
STUDYDADDY
Get Homework Help
From Expert Tutor
Get Help

The Relationship Between Intentional Self-Injurious Behavior and the Loudness Dependence of Auditory Evoked Potential in Research Volunteers

Angelika Marsic,[1] Mitchell E. Berman,[2] Tammy D. Barry,[1]
and Michael S. McCloskey[3]

[1] *The University of Southern Mississippi*
[2] *Mississippi State University*
[3] *Temple University*

Objective: Serotonergic (5-HT) functioning has been shown to be inversely associated with intentional self-injurious behaviors. The purpose of this study was to examine the association between three related self-report measures of intentional self-injurious behaviors (suicidal thoughts/behavior, history of nonsuicidal self-injury, history of severe self-harm when angry) and a putative electrophysiological index of 5-HT activity, the loudness dependence of auditory evoked potential (LDAEP). **Method:** Auditory evoked potentials were recorded from 41 men (mean age = 20.69, standard deviation [*SD*] = 2.98) during the administration of various tone loudness stimuli, followed by completion of the self-report measures. **Results:** The component slope was associated with all measures of self-injurious behavior in the expected direction. **Conclusion:** The LDAEP has the potential to be used as a noninvasive index of intentional self-harm disposition. Additional studies are needed using other populations, including women and treatment-seeking individuals, to determine if the LDAEP more broadly discriminates risk of self-injuring. J. Clin. Psychol. 71:250–257, 2015.

Keywords: Self-injury; 5-HT functioning; LDAEP; N1/P2; EEG

The prevalence rate for intentional self-injurious behavior (SIB) ranges from 1% to 4% in adults (Briere & Gil, 1998; Klonsky, Oltmanns, & Turkheimer, 2003; Prinstein, 2008) and from 17% to 38% in college students, with lifetime prevalence estimates of 35% (e.g., Gratz, 2001; Whitlock, Eckenrode, & Silverman, 2006). Not unexpectedly, the rates of SIB are even higher in clinical populations (21%–61% in adolescents and young adults and 21% in adults; e.g., Briere & Gil, 1998; Darche, 1990; DiClemente, Ponton, & Hartley, 1991; Prinstein, 2008). The most severe form of SIB—suicide—is the second leading cause of death among 25- to 34-year-olds, the third leading cause of death among 15- to 24-year-olds, and the 11th leading cause of death overall in the United States (Centers for Disease Control and Prevention [CDC], 2007). There are approximately 100–200 attempts for every completed suicide among young adults aged 15–24 years (Goldsmith, Pellmar, Kleinman, & Bunney, 2002).

One of the most extensively studied biological correlates of SIB is serotonergic (5-HT) neurotransmitter activity. Specifically, attenuated 5-HT functioning has been associated with SIBs across the spectrum of lethality (e.g., Arango et al., 1990; Audenaert et al., 2001; Kamali, Oquendo, & Mann, 2001; Malone, Corbitt, Li, & Mann, 1996; McCloskey, Ben-Zeev, Lee, Berman, & Coccaro, 2009). For example, a meta-analysis by Lester (1995) reviewed 27 neurochemical studies of the association between 5-HT and SIB involving 1202 psychiatric patients and controls. The results provided strong evidence for the role of serotonin in suicidal behavior. Individuals who had attempted suicide had lower levels of cerebral spinal fluid (CSF) 5-hydroxyindoleacetic acid (5-HIAA; a 5-HT metabolite) compared to psychiatric controls. Asberg (1997) reviewed 33 studies and found that low levels of CSF 5-HIAA were associated with

Please address correspondence to: Angelika Marsic, 3264 Willowbrook Avenue, Palmdale, CA 93551. E-mail: Angelika.Marsic@gmail.com

Published online in Wiley Online Library (wileyonlinelibrary.com/journal/jclp). DOI: 10.1002/jclp.22136

suicidality in unipolar depression and personality disorders. Diminished levels of CSF 5-HIAA have also been found in depressed patients with a high-lethality suicide attempt compared to depressed individuals with a low-lethality suicide attempt (Mann & Malone, 1997). Furthermore, lower CSF 5-HIAA levels have been found in individuals engaging in nonlethal SIBs (López-Ibor, Saiz-Ruiz, & Pérez de los Cobos, 1985).

