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Heart rate variability biofeedback: Theoretical basis, delivery, and its potential for the treatment of substance use disorders

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Abstract

Heart rate variability biofeedback (HRV BFB) is a biobehavioural clinical intervention that is gaining growing empirical support for the treatment of a number of psychological disorders, several of which are highly comorbid with substance use disorders (SUDs). The present article reviews the autonomic nervous system bases of two key processes implicated in the formation and maintenance of addictive pathology—affect dysregulation and craving—and asks if HRV BFB may be an effective intervention to ameliorate autonomic nervous system dysregulation in these processes, and as such, prove to be an effective intervention for SUDs. A detailed description of HRV BFB and its delivery is provided. Preliminary evidence suggests HRV BFB may be an effective addendum to current first-line SUD treatments, though no firm conclusions can be drawn at this time; more research is needed.

Keywords

Affect dysregulation; craving; heart rate variability biofeedback; substance use disorders

Introduction

In the past 30 years, major advances have been made in the treatment of substance use disorders (SUDs). These advances in treatment have largely been born out of the recognition that SUDs are complex behavioural phenomenon with biological, psychological and social antecedents. In spite of such advances, however, maintaining gains during and after SUD treatment remains a challenge for the majority of individuals (Miller, Walters, & Bennett, 2001; O'Brien & McLellan, 1996).

Interventions such as cognitive-behavioural therapy (CBT) are effective in that they target multiple cognitive and behavioural deficiencies and vulnerabilities that are known to maintain behaviours associated with SUDs. It is possible, however, that such treatments are

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failing to address important factors that are active in sustaining SUD pathology, because phenomena that lead to relapse, such as affect dysregulation and craving, are mediated by physiological as well as cognitive processes. As such, biobehavioural interventions like heart rate variability biofeedback (HRV BFB) that target physiological components of affect dysregulation and craving offer promise as complimentary treatments to current first-line interventions.

Affect dysregulation and craving in SUDs

Individuals' efforts to self-regulate positive and negative affect is central to the development and maintenance of SUDs (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Le Moal & Koob, 2007). This is supported by the observation that individuals with SUDs experience high emotional intensity (Thorberg & Lyvers, 2006), high emotional lability (Simons, Carey, & Wills, 2009) and low distress tolerance (Gorka, Ali, & Daughters, 2011). Further, individuals with SUDs frequently cite difficulty regulating affect as a precursor to early use, as well as a major contributor to relapse (Berking et al., 2011; Witkiewitz & Villarroel, 2009).

Craving, though difficult to measure (Tiffany & Wray, 2009), is another central feature of addictive disorders (Franken, 2003; Weiss, 2005) and was recently added to the Diagnostic Manual of Mental Disorders as diagnostic marker for SUDs (APA, 2014). In addition to playing an important antecedent role in the development and maintenance of SUD-related behaviours (Hasin, Fenton, Beseler, Park, & Wall, 2012), craving also predicts relapse after periods of abstinence (Paliwal, Hyman, & Sinha, 2008; Sinha, 2011).

As well as acting as independent antecedents and maintainers of SUD pathology, affect dysregulation and craving interact. Difficulty in regulating affective states may predispose an individual to use alcohol and other drugs to cope emotionally (Bradley, 2003), and successive exposures to alcohol and other drugs leads to craving (Everitt et al., 2008; Robinson & Berridge, 2008). Although substance use may to a certain extent reduce negative affect (Cooper, Frone, Russell, & Mudar, 1995; Cox & Klinger, 2011) and ameliorate craving in the moment, chronic substance use exacerbates the problem by adding negative affect and perpetuating the cycle of craving, while impairing neural control of affective states, leading to a vicious cycle that contributes to the escalating nature of SUDs (Koob & Le Moal, 2001; Robinson & Berridge, 2008).

Affect regulation and craving are commonly conceptualized as a wholly cognitive constructs. In reality, however, affect and craving have fundamental physiological attributes. As such, they can be thought of as “biobehavioural” phenomenon because they involve integrated psychological and physiological processes, occurring both within, and outside of conscious awareness (Diamond & Aspinwall, 2003; Forgas, 2008; Gross, 1998; Verheul, van den Brink, & Geerlings, 1999). Further, the adaptive utility of affect regulation processes and craving, and their specific influence on SUDs, can be attributed to the integrated brain–body systems that control them (Critchley, 2005, 2009; Ingjaldsson, Laberg, & Thayer, 2003; Porges, 2009; Quintana, McGregor, Guastella, Malhi, & Kemp, 2013).

Physiological components of affect regulation and craving

The experience of affect and craving, usually driven by emotional states or environmental factors, is attendant with moment-to-moment changes in physiological state that prepare an individual as a whole for action (Garland, Carter, Ropes, & Howard, 2012; Levenson, 2003; Quintana, Guastella, McGregor, Hickie, & Kemp, 2013). Such changes may include, but are not limited to, important changes in the cardiovascular system, including increases or decreases heart rate and blood pressure, and changes in blood perfusion to specific regions of the brain to meet the metabolic demands of active neurons. A flexible autonomic nervous system allows for rapid generation or modulation of physiological states in accordance with situational demands (Porges, 2009). In contrast, autonomic rigidity results in a lessened capacity to generate or alter physiological responses in synchrony with affective changes, or changes in the environment (Appelhans & Luecken, 2006; Lehrer & Eddie, 2013).

