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Authors

Bylsma, Lauren M
Mauss, Iris B
Rottenberg, Jonathan

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Is the Divide a Chasm?: Bridging Affective Science with Clinical Practice

Lauren M. Bylsma, Ph.D.,
Department of Psychiatry, University of Pittsburgh

Iris B. Mauss, Ph.D., and
Department of Psychology, University of California, Berkeley

Jonathan Rottenberg, Ph.D.
Department of Psychology, University of South Florida

Abstract

This special section endeavors to facilitate the integration of biologically-based assessments of emotion into the clinical setting. This goal is consistent with the Research Domain Criteria (RDoC) initiative, which aims to identify transdiagnostic biobehavioral mechanisms that underlie mental disorders. We focus on four challenges to applying biologically-informed research on emotion and emotion regulation to clinical contexts: (1) How do we assess emotion in an RDoC framework? (2) How do we integrate measures of emotion with other systems in a wider context? (3) What do physiological indices of emotion tell us about clinical phenomena? and (4) How do we integrate physiological assessments into clinical practice? Throughout this comment, we refer to the articles in this special section to make our points, and, when possible, offer suggestions for future work to continue to address these challenges.

Overview

The goal of this special section is to advance the integration of biologically-based assessments of emotion into clinical assessment, diagnosis, and treatment. This is an important goal for several reasons. First, such an initiative can help to narrow the now wide gap between basic affective science and clinical practice. Despite the proliferation of emotion research in the last decade, what we know from laboratory work on emotional reactivity and regulation is still only infrequently directly applied to clinical assessment or treatment settings. The goal of this special section is also germane to the Research Domain Criteria (RDoC) initiative, which aspires to identify transdiagnostic biobehavioral mechanisms associated with psychopathology (Sanislow, Pine, Quinn, Kozak & Garvey, 2010). Indeed, aspects of emotion reactivity and emotion regulation fit well into the RDoC initiative, and there is growing evidence of transdiagnostic emotion processes that can be measured using multiple units of analysis (including via physiology). For example, the LeMoult, Yoon & Joormann article (this issue) examines physiological indices of worry and rumination, which are relevant to multiple forms of psychopathology, including mood and anxiety disorders.

We laud these goals and the special section for calling attention to them. The empirical contributions in this issue highlight the clear progress that is being made towards these lofty goals. At the same time, the special section and the articles in it have provoked us to reflect, and to identify several major challenges in realizing these goals. Some of these challenges have been described elsewhere (Lilenfield, 2014). In this comment, we focus on four challenges to applying biologically-informed research on emotion and emotion regulation to clinical contexts. Where possible, we offer suggestions for how to begin addressing these challenges.

Challenge 1: How do we assess emotion in an RDoC framework?

Clinicians and researchers who work with emotion have long been mindful of the problem that emotion is a multi-headed beast (Levenson, 2003). When we say that someone is afraid, we can confirm with many types of data: self-reports of anxiety, sweaty palms, wide eyes, racing hearts, frozen posture, a strong urge to flee or avoid a danger, scanning the environment for threat. While RDoC focuses on multiple units of analysis, biological measures are given the most emphasis. There is a particular focus on neural measures, as mental disorders are characterized primarily as dysfunctions in brain circuitry, with the ultimate goal to develop treatments targeting specific brain regions (Sanislow et al, 2010). Although this might make sense from some perspectives, the idea that biology is pre-eminent is in tension with basic research in affective science, which has long viewed emotions (and, by extension, dysregulation of emotions) as multi-system phenomena (Lang, 1988; Levenson, 2003; Mauss & Robinson, 2009). Indeed, part of what makes affective science challenging is that there are a large number of reasonably valid indicators of emotion, from self-report of experience, to facial behavior, to a variety of physiological indices of central and peripheral nervous system activity.

A first problem in taming the multi-headed beast of emotion is that correlations between these different systems of emotion response are only modest (e.g., Mauss et al., 2005). Remarkably, modest intercorrelations are found even when we examine measures that are putatively within the same system, such as autonomic nervous system activity. There remain debates within affective science concerning why the correspondence between different indicators is so low and inconsistent (Barrett, 2012). Thus, it may be insufficient to measure only a single response or response system. If clinicians respond by measuring multiple responses or systems, affective science cannot yet provide strong guidance concerning how to integrate what is often discrepant information across systems. Findings such as these fed worries about the very scientific validity of the construct of emotion (Barrett, 2012; 2006). At a minimum, the phenomenon of modest intercorrelations among emotion indicators casts doubt on the idea that we could ever safely take a part of emotion (biology) for the whole.