Commonly used biological indexes of central 5-HT functioning can be costly and invasive (e.g., lumbar puncture; pharmacochallenge). However, a neurophysiological approach that takes advantage of electrical brain wave activity measured at the scalp (the loudness dependence of the auditory evoked potential: LDAEP; Hegerl & Juckel, 1993) may provide a noninvasive means to assess behaviorally relevant central 5-HT functioning. Although auditory evoked potentials are generated by a complex interrelationship of different neurotransmitters, there is mounting evidence that the LDAEP is most likely modulated by serotonergic system activities (Hegerl & Juckel, 1993). The LDAEP is a measure of auditory cortex activity as represented by the auditory evoked potential slopes (Hegerl, Gallinat, & Juckel, 2001). The intensity dependence of the auditory evoked N1/P2 component (i.e., dB level of the tone dependence) has been proposed to be inversely related to central serotonergic activity. That is, low serotonergic innervation of the auditory cortex ostensibly produces a more pronounced LDAEP N1/P2 component (i.e., increased N1/P2 amplitude to increasing intensity tones) and vice versa (Hegerl & Juckel, 1993).

In humans, the N1/P2 component comprises two overlapping subcomponents generated by the superior temporal plane (mainly primary auditory cortex) and the lateral temporal gyri (secondary auditory cortex; Hegerl & Juckel, 1993). The N1/P2 component, occurring about 70–200 ms poststimulus, is used as a combined ratio parameter because it has higher loudness dependence reliability than when loudness dependence is measured separately for N1 and P2.

In addition, the relationship with clinical features and personality factors is stronger with the loudness dependence of the combined parameter than with individual amplitudes (Hegerl, Gallinat, & Mrowinski, 1994). The N1/P2 component also exhibits prominent and stable interindividual differences. For example, Hegerl, Prochno, Ulrich, and Muller-Oerlinghausen (1988) found test-retest reliability of .77 for the Cz site (i.e., midline position of the central lobe) and .74 for the amplitude/stimulus intensity function (ASF) slope among healthy participants. ASF reflects the N1/P2 amplitude changes as the tone intensity increase. Hegerl and Juckel (1993) reported a test-retest correlation of .90 for the intraindividual stability of the intensity dependence N1/P2 component, mainly generated by the activity of the primary auditory cortex.

The experimental evidence for a relationship between the LDAEP and 5-HT was first observed in animals (Hegerl et al., 1993; Juckel, Molnar, Hegerl, Csepe, & Karmos, 1997). However, experimental studies in humans (i.e., using pharmacological agents to augment serotonin levels in the brain) of the LDAEP as an index of acute 5-HT changes have yielded mixed results. For example, 5-HT augmentation in a double-blind, placebo-controlled study was shown to produce a significant decrease in N1/P2 slope with increasing tone loudness, lending support for the validity of the LDAEP as a 5-HT index (Nathan, Segrave, Phan, O'Neill, & Croft, 2006). Follow-up studies with healthy participants have failed to replicate these findings (Guille et al., 2008; Uhl et al., 2006), indicating that, at least in healthy subjects, the LDAEP may not be a good indicator of *acute* changes in central 5-HT activity.

The lack of evidence for acute changes in 5-HT activity as a function of pharmacochallenge with 5-HT agents does not preclude the use of the LDAEP as a valid biological indicator in vulnerable individuals. Several clinical studies have found a strong LDAEP in individuals characterized by psychiatric disorders ostensibly marked by 5-HT dysfunction. For example, Gallinat, Bottlender, and Juckel (2000) found that a significantly higher number of depressive patients fell into a strong LDAEP group (seemingly reflecting attenuated 5-HT activity), and that those same individuals exhibited a significant decrease in depressive symptoms after a selective serotonin reuptake inhibitor (SSRI) treatment compared to depressive patients with a less prominent LDAEP.

Hegerl and colleagues (1998) found that patients with high levels of serotonin syndrome (i.e., enhanced central 5-HT activity) exhibited a weaker LDAEP than those with low serotonin syndrome. Chen and colleagues (2005) found a sharper LDAEP slope in a depression–suicide group as opposed to a depression–nonsuicidal group, demonstrating a potential utility of the LDAEP in discriminating suicidality among depressed individuals (Chen et al., 2005). Based on these findings, O'Neill, Croft, and Nathan (2008) concluded that although evidence for the LDAEP as an indicator of *acute* serotonergic changes among humans is conflicting in nature, evidence for the LDAEP as a useful biological index of 5-HT functioning in vulnerable individuals is more compelling.