HRV as a window into the central autonomic network

Psychophysiological regulatory processes are mediated by a neural substrate known as the central autonomic network (Benarroch, 1997; Card & Sved, 2011; Jellinger, 1998). The central autonomic network comprises the prefrontal cortex, insular cortex, amygdala, hypothalamus, periaqueductal gray matter, parabrachial complex, nucleus of the tractus solitarius and ventrolateral medulla, as well as the peripheral autonomic nervous system (Benarroch, 1993). The central autonomic network serves to modulate biobehavioural resources in emotion by flexibly adjusting physiological arousal in accordance with changing situational demands (Friedman & Thayer, 1998), and therefore, serves a key role in affect regulation and substance craving (Verheul, van den Brink, & Geerlings, 1999) as well as goal-directed motor behaviours.

Efferent pathways, descending from the brain's central autonomic network carry both sympathetic and parasympathetic signals that converge on the heart's central pacemaker, the sinoatrial node (Katz, 2010). Sympathetic (i.e. thoracic visceral) and parasympathetic (i.e. vagus) nerves act on cardiac neurons in the heart's sinoatrial node to increase or decrease heart rate (Bibevski & Dunlap, 2011). Thus, the amount of time, or period, between each heartbeat is continually changing depending on the balance of sympathetic and parasympathetic input being received by the sinoatrial node. As such, neurocardiac communication can be assessed by measuring the psychophysiological function of HRV, the subtle changes in the time-intervals between heart beats (Benarroch, 1997; Thayer & Brosschot, 2005). HRV is calculated by measuring the variance in the R-spike to R-spike intervals of the electrocardiogram (ECG), or the approximation of heart beat-to-beat intervals derived by basic capillary plethysmography.

Heart–brain bidirectional communication

The body's feedback to the central autonomic network further contributes to HRV. This is accomplished through afferent signalling (i.e. signalling from the viscera to the brain) via the baroreflex mechanism (Benarroch, 2008; Goldstein, Bentho, Park, & Sharabi, 2011; Vaschillo, Vaschillo, & Lehrer, 2006). This physiological reflex arc plays a key role in the regulation of blood pressure and heart rate, and in doing so aids the integration of cognitive and physiological aspects of emotion regulation (Thayer & Brosschot, 2005; Vaschillo, Vaschillo, Buckman, Pandina, & Bates, 2011). The sensory component of the baroreflex consists of stretch receptors in the body's arteries, which detect moment-to-moment changes in arterial blood pressure, signalling this information back up to central autonomic network structures in the mid-brain. This information is integrated with information from the cortex concerning the perceived appropriateness of a given state of cardiovascular arousal, and in a constant loop, information is signalled back down to the heart and other organs. Thus, the central autonomic network communicates with the viscera via a series of feedforward and feedback loops contained within the autonomic nervous system (Pessoa, 2008) to modulate autonomic nervous system functions, as needed, to adapt to physical and cognitive-emotional challenges (Benarroch, 1997).

HRV as marker of health, affective regulation and SUD problems

The autonomic function of HRV has proven to be an informative indicator of brain–body integration that is relevant to affective regulation and craving. Relatively higher levels of HRV have been linked consistently to emotional resilience and stress vulnerability (Appelhans & Luecken, 2006; Ingjaldsson, Laberg, & Thayer, 2003; Thayer, Hansen, & Johnsen, 2010), as well as an individual's overall physical health (Britton et al., 2007; Lehrer et al., 2006; Vanderlei, Pastre, Hoshi, Carvalho, & Godoy, 2009). At rest, well-functioning systems generally exhibit a high degree of complexity and flexibility (i.e. high HRV), whereas illness is characterized by lower HRV, possibly indicative of a decoupling of autonomic nervous system components (Goldberger, Peng, & Lipsitz, 2002; Pincus & Goldberger, 1994). This is especially true in the case of SUDs, wherein individuals with SUDs have been shown to have lower resting HRV than individuals without substance use problems (Ingjaldsson, Laberg, & Thayer, 2003; Weise, Müller, Krell, Kielstein, & Koch, 1986). While this association could potentially be a factor of the direct pharmacological actions of alcohol and other drugs, or a sequela of lifestyles attendant with chronic heavy substance use, some suggest this is not the case (Peterson, Pihl, Seguin, Finn, & Stewart, 1993; Zhang, Abdel-Rahman, & Wooles, 1988). The fact that individuals with a variety of mental disorders also exhibit lower resting HRV in the absence of neurotoxic substances, speaks to this postulate. Lower background, or resting state levels of HRV are found in individuals with posttraumatic stress disorder (Cohen et al., 2000), panic disorder (Klein, Cnaani, Harel, Braun, & Ben-Haim, 1995) and phobic anxiety (Kawachi, Sparrow, Vokonas, & Weiss, 1995). Further, individuals with major depression have lower HRV than individuals without depression (Nahshoni et al., 2004; Udupa et al., 2007), and HRV has been shown to be negatively related to depression severity (Agelink, Boz, Ullrich, & Andrich, 2002; Agelink et al., 2001).

How awareness of biobehavioural processes can inform treatment

In addition to the information value of HRV in assessing difficulties in affective regulation, further understanding of psychophysiological modulation of arousal may have important treatment implications. While reduced HRV is associated with certain anxiety disorders and depression (Gorman & Sloan, 2000), non-pharmacological treatments for these disorders

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have been shown to reverse this effect. For instance, Carney and colleagues (2000) observed significantly increased HRV in individuals who had responded successfully to CBT for major depression. Similarly, Chambers and Allen (2002) found that increased HRV from before to after treatment was robustly related to a decrease in depression severity in a sample of women receiving acupuncture for major depression. In addition to the spontaneous increases in HRV that have been observed following successful treatment for depression, other studies have attempted to effect changes in affective disorders by directly modifying HRV. Biofeedback techniques, which utilize rhythmic breathing to activate the baroreflex mechanism, enhancing vagal tone and thus increasing HRV, have been shown to reduce depression (Karavidas et al., 2007; Siepmann, Aykac, Unterdorfer, Petrowski, & Mueck-Weymann, 2008), and symptoms associated with posttraumatic stress disorder (Zucker, Samuelson, Muench, Greenberg, & Gevirtz, 2009). This literature suggests that biofeedback techniques that increase vagal tone and baroreflex gain (i.e. baroreflex engagement) may have the potential for the treatment for SUDs (Cheetham, Allen, Yücel, & Lubman, 2010), as well as other disorders associated with decreased HRV.