Apart from this issue of the part-whole relationship (see Barrett 2009 for further discussion), the field has not come to consensus on what are the biological indicators of emotion (despite many promising leads). For example, meta-analytic studies have shown that several brain areas are sensitive to emotion (e.g., amygdala, dorsolateral prefrontal cortex), but no brain areas have been linked invariably to specific discrete emotions (Lindquist et al., 2012). Similarly, while studies of the autonomic nervous system have been successful at measuring

components of emotion, such as arousal (Lang, 1988), these measures have shown only a limited ability to distinguish between various emotions (Cacioppo, Berntson, Larsen, Poehlmann & Ito, 2000).

Although RDoC and other critics of the DSM system have rightly criticized the overreliance on self-report for diagnosis and assessment, subjective experience is a critical component of mental disorders and likely to remain so in future diagnostic manuals. For the foreseeable future, if a patient reports excellent emotional health, there will be no biological basis to refer him/her to psychotherapy. Along these lines, Franklin, Jamieson, Glenn & Nock (2015) and Miller (2010) observe, subjective mental phenomena (such as emotion) are not the same as objective physical phenomena, and by forcing this translation important information is lost. Kosslyn and Koenig (1992) provide the analogy of "replacing a description of architecture with a description of building materials. Although the nature of the materials restricts the kind of building that can be built, it does not characterize their function or design" (p. 4)." It is uncertain that an understanding of neurotransmitters and brain circuits alone can adequately describe the complexity of emotions.

As materialists, we and most scientists believe that psychological states are ultimately mediated by the brain and involve brain circuitry on some level. Biological measures are important and we all use them in our own research. Biological indices may constrain other aspects of the emotional system or identify risk factors before symptoms even arise. At the same time, whether biological measures of emotion should be seen as the "the first among equals" is an empirical question and should be decided on the basis of incremental clinical utility –for diagnosis or treatment decisions—for use of biological indices over other measures of emotion (e.g., Youngstrom & De Los Reyes, 2015). It remains to be seen whether the brain (or another biological system) is an optimal level of analysis to understand a construct like emotion that is also shaped by strong cultural and social forces that are exceedingly difficult to reduce to biology (see Berenbaum, 2013, Miller, 2010, for discussions). Our concern is that it is premature to assume biological measures are primary (and causally occur first), and that this holding this assumption might close off other important lines of work to understand emotion in all its complexity.

Challenge 2: How do we integrate measures of emotion with other systems in a wider context?

Apart from the difficulty of integrating biological measures of emotion across multiple systems (e.g., subjective experience, behavior, physiology, neural measures), there is the challenge of integrating emotion in the larger systems in which it is embedded, such as social, cultural, and developmental frameworks. Developmental processes and environmental factors are a component of RDoC but they are not a major focus of the framework, which is seeking relatively invariant markers of psychopathology.

Franklin et al. (2013) proposed that developmental psychopathology frameworks could inform the challenge of the RDoC endeavor to integrate across multiple systems. Developmental frameworks do not give the primacy to biological measures that RDoC does (Cicchetti, 1993; Rutter & Stroufe, 2000), but instead focuses on interactions between biological measures (including psychophysiology) and the environment, such as adverse life

events, and emphasizes moderators of these pathways and changes across the lifespan (Franklin, et al., 2013). Indeed, examination of cross-level interactions in multiple systems may be critically important to gain a true understanding of causal mechanisms underlying psychopathology (e.g., Kendler, 2014).

A key point is that biological markers of emotion dysregulation are not invariant. Whether a biological pattern reflects a specific aspect of emotion dysregulation may change both across the lifespan and across the development of a disorder (i.e., from a period of vulnerability to first onset of a disorder to remission and recurrence of a disorder). It is clear that the transactional nature of the relationship among variables is complex and various risk factors do not confer uniform risk for all individuals in the same way or the same over time across the lifespan (Cicchetti, 1993; Rutter & Stroufe, 2000; Beauchaine, Gatzke-Kopp, & Mead, 2007).