The purpose of this study was to examine the relationship between intentional SIBs and the LDAEP in a sample of young adults. If the LDAEP reflects relatively stable central 5-HT activity, an association between the LDAEP slope and measures of SIBs should emerge. To date no study has examined the relationship between the LDAEP N1/P2 slope and intentional SIBs in a nonclinical population. Given that previous studies have found that an increase in the auditory evoked N1/P2 component slope with increasing tone loudness (i.e., strong LDAEP) is inversely related to indexes of central serotonergic activity, it was expected that strong LDAEP would be positively related to various measures of SIB.

Method

Participants

Forty-one men recruited from undergraduate classes took part in the study. The majority of the participants self-identified as Caucasian (56.1%), followed by African American (39%) and "other" (4.9%) race or ethnicity. Participants ranged in age from 18 to 31 years (mean [M] = 20.69, SD = 2.98). A history of schizophrenia or mood, anxiety, or substance dependence disorder was exclusionary. In addition, any hearing impairment, a history of seizures, and a history of traumatic brain injury were exclusionary. Potential participants were excluded if they were currently under medical treatment. Participants were asked to not consume alcohol or caffeinated beverages in the 24 hours before the study day. The study was reviewed and approved by the Institutional Review Board for the Protection of Human Subjects.

Measures

Health Screening Questionnaire. A brief health screening questionnaire was created for the current study, including items on the participant's age, gender, and race. Along with this demographic information, items addressing the health and psychiatric exclusion criteria listed above were included.

Suicidal Behaviors Questionnaire (SBQ; Cole, 1988). The SBQ is a four-item self-report measure that assesses suicidal thoughts, plans, and behavior. The SBQ questions are as follows: "Have you ever thought about or attempted to kill yourself?"; "How often have you thought about killing yourself in the past year?"; "Have you ever told someone that you were going to commit suicide, or that you might do it?"; "How likely is it that you will attempt suicide one day?" Items are rated on a Likert-format scale, with values ranging from 0–6, 0–4, 0–2, and 0–4, respectively. Scores range from 0 to 16 (with higher scores implying greater suicidal disposition). The SBQ has adequate internal consistency ($\alpha = .80$) for a nonclinical sample and good test-retest stability over time ($r = .95$; Cotton, Peters, & Range, 1995).

Furthermore, the SBQ has good construct validity, as shown by a significant positive correlation ($r = .69$) with the Scale for Suicidal Ideation in a nonclinical sample (Cotton et al., 1995) and with laboratory measures of self-aggression (Berman & Walley, 2003). Internal consistency for the current sample was adequate ($\alpha = .71$).

Deliberate Self-Harm Inventory (DSHI; Gratz, 2001). The DSHI is a 17-question, self-report scale of nonsuicidal SIBs. The DSHI comprises NSSI behaviors that do not have the goal of ending one's life (e.g., self-cutting, burning, scratching, biting, and punching); items include, for example, "Have you ever intentionally (i.e., on purpose) carved words into your skin?" and "Have you ever intentionally (i.e., on purpose) used bleach, comet, or oven cleaner to scrub your skin?" Individuals endorse Yes or No for each item. A DSHI total score is obtained by summing the number of endorsed self-harm behaviors. The DSHI has shown adequate internal consistency (α = .82) and test-retest stability (*r* = .92; Gratz, 2001). Adequate correlations with related self-report measures of self-harm behaviors have been found (e.g., DSHI and the self-harm items on the Mental Health History Form, *r* = .49; Gratz, 2001). DSHI internal consistency for the current sample was also adequate (α = .84).