HRV BFB, an overview

HRV BFB originated from observation that maximal increases in the amplitude of heart rate oscillation are produced when the cardiovascular system is rhythmically stimulated by paced breathing at a frequency of ~0.1 Hz (6 breaths/min; Song & Lehrer, 2003; Vaschillo, Lehrer, Rishe, & Konstantinov, 2002). This effect is linked to respiratory sinus arrhythmia and resonance properties of the cardiovascular system resulting from activity of the baroreflex (Vaschillo et al., 2002, 2006). Resonance usually occurs in the low frequency range (Wills et al., 2001), typically between 0.075 and 0.108 Hz (i.e. between 4.5 and 6.5 breaths/min), with the average resonance frequency being slightly <0.1 Hz, or ~5.5 breaths/min (Vaschillo et al., 2002). Resonance effects produce very large increases in both HRV and baroreflex gain. Individuals' optimal breathing rate (i.e. their resonance frequency) for effecting large increases in HRV and baroreflex gain varies slightly from person to person based on individual differences in physiology and factors like height (Vaschillo et al., 2006).

A growing number of studies shows HRV BFB's potential for treating a variety of physical and mental disorders, including depression (Karavidas et al., 2007; Siepmann et al., 2008; Zucker et al., 2009), post-traumatic stress disorder (Tan, Dao, Farmer, Sutherland, & Gevirtz, 2011; Zucker et al., 2009), various anxiety disorders and stress symptoms (Henriques, Keffer, Abrahamson, & Horst, 2011; McCraty, Atkinson, Lipsenthal, & Arguelles, 2009; Nolan et al., 2005; Zucker et al., 2009), food craving (Meule, Freund, Skirde, Vögele, & Kübler, 2012), hypertension (Lin et al., 2012; McCraty et al., 2009; Nolan et al., 2005), chronic pain (Hallman, Olsson, von Schéele, Melin, & Lyskov, 2011; Hassett et al., 2007; Sowder, Gevirtz, Shapiro, & Ebert, 2010), asthma (Lehrer, Smetankin, & Potapova, 2000; Lehrer et al., 2004) and heart disease (Nolan et al., 2005). HRV BFB has also been shown to reduce symptom severity and improve quality of life for individuals with chronic obstructive pulmonary disease (Giardino, Chan, & Borson, 2004), and heart failure (Moravec, 2008), as well as for individuals reporting medically unexplained symptoms (Katsamanis et al., 2011).

Concise HRV BFB training protocols, as well as single sessions of HRV BFB training delivered in laboratory experiments, have also been shown to enhance cognitive processing in PTSD (Ginsberg, Berry, & Powell, 2009), as well as emotion and cognitive regulation in individuals with cognitive impairments, and healthy adults. For instance, Kim et al. (2013) found brief HRV BFB training with recommended practice >10 weeks improved emotion and cognitive regulation in individuals with severe, chronic brain injury, while Prinsloo et al. (2011) showed a single session of HRV BFB training improved cognitive performance in healthy adults during a modified stress inducing Stroop task. Four to five sessions of HRV BFB training, plus practice, has also been shown to reduce performance anxiety and improve subjective performance in classical musicians (Thurber, Bodenhamer-Davis, Johnson, Chesky, & Chandler, 2010). Further, a single session of slow, paced breathing approximating resonance frequency, with or without biofeedback, immediately before performance was shown to significantly reduce performance anxiety in musicians with high baseline anxiety (Wells, Outhred, Heathers, Quintana, & Kemp, 2012).

Delivering HRV BFB

Until recently, HRV BFB was delivered using sophisticated ECG or plethysmograph recording equipment, however, a number of practical, affordable options are now available for clinicians wishing to do HRV BFB in their clinics or private practices. While HRV BFB may be concisely delivered in few sessions, training is best conducted over 10, 30–45-min sessions, as originally outlined by Lehrer, Vaschillo, & Vaschillo, 2000.

Typically in Session 1, trainees are oriented to the rationale for HRV BFB and provided with psychoeducation about the theoretical basis for the intervention. The role of autonomic nervous system dysregulation in the particular disorder being treated is used as a basic rationale for providing HRV BFB training. Trainees learn that with regular practice of the technique, they will help improve their autonomic functioning, and thus effect changes in their presenting problem. Moreover, when being applied to disorders of affect, participants learn that the breathing technique can be used acutely to help emotion self-regulation, in the moment.

Session 1 is also used to determine trainees' resonance frequency. This is done by instructing the client to breathe using a pacing stimulus for 3 min at each of several frequencies in the neighbourhood of 0.1 Hz (i.e. 6.5, 6, 5.5, 5 and 4.5 breaths/min). The clinician observes aspects of the trainees practice such as the coherence between respiration and HRV, as well as their apparent comfortability with each breathing rate. Assessing resonance frequency is challenging with basic HRV recording devices that do not measure respiration. However, by observing the HRV waveform one can get a sense of a trainees' optimal breathing rate. It is worth noting that individuals' resonance frequency commonly changes slightly as they become more adept at the practice through the 10 sessions of training. Participants are not exposed to actual biofeedback in the first session, but rather are instructed to practice paced breathing using a breathing pacer set to their resonance frequency, or 6 breaths/min if their resonance frequency is not known (a pace close to the mean resonance frequency across individuals). There are numerous free breathing pacers available for computer monitors and smart phones.

