR [3,4]).

Even if limited to the examination of individual differences, the data presented by Feldman et al. (1) are not conclusive. The studies they review used star mirror-tracing, speech, and handgrip as the laboratory stressors. The use of such tasks limits the conclusions in two ways. First, the chosen tasks do not involve very powerful emotional interventions. Handgrip and star mirror-tracing are not tasks one would choose if one were interested in emotional responses, and even a speech task need not be highly emotional. (Along these lines, we note that Table 3 of the paper shows that in only 3 of the 16 cases was the average of self-reported negative emotionality above the midpoint—"moderate" on the scale.) Much more powerful emotions can be created in the laboratory than those engendered by squeezing a handgrip dynamometer. The use of strongly emotional tasks would not guarantee an association between subjective and physiological responses, but the use of emotionally neutral tasks does make finding an association between emotional reports and CVR very unlikely.

The second limitation is that the tasks used, like most standardized tasks used in experimental investigations, are intended to reduce individual difference variability, in order to maximize the odds of detecting the effects of various manipulations. In an ideal experiment, all subjects within a particular experimental condition would interpret the task in the same way and would evidence uniform psychological responses. In such a case, the correlation between emotional responses and CVR would have to be zero. The small correlations that are reported suggest that the investigators were largely successful at standardizing their tasks, with the remaining variability attributable mostly to random error. By way of contrast, an ambiguous task, such as "discuss whatever you like with your spouse," would lead to quite different interpretations by different participants. Experimental researchers avoid using such open-ended tasks; however, if one wants to find individual difference level associations between emotionality and magnitude of CVR, then such an unstructured, ambiguous task is exactly what should be used. The participants who fight with their spouses will likely show high CVR and report great emotionality, while those who discuss the weather will report little emotion and show little CVR. With adequate reliability of the measures, possibly enhanced by aggregation over sessions (5), we believe such an approach has some potential to find appreciable correlations between emotionality and CVR.

Beyond the specifics of whether the correct laboratory tasks were chosen, there are also broader issues about generalizing from laboratory reactivity to cardiovascular responses outside the laboratory. It remains unclear how well responses to laboratory tasks

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represent the affective and cardiovascular processes that occur in individuals reacting to real-world stressors (6). Emotional reactions in such laboratory reactivity paradigms may not be an adequate model of the broader reality—the one in which exaggerated blood pressure responses can lead to hypertension.

Finally, we would like to suggest that when examining cardiovascular responses and emotionality, it is likely to be fruitful to look not just at cardiovascular reactivity but also at poststress cardiovascular recovery. People who remain angry after a traffic confrontation, for example, may show slower cardiovascular recovery than those who can dismiss the matter from their minds. Similarly, emotional tasks appear to be associated with far slower recovery than nonemotional tasks (7), even when the reactivity during the task is similar (4). Some evidence suggests that cardiovascular recovery of prestress blood pressure levels may be an independent risk factor for hypertension (8–10) and may serve as a useful extension to the reactivity hypothesis (11). Little attention has been paid to the role of affect during poststress recovery, however, and this remains a potentially useful avenue of research.

In conclusion, affect may indeed account for only a small portion of the within-task variability in cardiovascular reactions to constrained laboratory tasks. However, we do not believe that this indicates that emotions do not contribute powerfully to cardiovascular responses.

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