The Life History of Aggression Scale-Self-Aggression subscale (LHA-SA; Coccaro, Berman, & Kavoussi, 1997). The LHA is an 11-item measure of past aggressive, self-aggressive, and antisocial behaviors. The LHA assesses the frequency and intensity of these behaviors, rather than aggressive traits or ideation, and it provides information about these from age 13 on. For the current study, we used the self-report two-item Self-Aggression subscale of the LHA (e.g., "Deliberately tried to physically hurt yourself in anger or desperation" and "Deliberately tried to end your life or kill yourself in anger or desperation"), which is rated on a 6-point scale, ranging from 0 (*no occurrences*) to 5 (*more events than can be counted*), reflecting the total number of occurrences. The LHA-SA was found in previous studies to have somewhat low internal consistency (α = .45) due to gender differences (females, α = .71; males, α = .18) but had adequate inter-rater agreement (*r* = .84) and test-retest reliability (*r* = .97; Coccaro et al., 1997). LHA-SA internal consistency for the men in this study, however, was adequate (α = .72).

LDAEP. LDAEP stimulus presentation, data acquisition, and analyses were accomplished using equipment and software obtained from the James Long Company, a 16-channel custom optically isolated bioamplifier. LDAEPs were recorded with 15 electrodes arranged according to the 10–20 electroencephalogram (EEG) electrode system, using M1 as a reference and AFz as ground. Impedances were kept below 5 kΩ throughout the testing. Pure sinus tones (1000 Hz, some with 100 ms duration with 10 ms rise and 10 ms fall time, and some with some with 50 ms duration with 10 ms rise and 10 ms fall time, inter stimulus interval (ISI) randomized between 1800 and 2200 ms) of five intensities (60, 70, 80, 90, 100 dB) were presented biaurally in a pseudorandomized form by headphones.

Data were collected with a sampling rate of 500 Hz and an analogous bandpass filter (0.16–50 Hz). Seventy sweeps of each stimulus intensity and time duration were presented (700 sweeps in all, with 350 sweeps of 50 ms tone duration, and 350 sweeps of 100 ms tone duration). Poststimulus peak latencies were determined between 80–120 ms for N1 and 150–230 ms for P2 components.

Procedure

Upon arrival, participants completed the informed consent process, after which a brief screening interview was administered. If the participant did not meet any exclusionary criteria, then he was instructed to complete the demographic questionnaire, which was computer administered. Next, the participant was prepared for the EEG recording. An appropriately sized electrocap comprising 15 electrodes, following the International 10–20 system, was fitted on the participant's head. The scalp was prepared by application of a mildly abrasive gel (OmniPrep). Electrooculography (EOG) electrodes were placed on the outer canthi of each eye and on the supraorbital and infraorbital ridge of the left eye, to allow for detection and removal of ocular artifacts.

According to lab standards, each electrode site displayed impedance of less than 5 kΩ, while the impedance on the EOG sites were kept at less than 10 kΩ. The left mastoid electrode site was used as a reference site during the collection phase. However, during the analysis, the right mastoid was averaged with the left mastoid to serve as the final reference to avoid the left or right hemisphere bias that is often found when using just one reference site (Luck, 2005).

The participant was instructed to refrain from moving his eyes during testing to ensure minimal contamination of the data. Specifically, a fixation point was displayed on the screen for the duration of the EEG experiment, and the participant was asked to softly focus on that point and refrain from any eye movement other than regular blinking. In addition, the participant was asked to refrain from making any body movements. After the EEG data collection, the participant completed the self-report measures of SIB (SBQ, SHI, LHA-SA). Finally, the participant was debriefed and psychology course research credit was applied.

Results

EEG Analysis

Prior to analyzing the N1 and P2 amplitudes, a grand mean waveform for each electrode site was created. Based on visual inspection of the grand mean waveform and findings from previous research, appropriate latency time intervals were determined (Hegerl & Juckel, 1993). Previous research demonstrated that the N1/P2 amplitude is most pronounced at the Cz site. Therefore, our analysis used the Cz site to be consistent with previous research studies. N1 amplitudes were determined by computing the average amplitude between a latency of 80 and 120 ms. P2 amplitudes were determined by computing the average amplitude between a latency of 150 and 230 ms. The N1/P2 amplitude was calculated as the difference between N1 and P2 (P2-N1) at the Cz site. Finally, the N1/P2 slope for each participant was calculated using tone intensity as the independent variable and N1/P2 amplitude as the dependent variable. Individual slopes were used in the bivariate analyses.