In Session 2, trainees are instructed in abdominal breathing, as well as pursed-lips breathing techniques, which makes exhalation less effortful (see Lehrer, Vaschillo and Vaschillo, 2000 for detailed instructions in these techniques).

In Session 3, participants are introduced to biofeedback for the first time. As such, rather than using a computerized pacer to guide their breathing they are instructed to use their cardiotachometer line displayed on a monitor in front of them. The cardiotachometer line represents their continually changing heart rate derived from the ECG or plethysmograph, that is, their dynamic HRV. When respiration-monitoring equipment is used, a separate line represents their breathing. Participants are told to maximally increase the range from peak to trough of the HRV waveform. If respiration-monitoring equipment is also being used, participants are instructed to make the breathing line match or overlap with the cardiotachometer line as best they can. Though providing a respiration measure can be helpful for patients learning the breathing technique, it is not necessary. In fact, few commonly available HRV BFB devices (including phone applications) actually include breathing measures.

Participants are further instructed how to practice resonance frequency breathing outside of session, either using handheld biofeedback devices, a computerized pacer set to their resonance frequency (if known), or a clock. Practice forms an important part of the training. Participants are instructed to practice ~20 min twice a day between sessions.

Sessions 4–10 focus on providing in-session guided practice, precisely defining trainees' resonance frequency, reinforcing HRV BFB home practices and clinical gains, as well as addressing problems or concerns as they arise.

A number of affordable and practical options now exist for clinicians wanting to deliver HRV BFB in session, and for individuals to practice at home. Prices for HRV BFB equipment ranges greatly. Research grade HRV BFB suites usually cost several thousand dollars. For most clinicians, however, such equipment will be wholly excessive. The sort of equipment required to do high quality clinical work can be purchased for several hundred US dollars. For home practice, there are a number of good portable HRV BFB devices on the market (usually sold as stress reducers) for ~$100 US. Recently, a very effective HRV BFB application for iPhone was released, which uses the phone's camera as a plethysmograph. The application costs $5 US.

Contraindications and risks associated with HRV BFB

Presently there are no known contraindications or risks associated with HRV BFB, although breathing at resonance frequency for more than a few hours a day may theoretically be iatrogenic. HRV BFB has been used safely to improve cardiac functioning post myocardial infarction, and in individuals with heart disease (Del Pozo, Gevirtz, Scher, & Guarneri, 2004; Nolan et al., 2005), as well as in the treatment of chronic obstructive pulmonary disease (Giardino et al., 2004).

HRV BFB's potential as a treatment for SUDs

Presently, only one study has investigated the efficacy of HRV BFB for the treatment of SUDs, though at the time of publication at least three clinical trials are known to be underway. Eddie Kim, Lehrer, Deneke, and Bates (2014) investigated the efficacy of a brief, 3-session HRV BFB intervention added to a traditional 28-day SUD inpatient treatment program, to reduce alcohol and drug craving. Forty-eight young adult men received either treatment as usual plus three sessions of HRV BFB training over three weeks, or treatment as usual only. Study participants receiving HRV BFB training were instructed to practice daily using a portable HRV BFB device. Findings showed that men receiving HRV BFB demonstrated a greater, medium effect size reduction in alcohol and drug craving compared to those receiving treatment as usual only (Cohen's $d = 0.35$), although this difference did not reach statistical significance ($p>0.05$).

In addition, an interaction effect was observed in analyses that accounted for baseline craving levels, wherein HRV levels at treatment entry were predictive of changes in craving in the treatment as usual group only. In this group, low baseline levels of HRV predicted increases in craving, whereas higher baseline HRV levels predicted greater decreases in craving from start to end of treatment. In the HRV BFB group, however, there was no such association. That is, HRV BFB appeared to dissociate individual differences in baseline HRV levels from changes in craving. These findings suggest that HRV BFB may prove to be an especially effective treatment component for reducing craving among individuals entering SUD treatment with low levels of HRV. However, individuals entering treatment with high levels of HRV may not benefit from this intervention in terms of reductions in craving.

These findings should be considered in light of the fact this pilot study utilized a small sample and lacked statistical power. It is also possible that larger, well-powered, trials may produce significant reductions in craving. It is also possible that HRV BFB may be effective outside of the confines of inpatient treatment to help individuals regulate themselves in-the-moment, when craving arises. Future studies should test longer treatment protocols, and monitor patients after discharge from treatment.

Conclusion

Although there is a growing literature on HRV BFB's efficacy for a number of disorders, no large-scale randomized controlled trials of HRV BFB for mental disorders have been conducted. As such, there is not yet sufficient empirical evidence to support delivering HRV BFB as a stand-alone treatment for any one psychological disorder. Replication of the observed results reviewed here with larger samples, and in diverse populations is necessary.

The lack of large-scale randomized controlled trials, however, does not invalidate the noteworthy findings discussed here. At present, the literature supports the use of HRV BFB as an adjunct treatment for a number of disorders, including major depression and PTSD. This is because HRV BFB has been shown to produce salutary effects, has no known adverse side effects, is non-toxic, free to practice, and can be used acutely or chronically depending on the needs of the patient. Given the apparent safety of the intervention, and its practicality, as well as the generally favourable feedback from study participants, there is no reason it should not be recommended to patients in treatment for SUDs.