The existence of these moderating factors may partly explain why the literature on emotional reactivity and regulation is often quite mixed and inconsistent, such as findings on RSA in depression (Bylsma, Salomon, Taylor-Clift, Morris & Rottenberg, 2014). This underscores the need for future work on biological markers of emotion to incorporate developmental, environmental, and/or contextual factors. For example, there is growing evidence that context impacts whether or not an emotion regulation strategy is adaptive or maladaptive (Aldao, 2013; Troy, Shallcross & Mauss, 2013).

In sum, even if we could find an excellent set of physiological measures indexing some emotional process, we still must consider how that relationship might change with context or developmental stage. While not a current emphasis of RDoC, it is clear that developmental processes and environmental factors are also critical in integrating across multiple units of analysis and understanding emotional process as they relate to psychopathology.

Challenge 3: What do physiological indices of emotion tell us about clinical phenomena?

Given this pair of challenges from basic research, it not surprising that when we survey the data we see that biological measures of emotion exhibit only a probabilistic relationship to symptoms of psychopathology. Take, for example, the finding of low RSA levels in depression. Meta-analytic evidence provides support for a modest negative association between levels of RSA and depression (Rottenberg, 2007). The modest effect size leads to the expectation that individual research studies will often not find an effect. In line with this expectation, the Kircanski et al. paper from this special section that did not find any baseline RSA differences in MDD versus controls. The articles in this special section, in many ways, reflect the state of the art of the field in which designs are mostly correlational, and research findings show the presence of a modest association between a physiological measure and a clinical phenomenon. Relatedly, it remains unclear what the prediction accuracies are for the measures of emotion dysfunction that are available. For example, although we can say individuals with MDD are more likely to exhibit low RSA or blunted RSA reactivity, there is no established clinical threshold or established prediction accuracy for how well we can predict someone might have depression now or in the future based on these physiological values (e.g., at what values does RSA level or reactivity become pathological?). In these respects, at this stage of knowledge, we are asking clinicians and treatment researchers to

take something of a leap of faith to use physiological measures that have only a modest association with other measures of emotion or clinical outcomes.

The modest relationships among measures and symptoms call attention to the need for better norms in physiological measures, so that they can be more portable to a clinical environment. As Aldao and De Los Reyes (2015) note, it can be unclear how physiological indicators relate to clinically relevant information due to lack of adequate normative comparisons. For example, for any given laboratory task and psychophysiological measure of emotional reactivity or regulation, we lack clear normative data for normal healthy populations (complicating interpretation context of psychopathology). While the range normative levels of physiological variables is for resting baselines, we lack established norms for appropriate physiological reactivity to many if not most of the paradigms used in emotion research.

One critical problem is neglect of measurement error in laboratory studies that measure emotion and similar phenomena (see Berenbaum, 2013, for further discussion). As this point, self-report or behavioral measures of behavior, cognition, or emotion are often superior predictors of psychiatric problems than biological measures (e.g., Haeffel et al., 2008; Kwapil, 1998; Lilenfield, 2014). A next step for research that justifies the incorporation of these measures into clinical assessments would be to show that aphysiological measure has incremental validity over another existing indicator of disorder, of course, of response to treatment, etc.

Part of this issue can be addressed by improving measurement error in our laboratory psychophysiological studies. For example, laboratory measures often have low temporal and cross-situational consistency and are easily influenced by slight changes in the environmental context or state variables (Epstein, 1979, 1980). This could be particularly relevant for emotion regulation, where there is evidence that its effectiveness and associations with outcomes depend upon the context (Aldao, 2013; Troy et al., 2013). It would benefit laboratory studies to obtain more data on test-retest reliability of psychophysiological measures of emotion, conduct more replication studies across a variety of large samples (both healthy and disordered), and systematically examine relevant contextual factors. Ambulatory physiological assessments in daily life may also be useful to address contextual factors that may be difficult to replicate in the laboratory environment.

In keeping with most prior work on biological measures of emotion in psychopathology, three of the four articles in this special section focus on DSM diagnostic categories. Consistent with the RDoC initiative, prior findings may be muddied since they are based on problematic DSM categories, including high levels of co-morbidities and symptom heterogeneity within the diagnostic categories. Along these lines, some findings have suggested that disorders such as depression may instead reflect heterogeneous symptom clusters that overlap significantly with other related disorders (e.g., Fried, 2015; Fried & Nesse, 2014). Thus, in order to validate RDoC constructs using biological measures, what we additionally need are larger scale studies recruiting individuals with a wide range of symptoms and diagnostic presentations, ranging from mild to clinically impairing levels. Indeed, we must be cautious of focusing our search on a specific underlying mechanism for

a DSM-defined mental disorder when there may be multiple distinct mechanisms corresponding to several distinct syndromes, or where some disorders thought to be distinct may actually reflect the same underlying pathology (Kendler, 2014).