Statistical Analyses

As would be expected, the frequency of reported thoughts and behaviors differed across measures. For the SBQ (which on face is strongly weighted to assessing suicidal thoughts), 16 of 41 participants positively endorsed at least one item. The rates for actual nonsuicidal SIBs on the DSHI were somewhat lower, with 11 of 41 participants endorsing at least one behavior. Rates were lowest for the LHA-SA, which assesses more serious forms of intentional self-harm behavior. Specifically, 7 of 41 participants positively endorsed one of the two LHA-SA items (six endorsed "Deliberately tried to physically hurt yourself in anger or desperation" and three endorsed "Deliberately tried to end your life or kill yourself in anger or desperation" at some time after 13 years of age).

Spearman rho bivariate analyses tested one-tailed at alpha = .05 revealed that the measures of SIB were significantly correlated with one another. Specifically, SBQ scores were associated with scores on the DSHI ($r = .53$, $p = .003$) and the LHA-SA ($r = .49$, $p = .001$). Moreover, scores on the DSHI and the LHA-SA were strongly correlated ($r = .53$, $p < .001$). Exploratory analysis for the two LHA-SA items revealed that deliberate physical self-harm and deliberate attempt to end one's life were also associated ($r = .46$, $p = .001$).

The LDAEP index (N1/P2 slopes) was positively correlated with scores on the SBQ ($r = .31$, $p = .03$), the DSHI ($r = .42$, $p = .003$), and the LHA-SA ($r = .32$, $p = .02$). Exploratory analyses for the two LHA-SA items and N1/P2 slopes revealed a nonsignificant association in the expected direction for deliberate self-harm when angered ($r = .26$, $p = .052$). Interestingly, despite the small proportion of participants with a history of actual suicide attempts in the sample, the LDAEP index was significantly related to this most lethal index of self-injury ($r = .36$, $p = .01$). Therefore, the overall pattern of associations suggests that higher slopes of N1/P2 amplitudes are related to higher scores on measures of SIBs across the spectrum of lethality.

Discussion

The LDAEP has been proposed as a putative, reliable, noninvasive index of 5-HT functioning in the central nervous system (Hegerl et al., 1994, 2001). Although an increasing number of studies has examined the relationship between the LDAEP and central serotonergic functioning in individuals with more serious psychopathologies marked by serotonin dysfunction (e.g., depression), to our knowledge this is the first study to date that has explored the relationship between the LDAEP and a history of SIB in a nonclinical population.

Consistent with expectations, the LDAEP slope (with greater slopes indicative of attenuated 5-HT functioning) was positively associated with various indexes of SIB (including suicidal thoughts, nonsuicidal self-injury, and past suicide attempts), suggesting that the N1/P2 slope LDAEP index can identify SIB in a nonclinical population. The present findings support the notion that the LDAEP could potentially be used to prospectively discriminate individuals who are at risk of self-harming. This possibility can be tested in future studies by recruiting individuals with a notable history of self-injurious or suicidal behaviors and individuals who are experiencing urges currently and conducting LDAEP assessments across time.

Limitations

These findings also provide qualified evidence that the LDAEP is potentially a useful index of 5-HT functioning. However, this suggestion should be interpreted with caution, taking into consideration the study limitations. First, as with most event related potential (ERP) studies, the sample size was relatively small. Second, this study exclusively relied on self-report measures of lifetime SIB. Behavioral measures of SIB (e.g., Implicit Association Tasks or self-shock tasks such as the Self-Aggression Paradigm; Berman & Walley, 2003) and information from third-party sources would provide a more comprehensive approach to understanding the boundaries of the relationship between the LDAEP and SIBs.

Future studies should incorporate community and clinical samples, including men and women, which are more representative of people who engage in SIBs. Although the participants were screened for psychopathology, no measures were administered assessing subclinical levels of internalizing symptoms (e.g., depression and anxiety) that may have mediated the SIB–LDAEP association. Importantly, we did not use a structured or semistructured interview to fully assess various forms of psychopathology associated with self-harm, such as personality disorders.

Because it is an indirect measure, the LDAEP provides no information about specific 5-HT receptors or areas of the brain that have 5-HT innervations outside the auditory cortex. In the current study, we analyzed the LDAEP from the Cz electrode (as per previous research) instead of using dipole source analysis, which would allow for the examination of the LDAEP generated only in the primary auditory cortex. However, the differences between the two measurement techniques have not been found to be large (Nathan et al., 2006).