HRV BFB, moreover, is not contraindicated with well-established treatments for SUDs. CBT, for example, teaches patients a variety of skills designed to be used strategically, and many CBT protocols already include some form of breathing exercise for self-regulation and/or relaxation. As such, interventions like HRV BFB, more specifically its core practice of resonance frequency breathing, is complimentary to CBT principles in that it provides patients with an additional tool to self-manage aversive symptomology, and potentially ameliorate underlying pathology. In addition, as with many CBT skills, it may be applied either acutely or chronically, depending on the needs of the client, and can be used interchangeably with other techniques. There is no reason too why resonance frequency breathing can't be used in the context of 12-step treatment, given it lends itself well to the cultivation of a meditation practice suggested in Step 11. Advances in technology also mean that HRV BFB can be practiced using hand-held devices, and more recently smart-phone applications. For these reasons, HRV BFB has real potential as an add-on intervention for established SUD treatment protocols, and deserves further investigation in clinical trials.

References

Agelink MW, Boz C, Ullrich H, Andrich J. Relationship between major depression and heart rate variability: Clinical consequences and implications for antidepressive treatment. Psychiatry Research. 2002; 113:139–149. [PubMed: 12467953]

Agelink MW, Majewski T, Wurthmann C, Postert T, Linka T, Rotterdam S, Klieser E. Autonomic neurocardiac function in patients with major depression and effects of antidepressive treatment with nefazodone. Journal of Affective Disorders. 2001; 62:187–198. [PubMed: 11223106]

APA. Diagnostic and statistical manual of mental disorders. 5th. Washington, DC: American Psychiatric Association; 2014.

Appelhans BM, Luecken LJ. Heart rate variability as an index of regulated emotional responding. Review of General Psychology. 2006; 10:229–240.

Baker TB, Piper ME, McCarthy DE, Majeskie MR, Fiore MC. Addiction motivation reformulated: An affective processing model of negative reinforcement. Psychological Review. 2004; 111:33–51. [PubMed: 14756584]

Benarroch EE. The central autonomic network: Functional organization, dysfunction, and perspective. Mayo Clinic Proceedings. 1993; 68:988–1001. [PubMed: 8412366]

Benarroch, EE. The central autonomic network. In: Low, PA., editor. Clinical autonomic disorders. 2nd. Philadelphia, PA: Lippincott-Raven; 1997. p. 17-23.

Benarroch EE. The arterial baroreflex. Neurology. 2008; 71:1733–1738. [PubMed: 19015490]

Berking M, Margraf M, Ebert D, Wupperman P, Hofmann SG, Junghanns K. Deficits in emotion-regulation skills predict alcohol use during and after cognitive-behavioral therapy for alcohol dependence. Journal of Consulting and Clinical Psychology. 2011; 79:307–318. [PubMed: 21534653]

Bibevski S, Dunlap ME. Evidence for impaired vagus nerve activity in heart failure. Heart Failure Reviews. 2011; 16:129–135. [PubMed: 20820912]

Bradley, SJ. Affect regulation and the development of psychopathology. New York, NY: The Guilford Press; 2003.

Britton A, Shipley M, Malik M, Hnatkova K, Hemingway H, Marmot M. Changes in heart rate and heart rate variability over time in middle-aged men and women in the general population (from the Whitehall II Cohort Study). The American Journal of Cardiology. 2007; 100:524–527. [PubMed: 17659940]

Card, JP.; Sved, AF. Central autonomic pathways. In: Llewellyn-Smith, IJ.; Verberne, AJM., editors. Central regulation of autonomic functions. New York, NY: Oxford University Press; 2011. p. 3-17.

Carney RM, Freedland KE, Stein PK, Skala JA, Hoffman P, Jaffe AS. Change in heart rate and heart rate variability during treatment for depression in patients with coronary heart disease. Psychosomatic Medicine. 2000; 62:639–647. [PubMed: 11020093]

Chambers AS, Allen JJB. Vagal tone as an indicator of treatment response in major depression. Psychophysiology. 2002; 39:861–864. [PubMed: 12462513]

Cheetham A, Allen NB, Yücel M, Lubman DI. The role of affective dysregulation in drug addiction. Clinical Psychology Review. 2010; 30:621–634. [PubMed: 20546986]

Cohen H, Benjamin J, Geva AB, Matar MA, Kaplan Z, Kotler M. Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: Application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. Psychiatry Research. 2000; 96:1–13. [PubMed: 10980322]

Cooper ML, Frone MR, Russell M, Mudar P. Drinking to regulate positive and negative emotions: A motivational model of alcohol use. Journal of Personality and Social Psychology. 1995; 69:990–1005. [PubMed: 7473043]

Cox, WM.; Klinger, E., editors. Handbook of motivational counseling. Chichester, UK: Wiley-Blackwell; 2011. A motivational model of alcohol use: Determinants of use and change; p. 131-158.

Critchley HD. Neural mechanisms of autonomic, affective, and cognitive integration. Journal of Comparative Neurology. 2005; 493:154–166. [PubMed: 16254997]

Critchley HD. Psychophysiology of neural, cognitive and affective integration: fMRI and autonomic indicants. International Journal of Psychophysiology. 2009; 73:88–94. [PubMed: 19414044]

Del Pozo JM, Gevirtz RN, Scher B, Guarneri E. Biofeedback treatment increases heart rate variability in patients with known coronary artery disease. American Heart Journal. 2004; 147:545.

Diamond LM, Aspinwall LG. Emotion regulation across the life span: An integrative perspective emphasizing self-regulation, positive affect, and dyadic processes. Motivation and Emotion. 2003; 27:125–156.