Challenge 4: How do we integrate physiological assessments into clinical practice?

A key focus of this special section is to integrate physiological measures into assessment and interventions in clinical practice. The editors rightly point out that recent advances in technology may help integrate physiological measures into clinical practice by reducing some of earlier challenges with availability, affordability and ease of use. As the articles in the special section highlight, physiological measures have the potential to improve clinical assessment and intervention, particularly if they can be utilized in an efficient, user-friendly, and cost-effective manner. This special section takes us one step further into making that a reality. Nevertheless, significant practical challenges remain before psychophysiological measures can be brought into widespread clinical practice.

One lingering issue is the cost of the software and specialized equipment. As mentioned elsewhere in this special section, free software may help alleviate resource issues. Indeed Kircanski et al. (this issue) used ANSLAB for their physiological processing, which is a free software program (Wilhelm, Grossman, & Roth, 1999). Weeks, Srivastav, Howell & Menatti (this issue) used expensive software, but note that it is possible to do similar analyses of vocal pitch using freely available software and an inexpensive voice recorder (which most clinicians probably have in their practice).

Although data collection has become much easier, it would also be unwise to minimize the expertise and time investment still required to utilize physiological measures in a reliable and valid way. Areas of expertise and time investment include how to troubleshoot problems of data acquisition, data cleaning and processing, and analysis and interpretation of the resulting data. Gaining such expertise is impeded by the fact that no comprehensive and widely accepted manuals exist, and it can take years to become technically proficient. While free software may be available, often free programs require even more technical skill, have less user-friendly features, and may provide more limited technical support, relative to the more expensive software programs. Weeks et al. (this issue) noted that they did not use the free software because they needed to ensure that the background noise was thoroughly removed of any artifacts. This would be a significant concern in clinical settings where it may be challenging to achieve all the environmental control of a typical laboratory environment. Would clinicians using freely available software be given the tools to ensure that artifacts such as electrical interference don’t contaminate data collection? None of these issues is insurmountable, but each could bear further thought.

Of note, problems with incorporating psychophysiological measures into the clinical setting may vary by the type of measure. For example, measures such as heart rate, skin conductance, or blood pressure may be more readily incorporated into the clinical setting, as these do not require as much expertise or special software to acquire and process the data. In contrast, measures such as pre-ejection period require greater expertise for accurate data collection with impedance cardiography, and the quantification of RSA hinges on high

sampling rates and more sophisticated algorithms than might be readily accessible in clinical settings.

The Dunn, Aldao & De Los Reyes article (this issue) tries to address some of these challenges by using Chernoff faces, a method for graphically depicting physiological arousal to aid in clinical decision making, which is an innovative idea that can make interpretation of complex physiological signals easier for clinicians. If developed, even patients may ultimately be able to gain insight into their own physiological responding, in some cases. It may be helpful for the patient to become aware of their physiological arousal and reinforce their success with the exposures during the course of therapy. However, even with this method, of course, some expertise is still needed to ensure that good signals are being acquired, and that the data are properly processed.

In particular, ambulatory devices are becoming more readily available, including some marketed to the general public for monitoring related to aspects such as exercise, fitness and sleep, which have necessitated making the devices cheaper and more user-friendly. Many of these devices are linked to “apps” on smartphones that are very easy to use. Some of these may be more scientifically sound than others, and further validation is needed. However, there is potential for similar devices and software to be developed for use in a clinical setting. For example, Wichers et al. (2011) reviews the potential applications of momentary assessment technology for depression in a clinical setting.