Although the LDAEP is putatively associated with serotonergic functioning, we do not know what specific features of cognitive functioning it reflects. It is also important to note that serotonergic dysfunction is not specific to SIBs; therefore, multiple measures in addition to the LDAEP will likely be needed to predict future self-harm. SIB is a multidetermined behavior (i.e., associated with numerous concurrent risk factors and performing numerous functions; see Garlow et al., 2008; Gratz, 2002; Prinstein, 2008), which means that no single instrument will ever suffice in identifying individuals at risk of SIB.

Conclusion

Despite these limitations, the present findings have implications in the terms of broad clinical application. Serotonergic dysfunction plays a role in a variety of psychological disturbances. However, progress in identification of emotional and behavioral disturbances, ostensibly marked by low 5-HT functioning, has been hindered by the lack of reliable and noninvasive indicators of central 5-HT functioning (Wutzler et al., 2008). The LDAEP may prove to be a cost-effective, reliable, noninvasive biological index for assessing central serotonergic activity.

References

Arango, V., Ernsberger, P., Marzuk, P. M., Chen, J. S., Tierney, H., Stanley, M., . . . Mann, J. J. (1990). Autoradiographic demonstration of increased serotonin 5-HT2 and beta-adrenergic receptor binding sites in the brain of suicide victims. Archives of General Psychiatry, 47, 1038–1047.

Asberg, M. (1997). Neurotransmitters and suicidal behaviour: The evidence from cerebrospinal fluid studies. In D. M. Stoff & J. J. Mann (Eds.). The neurobiology of suicide from the bench to the clinic (pp. 158–181). New York: New York Academy of Sciences.

Audenaert, K., van Laere, K., Dumont, F., Slegers, G., Mertens, J., van Heeringenl, C., & Dierck, R. A. (2001). Decreased frontal serotonin 5-HT2a receptor binding index in deliberate self-harm patients. European Journal of Nuclear Medicine, 28, 175–182.

Berman, M. E., & Walley, J. C. (2003). Imitation of self-aggressive behavior: An experimental test of the contagion hypothesis. Journal of Applied Social Psychology, 33, 1036–1057.

Briere, J., & Gil, E. (1998). Self-mutilation in clinical and general population samples: Prevalence, Correlates, and Functions. American Journal of Orthopsychiatry, 68, 609.

Centers for Disease Control and Prevention. (2007). Web-based injury statistics query and reporting system (WISQARS). Retrieved from: www.cdc.gov/injury/wisqars/index.html

Chen, T. J., Yu, Y. W. Y., Chen, M. C., Wang, S. Y., Tsai, S. J., & Lee, T. W. (2005). Serotonin dysfunction and suicide attempts in major depressives: An auditory event-related potential study. Neuropsychobiology, 52, 28–36.

Coccaro, E. F., Berman, M. E., & Kavoussi, R. J. (1997). Assessment of life-history of aggression: Development and psychometric characteristics. Psychiatry Research, 73, 147–157.

Cole, D. A. (1988). Hopelessness, social desirability, depression, and parasuicide in two college student samples. Journal of Consulting and Clinical Psychology, 56, 131–136.

Cotton, C. R., Peters, D. K., & Range, L. M. (1995). Psychometric properties of the Suicidal Behaviors Questionnaire. Death Studies, 19, 391–397.

Darche, M. (1990). Psychological factors differentiating self-mutilating and non-self-mutilating adolescent inpatient females. Psychiatric Hospital, 21, 31–35.

DiClemente, R., Ponton, L., & Hartley, D. (1991). Prevalence and correlates of cutting behavior: Risk for HIV transmission. Journal of the American Academy of Child & Adolescent Psychiatry, 30, 735–739.

Gallinat, J., Bottlender, R., & Juckel, G. (2000). The loudness dependence of the auditory evoked N1/P2-component as a predictor of the acute SSRI response in depression. Psychopharmacology, 148, 404–411.

Garlow, S. J., Rosenberg, J., Moore, J. D., Haas, A. P., Koestner, B., Hendin, H., & Nemeroff, C. B. (2008). Depression, desperation, and suicidal ideation in college students: Results from the American Foundation for Suicide Prevention College Screening Project at Emory University. Depression and Anxiety, 25, 482–488.