Eddie D, Kim C, Lehrer P, Deneke E, Bates ME. A pilot study of brief heart rate variability biofeedback to reduce craving in young adult men receiving inpatient treatment for substance use disorders. Applied Psychophysiology and Biofeedback. 2014; 39:181–192. [PubMed: 25179673]

Everitt BJ, Belin D, Economidou D, Pelloux Y, Dalley JW, Robbins TW. Neural mechanisms underlying the vulnerability to develop compulsive drug-seeking habits and addiction. Philosophical Transactions of the Royal Society B: Biological Sciences. 2008; 363:3125–3135.

Forgas JP. Affect and cognition. Perspectives on Psychological Science. 2008; 3:94–101. [PubMed: 26158876]

Franken IHA. Drug craving and addiction: Integrating psychological and neuropsychopharmacological approaches. Progress in Neuro-Psychopharmacology and Biological Psychiatry. 2003; 27:563–579. [PubMed: 12787841]

Friedman BH, Thayer JF. Autonomic balance revisited: Panic anxiety and heart rate variability. Journal of Psychosomatic Research. 1998; 44:133–151. [PubMed: 9483470]

Garland EL, Carter K, Ropes K, Howard MO. Thought suppression, impaired regulation of urges, and Addiction-Stroop predict affect-modulated cue-reactivity among alcohol dependent adults. Biological Psychology. 2012; 89:87–93. [PubMed: 21967855]

Giardino ND, Chan L, Borson S. Combined heart rate variability and pulse oximetry biofeedback for chronic obstructive pulmonary disease: Preliminary findings. Applied Psychophysiology and Biofeedback. 2004; 29:121–133. [PubMed: 15208975]

Ginsberg JP, Berry ME, Powell DA. Cardiac coherence and posttraumatic stress disorder in combat veterans. Alternative Therapies in Health and Medicine. 2009; 16:52–60.

Goldberger AL, Peng CK, Lipsitz LA. What is physiologic complexity and how does it change with aging and disease? Neurobiology of Aging. 2002; 23:23–26. [PubMed: 11755014]

Goldstein DS, Bentho O, Park MY, Sharabi Y. Low-frequency power of heart rate variability is not a measure of cardiac sympathetic tone but may be a measure of modulation of cardiac autonomic outflows by baroreflexes. Experimental Physiology. 2011; 96:1255–1261. [PubMed: 21890520]

Gorka SM, Ali B, Daughters SB. The role of distress tolerance in the relationship between depressive symptoms and problematic alcohol use. Psychology of Addictive Behaviors. 2011; 26:621–626. [PubMed: 22121919]

Gorman JM, Sloan RP. Heart rate variability in depressive and anxiety disorders. American Heart Journal. 2000; 140:77–83. [PubMed: 11011352]

Gross JJ. The emerging field of emotion regulation: An integrative review. Review of General Psychology. 1998; 2:271–299.

Hallman DM, Olsson EMG, von Schéele B, Melin L, Lyskov E. Effects of heart rate variability biofeedback in subjects with stress-related chronic neck pain: A pilot study. Applied Psychophysiology and Biofeedback. 2011; 36:71–80. [PubMed: 21365308]

Hasin DS, Fenton MC, Beseler C, Park JY, Wall MM. Analyses related to the development of DSM-5 criteria for substance use related disorders: 2. Proposed DSM-5 criteria for alcohol, cannabis, cocaine and heroin disorders in 663 substance abuse patients. Drug and Alcohol Dependence. 2012; 122:28–37. [PubMed: 21963333]

Hassett AL, Radvanski DC, Vaschillo E, Vaschillo B, Sigal LH, Karavidas MK, et al. Lehrer P. A pilot study of the efficacy of heart rate variability (HRV) biofeedback in patients with fibromyalgia. Applied Psychophysiology and Biofeedback. 2007; 32:1–10. [PubMed: 17219062]

Henriques G, Keffer S, Abrahamson C, Horst SJ. Exploring the effectiveness of a computer-based heart rate variability biofeedback program in reducing anxiety in college students. Applied Psychophysiology and Biofeedback. 2011; 36:101–112. [PubMed: 21533678]

Ingjaldsson JT, Laberg JC, Thayer JF. Reduced heart rate variability in chronic alcohol abuse: Relationship with negative mood, chronic thought suppression, and compulsive drinking. Biological Psychiatry. 2003; 54:1427–1436. [PubMed: 14675808]

Jellinger KA. Central autonomic network: Functional organization and clinical correlations. European Journal of Neurology. 1998; 5:216–216.

Karavidas MK, Lehrer PM, Vaschillo E, Vaschillo B, Marin H, Buyske S, et al. Hassett A. Preliminary results of an open label study of heart rate variability biofeedback for the treatment of major depression. Applied Psychophysiology and Biofeedback. 2007; 32:19–30. [PubMed: 17333315]

Katsamanis M, Lehrer PM, Escobar JI, Gara MA, Kotay A, Liu R. Psychophysiologic treatment for patients with medically unexplained symptoms: A randomized controlled trial. Psychosomatics. 2011; 52:218–229. [PubMed: 21565593]

Katz, AM. Physiology of the heart. Lippincott Williams & Wilkins; 2010.