Another set of issues concerns the type of stimuli (e.g., emotional films, stressor tasks, emotion regulation tasks, etc.) that should be used in a clinical setting. An assumption might be that these should be the same as are used in laboratory settings. For example, the Dunn et al. article (this issue) uses standardized emotional film stimuli. Such film clips are a gold standard in laboratory studies using physiological measures of emotion reactivity because they are readily standardized across individuals (Rottenberg, Ray & Gross, 2007). Or, take the case of the Kircanski, Waugh, Camacho & Gotlib article (this issue), who used the Trier stress task in a sample of participants diagnosed with GAD and MDD. Again, this task is widely used in laboratory stress inductions (Dickerson & Kemeney, 2004). However, what constitutes a gold standard in the laboratory may not be the most helpful and appealing to the clinician and patient. For example, film and other stimuli used in the laboratory may not be easily accessible to a clinician and could require computers or other equipment to display. Further, the Trier stress task can be quite upsetting, which may make clinicians and patients hesitant to use this in the context of therapy when it does not have clear therapeutic value. Some of the laboratory tasks also require the use of confederates, which would be difficult to use in a therapy setting. Instead, for example, for a person with social anxiety, it could make the most sense to use social exposures from the person’s fear hierarchy as part of an initial clinical assessment, followed by additional social exposures throughout the course of therapy as the person works up their fear hierarchy. Or, in the case of a person with GAD, a worry exposure could be used. Or, for a person with MDD, the patient could be led to think about a current situation that is affecting their mood. In clinical settings, standardized laboratory stimuli are not needed, especially since good normative data are not usually available. Using idiosyncratic stimuli would also increase the feasibility of obtaining

physiological responses, since no additional time and resources would be spent on stimuli that aren't relevant to the person's treatment plan.

Another suggestion to make these tools more clinically relevant and appealing to clinicians is inspired by the use of Chernoff faces (Dunn et al., this issue). Perhaps instead of detecting facial features, it could be possible to convert the signals visually to something akin to a feelings thermometer, a common tool in cognitive-behavioral therapy. This could be a useful clinical tool in contexts such as exposure therapy for anxiety disorders. For example, as a patient works up the steps of his or her fear hierarchy with a therapist, a physiological assessment of the patient's arousal level can complement self-report ratings. This information could help to inform the clinician about when the patient successfully habituates to each level of the fear hierarchy and is ready to move on to a higher level. The patient could also potentially be getting some positive reinforcement from seeing the corresponding changes in physiology as he or she works up the fear hierarchy.

Although the aforementioned challenges will need to be addressed before physiological measures can be adequately used to make diagnostic or treatment decisions, we may begin to use physiology to enhance assessment and treatment in the ways we highlighted above, provided that we don't give undue weight to biological measures or ignore other experiential and behavioral measures that may be as or more important.

Summary and conclusion

This special section has drawn needed attention to the goal of integrating biological measures of emotion and emotion regulation into clinical practice. In this comment, we have highlighted the many additional steps that will need to be taken to accomplish this goal. RDoC is conceptualized as a dynamic long-term program of research that is a "vision for the future" (Insel, 2010). Our view is that we are still some time off from fully validated RDoC constructs that can be easily integrated into clinical practice. We, too, are excited about this vision, but the conceptual and practical challenges of this integration should not be underestimated.

References

Aldao A. The future of emotion regulation research: Capturing context. Perspectives on Psychological Science. 2013; 8:155–172. [PubMed: 26172497]

Aldao A, De Los Reyes A. Commentary: A practical guide for translating basic research on affective science to implementing physiology in clinical child and adolescent assessments. Journal of Clinical Child & Adolescent Psychology. 2015; 44(2):341–351. [PubMed: 25664768]

Barrett LF. Emotions as natural kinds? Perspectives on Psychological Science. 2006; 1:28–58. [PubMed: 26151184]

Barrett LF. The Future of Psychology: Connecting Mind to Brain. Perspectives in Psychological Science. 2009; 4:326–339.

Barrett LF. Emotions are real. Emotion. 2012; 12:413–429. [PubMed: 22642358]

Berenbaum H. Classification and psychopathology research. Journal of abnormal psychology. 2013; 122(3):894. [PubMed: 24016025]

Bylsma LM, Salomon K, Taylor-Clift A, Morris BH, Rottenberg J. Respiratory sinus arrhythmia reactivity in current and remitted major depressive disorder. Psychosomatic medicine. 2014; 76(1): 66–73. [PubMed: 24367127]

Cacioppo JT, Berntson GG, Larsen JT, Poehlmann KM, Ito TA. The psychophysiology of emotion. Handbook of emotions. 2000; 2:173–191.

Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. Psychological bulletin. 2004; 130(3):355. [PubMed: 15122924]

Epstein S. The stability of behavior: I. On predicting most of the people much of the time. Journal of Personality and Social Psychology. 1979; 37:1097e1126.