Kawachi I, Sparrow D, Vokonas PS, Weiss ST. Decreased heart rate variability in men with phobic anxiety (data from the Normative Aging Study). The American Journal of Cardiology. 1995; 75:882–885. [PubMed: 7732994]

Kim S, Zemon V, Cavallo MM, Rath JF, McCraty R, Foley FW. Heart rate variability biofeedback, executive functioning and chronic brain injury. Brain Injury. 2013; 27:209–222. [PubMed: 23384218]

Klein E, Cnaani E, Harel T, Braun S, Ben-Haim SA. Altered heart rate variability in panic disorder patients. Biological Psychiatry. 1995; 37:18–24. [PubMed: 7893854]

Koob GF, Le Moal M. Drug addiction, dysregulation of reward, and allostasis. Neuropsychopharmacology. 2001; 24:97–129. [PubMed: 11120394]

Le Moal M, Koob GF. Drug addiction: Pathways to the disease and pathophysiological perspectives. European Neuropsychopharmacology. 2007; 17:377–393. [PubMed: 17169534]

Lehrer P, Eddie D. Dynamic processes in regulation and some implications for biofeedback and biobehavioral interventions. Applied Psychophysiology and Biofeedback. 2013; 38:143–155. [PubMed: 23572244]

Lehrer P, Smetankin A, Potapova T. Respiratory sinus arrhythmia biofeedback therapy for asthma: A report of 20 unmedicated pediatric cases using the Smetankin method. Applied Psychophysiology and Biofeedback. 2000; 25:193–200. [PubMed: 10999237]

Lehrer P, Vaschillo E, Lu SE, Eckberg D, Vaschillo B, Scardella A, Habib R. Heart rate variability biofeedback: Effects of age on heart rate variability, baroreflex gain, and asthma. Chest. 2006; 129:278–284. [PubMed: 16478842]

Lehrer P, Vaschillo E, Vaschillo B. Resonant frequency biofeedback training to increase cardiac variability: Rationale and manual for training. Applied Psychophysiology and Biofeedback. 2000; 25:177–191. [PubMed: 10999236]

Lehrer P, Vaschillo E, Vaschillo B, Lu SE, Scardella A, Siddique M, Habib RH. Biofeedback treatment for asthma. Chest Journal. 2004; 126:352–361.

Levenson RW. Blood, sweat, and fears. Annals of the New York Academy of Sciences. 2003; 1000:348–366. [PubMed: 14766648]

Lin G, Xiang Q, Fu X, Wang S, Wang S, Chen SA, et al. Wang T. Heart rate variability biofeedback decreases blood pressure in prehypertensive subjects by improving autonomic function and baroreflex. The Journal of Alternative and Complementary Medicine. 2012; 18:143–152. [PubMed: 22339103]

McCraty R, Atkinson M, Lipsenthal L, Arguelles L. New hope for correctional officers: An innovative program for reducing stress and health risks. Applied Psychophysiology and Biofeedback. 2009; 34:251–272. [PubMed: 19466540]

Meule A, Freund R, Skirde AK, Vögele C, Kübler A. Heart rate variability biofeedback reduces food cravings in high food cravers. Applied Psychophysiology and Biofeedback. 2012; 37:241–251. [PubMed: 22688890]

Miller WR, Walters ST, Bennett ME. How effective is alcoholism treatment in the United States? Journal of Studies on Alcohol and Drugs. 2001; 62:211–220.

Moravec CS. Biofeedback therapy in cardiovascular disease: Rationale and research overview. Cleveland Clinic Journal of Medicine. 2008; 75(Suppl 2):S35–S38. Review. [PubMed: 18540144]

Nahshoni E, Aravot D, Aizenberg D, Sigler M, Zalsman G, Strasberg B, et al. Weizman A. Heart rate variability in patients with major depression. Psychosomatics. 2004; 45:129–134. [PubMed: 15016926]

Nolan RP, Kamath MV, Floras JS, Stanley J, Pang C, Picton P, Young QR. Heart rate variability biofeedback as a behavioral neurocardiac intervention to enhance vagal heart rate control. American Heart Journal. 2005; 149:1137.

O'Brien CP, McLellan AT. Myths about the treatment of addiction. The Lancet. 1996; 347:237–240.

Paliwal P, Hyman SM, Sinha R. Craving predicts time to cocaine relapse: Further validation of the Now and Brief versions of the cocaine craving questionnaire. Drug and Alcohol Dependence. 2008; 93:252–259. [PubMed: 18063320]

Pessoa L. On the relationship between emotion and cognition. Nature Reviews Neuroscience. 2008; 9:148–158. [PubMed: 18209732]

Peterson JB, Pihl RO, Seguin JR, Finn PR, Stewart SH. Heart-rate reactivity and alcohol consumption among sons of male alcoholics and sons of non-alcoholics. Journal of Psychiatry and Neuroscience. 1993; 18:190–198. [PubMed: 8373755]

Pincus SM, Goldberger AL. Physiological time-series analysis: What does regularity quantify? American Journal of Physiology-Heart and Circulatory Physiology. 1994; 266:H1643–H1656.

Porges SW. The polyvagal theory: New insights into adaptive reactions of the autonomic nervous system. Cleveland Clinic Journal of Medicine. 2009; 76(Suppl. 2):S86–S90. [PubMed: 19376991]

Prinsloo GE, Rauch HG, Lambert MI, Muench F, Noakes TD, Derman WE. The effect of short duration heart rate variability (HRV) biofeedback on cognitive performance during laboratory induced cognitive stress. Applied Cognitive Psychology. 2011; 25:792–801.

Quintana DS, Guastella AJ, McGregor IS, Hickie IB, Kemp AH. Heart rate variability predicts alcohol craving in alcohol dependent outpatients: Further evidence for HRV as a psychophysiological marker of self-regulation. Drug and Alcohol Dependence. 2013; 132:395–398. [PubMed: 23601925]

Quintana DS, McGregor IS, Guastella AJ, Malhi GS, Kemp AH. A meta-analysis on the impact of alcohol dependence on short-term resting-state heart rate variability: Implications for cardiovascular risk. Alcoholism: Clinical and Experimental Research. 2013; 37(Suppl. 1):E23–E29.

Robinson TE, Berridge KC. The incentive sensitization theory of addiction: Some current issues. Philosophical Transactions of the Royal Society of London - Series B: Biological Sciences. 2008; 363:3137–3146. [PubMed: 18640920]

Siepmann M, Aykac V, Unterdorfer J, Petrowski K, Mueck-Weymann M. A pilot study on the effects of heart rate variability biofeedback in patients with depression and in healthy subjects. Applied Psychophysiology and Biofeedback. 2008; 33:195–201. [PubMed: 18807175]

Simons JS, Carey KB, Wills TA. Alcohol abuse and dependence symptoms: A multidimensional model of common and specific etiology. Psychology of Addictive Behaviors. 2009; 23:415–427. [PubMed: 19769426]

Sinha R. New findings on biological factors predicting addiction relapse vulnerability. Current Psychiatry Reports. 2011; 13:398–405. [PubMed: 21792580]

Song HS, Lehrer PM. The effects of specific respiratory rates on heart rate and heart rate variability. Applied Psychophysiology and Biofeedback. 2003; 28:13–23. [PubMed: 12737093]

Sowder E, Gevirtz R, Shapiro W, Ebert C. Restoration of vagal tone: A possible mechanism for functional abdominal pain. Applied Psychophysiology and Biofeedback. 2010; 35:199–206. [PubMed: 20229150]

Tan G, Dao TK, Farmer L, Sutherland RJ, Gevirtz R. Heart rate variability (HRV) and posttraumatic stress disorder (PTSD): A pilot study. Applied Psychophysiology and Biofeedback. 2011; 36:27–35. [PubMed: 20680439]

Thayer JF, Brosschot J. Psychosomatics and psychopathology: Looking up and down from the brain. Psychoneuroendocrinology. 2005; 30:1050–1058. [PubMed: 16005156]

Thayer, JF.; Hansen, AL.; Johnsen, BH. Handbook of behavioral medicine. New York: Springer Science; 2010. The non-invasive assessment of autonomic influences on the heart using impedance cardiography and heart rate variability.

Thorberg FA, Lyvers M. Negative mood regulation (NMR) expectancies, mood, and affect intensity among clients in substance disorder treatment facilities. Addictive Behaviors. 2006; 31:811–820. [PubMed: 15993006]

Thurber MR, Bodenhamer-Davis E, Johnson M, Chesky K, Chandler CK. Effects of heart rate variability coherence biofeedback training and emotional management techniques to decrease music performance anxiety. Biofeedback. 2010; 38:28–40.

Tiffany ST, Wray J. The continuing conundrum of craving. Addiction. 2009; 104:1618–1619. [PubMed: 19558571]

Udupa K, Sathyaprabha TN, Thirthalli J, Kishore KR, Lavekar GS, Raju TR, Gangadhar BN. Alteration of cardiac autonomic functions in patients with major depression: A study using heart rate variability measures. Journal of Affective Disorders. 2007; 100:137–141. [PubMed: 17113650]

Vanderlei LCM, Pastre CM, Hoshi RA, Carvalho TD, Godoy MF. Basic notions of heart rate variability and its clinical applicability. Revista Brasileira de Cirurgia Cardiovascular. 2009; 24:205–217. [PubMed: 19768301]

Vaschillo E, Lehrer P, Rishe N, Konstantinov M. Heart rate variability biofeedback as a method for assessing baroreflex function: A preliminary study of resonance in the cardiovascular system. Applied Psychophysiology and Biofeedback. 2002; 27:1–27. [PubMed: 12001882]

Vaschillo, E.; Vaschillo, B.; Buckman, JF.; Pandina, RJ.; Bates, ME. The Investigation and Clinical Significance of Resonance in the Heart Rate and Vascular Tone Baroreflexes; Paper presented at the BIOSTEC-3rd International Joint Conference; Valencia, Spain. 2011.

Vaschillo E, Vaschillo B, Lehrer PM. Characteristics of resonance in heart rate variability stimulated by biofeedback. Applied Psychophysiology and Biofeedback. 2006; 31:129–142. [PubMed: 16838124]

Verheul R, van den Brink W, Geerlings P. A three-pathway psychobiological model of craving for alcohol. Alcohol and Alcoholism. 1999; 34:197–222. [PubMed: 10344781]

Weiss F. Neurobiology of craving, conditioned reward and relapse. Current Opinion in Pharmacology. 2005; 5:9–19. [PubMed: 15661620]

Weise F, Müller D, Krell D, Kielstein V, Koch RD. Heart rate variability in chronic alcoholics: A follow-up study. Drug and Alcohol Dependence. 1986; 17:365–368. [PubMed: 3757771]

Wells R, Outhred T, Heathers JAJ, Quintana DS, Kemp AH. Matter over mind: A randomised-controlled trial of single-session biofeedback training on performance anxiety and heart rate variability in musicians. PLoS One. 2012; 7:e46597. [PubMed: 23056361]

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Wills TA, Cleary S, Filer M, Shinar O, Mariani J, Spera K. Temperament and self-control related to early-onset substance use. Prevention Science. 2001; 2:145–163. [PubMed: 11678291]

Witkiewitz K, Villarroel NA. Dynamic association between negative affect and alcohol lapses following alcohol treatment. Journal of Consulting and Clinical Psychology. 2009; 77:633–644. [PubMed: 19634957]

Zhang X, Abdel-Rahman A, Wooles WR. Impairment of central mediation of the arterial baroreflex by acute ethanol administration. Alcohol. 1988; 5:221–228. [PubMed: 3415767]

Zucker TL, Samuelson KW, Muench F, Greenberg MA, Gevirtz RN. The effects of respiratory sinus arrhythmia biofeedback on heart rate variability and post-traumatic stress disorder symptoms: A pilot study. Applied Psychophysiology and Biofeedback. 2009; 34:135–143. [PubMed: 19396540]