Epstein S. The stability of behavior: II. Implications for psychological research. American Psychologist. 1980; 35:790e806.

Franklin JC, Jamieson JP, Glenn CR, Nock MK. How developmental psychopathology theory and research can inform the research domain criteria (RDoC) project. Journal of Clinical Child & Adolescent Psychology. 2015; 44(2):280–290. [PubMed: 24555423]

Fried EI. Problematic assumptions have slowed down depression research: why symptoms, not syndromes are the way forward. Frontiers in psychology. 2015; 6

Fried EI, Nesse RM. Depression is not a consistent syndrome: an investigation of unique symptom patterns in the STAR* D study. Journal of affective disorders. 2015; 172:96–102. [PubMed: 25451401]

Haeffel GJ, Gibb BE, Metalsky GI, Alloy LB, Abramson LY, Hankin BL, Swendsen JD. Measuring cognitive vulnerability to depression: Development and validation of the cognitive style questionnaire. Clinical Psychology Review. 2008; 28(5):824–836. [PubMed: 18234405]

Kendler KS. The structure of psychiatric science. Perspectives. 2014; 171(9):931–938.

Kosslyn, SM.; Koenig, O. Wet mind: The new cognitive neuroscience. Simon & Schuster; 1992.

Kwapil TR. Social anhedonia as a predictor of the development of schizophrenia-spectrum disorders. Journal of abnormal psychology. 1998; 107(4):558. [PubMed: 9830243]

Lang, PJ. What are the data of emotion?. In: Hamilton, V.; Bower, GH.; Frijda, NH., editors. Cognitive perspectives on emotion and motivation. New York: Kluwer Academic/Plenum Press; 1988. p. 173-191.

Levenson, RW. Blood, sweat, and fears: The autonomic architecture of emotion. In: Ekman, P.; Campos, JJ.; Davidson, RJ.; de Waal, FBM., editors. Emotions inside out. New York: The New York Academy of Sciences; 2003. p. 348-366.

Lilienfeld SO. The Research Domain Criteria (RDoC): an analysis of methodological and conceptual challenges. Behaviour research and therapy. 2014; 62:129–139. [PubMed: 25156396]

Lindquist KA, Wager TD, Kober H, Bliss-Moreau E, Barrett LF. The brain basis of emotion: a meta-analytic review. Behavioral and Brain Sciences. 2012; 35(03):121–143. [PubMed: 22617651]

Mauss IB, Robinson MD. Measures of emotion: A review. Cognition and emotion. 2009; 23(2):209–237. [PubMed: 19809584]

Miller GA. Mistreating psychology in the decades of the brain. Perspectives of Psychological Science. 2010; 5:716–743.

Rottenberg J. Cardiac vagal control in depression: A critical analysis. Biological Psychology. 2007; 74:200–211. [PubMed: 17045728]

Rottenberg, J.; Ray, RD.; Gross, JJ. Emotion elicitation using films. In: Coan, JA.; Allen, JJB., editors. The handbook of emotion elicitation and assessment. London: Oxford University Press; 2007. p. 9-28.

Sanislow CA, Pine DS, Quinn KJ, Kozak MJ, Garvey MA, Heinssen RK, Cuthbert BN. Developing constructs for psychopathology research: research domain criteria. Journal of abnormal psychology. 2010; 119(4):631. [PubMed: 20939653]

Troy AS, Shallcross AJ, Mauss IB. A person-by-situation approach to emotion regulation: Cognitive reappraisal can either hurt or help, depending on the context. Psychological Science. 2013; 24:2505–2514. [PubMed: 24145331]

Wichers M, Simons CJP, Kramer IMA, Hartmann JA, Lothmann C, Myin-Germeys I, van Os J. Momentary assessment technology as a tool to help patients with depression help themselves. Acta Psychiatrica Scandinavica. 2011; 124(4):262–272. [PubMed: 21838742]

Wilhelm FH, Grossman P, Roth WT. Analysis of cardiovascular regulation. Biomedical Sciences Instrumentation. 1999; 35:135–140. [PubMed: 11143335]

Youngstrom EA, De Los Reyes A. Moving Towards Cost-Effectiveness in Using Psychophysiological Measures in Clinical Assessment: Validity, Decision-Making, and Adding Value. Journal of Clinical Child and Adolescent Psychology. 2015; 44:352–361. [PubMed: 25664769]