Goldsmith, S. K., Pellmar, T. C., Kleinman, A. M., & Bunney, W. E. (2002). Reducing suicide: A national imperative. Washington (DC): National Academy Press.

Gratz, K. L. (2001). Measurement of deliberate self-harm: Preliminary data on the Deliberate Self-Harm Inventory. Journal of Psychopathology and Behavioral Assessment, 23, 253–263.

Guille, V., Croft, R. J., O'Neill, B. V., Illic, S., Phan, K. L., & Nathan, P. J. (2008). An examination of acute changes in serotonergic neurotransmission using the loudness dependence measure of auditory cortex evoked activity: effects of citalopram, escitalopram and sertraline. Human Psychopharmacology, 23, 231–241.

Hegerl, U., Bottlender, R., Gallinat, J., Kuss, H. J., Ackenheil, M., & Möller, H. J. (1998). The serotonin syndrome scale: First results on validity. European Archives of Psychiatry, 248, 96–103.

Hegerl, U., Gallinat, J., & Juckel, G. (2001). Event-related potentials: Do they reflect central serotonergic neurotransmission and do they predict clinical response to serotonin agonists? Journal of Affective Disorders, 62, 93–100.

Hegerl, U., Gallinat, J., & Mrowinski, D. (1994). Intensity dependence of auditory evoked dipole source activity. International Journal of Psychophysiology, 17, 1–13.

Hegerl, U., & Juckel, G. (1993). Intensity dependence of auditory evoked potentials as an indicator of central serotonergic neurotransmission: A new hypothesis. Biological Psychiatry, 33, 173–187.

Hegerl, U., Prochno, I., Ulrich, G., & Muller-Oerlinghausen, B. (1988). Are auditory evoked potentials suitable for predicting the response to lithium prophylaxis? A study on the effects of repeated measurement, age, gender, and personality on the amplitude/stimulus intensity function in healthy volunteers. Pharmacopsychiatry, 21, 329–330.

Juckel, G., Molnar, M., Hegerl, U., Csepe, V., & Karmos, G. (1997). Auditory-evoked potentials as indicator of brain serotonergic activity–First evidence in behaving cats. Biological Psychiatry, 41, 1181–1195.

Kamali, M., Oquendo, M. A., & Mann, J. J. (2001). Understanding the neurobiology of suicidal behavior. Depression and Anxiety, 14, 164–176.

Klonsky, E. D., Oltmanns, T. F., & Turkheimer, E. (2003). Deliberate self-harm in a nonclinical population: Prevalence and psychological correlates. American Journal of Psychiatry, 160, 1501–1508.

Lester, D. (1995). The concentration of neurotransmitter metabolites in the cerebrospinal fluid of suicidal individuals: A meta-analysis. Pharmacopsychiatry 28, 77–79.

López-Ibor, J., Saiz-Ruiz, J., & Pérez de los Cobos, J. (1985). Biological correlations of suicide and aggressivity in major depressions (with melancholia): 5-hydroxyindoleacetic acid and cortisol in cerebral spinal fluid, dexamethasone suppression test and therapeutic response to 5-hydroxytryptophan. Neuropsychobiology, 14, 67–74.

Luck, S. J. (2005). An introduction to the event-related potential technique. Cambridge, MA: The MIT Press.

Malone, K. M., Corbitt, E. M., Li, S., & Mann, J. J. (1996). Prolactin response to fenfluramine and suicide attempt lethality in major depression. British Journal of Psychiatry, 168, 324–329.

Mann, J. J., & Malone, K. M. (1997). Cerebrospinal fluid amines and higher lethality suicide attempts in depressed inpatients. Biological Psychiatry, 41, 162–171.

McCloskey, M. S., Ben-Zeev, D., Lee, R., Berman, M. E., & Coccaro, E. F. (2009). Acute tryptophan depletion and self-injurious behavior in aggressive patients and healthy volunteers. Psychopharmacology, 203, 53–61.

Nathan, P. J., Segrave, R., Phan, K. L., O'Neill, B., & Croft, R. J. (2006). Direct evidence that acutely enhancing serotonin with the selective serotonin reuptake inhibitor citalopram modulates the loudness dependence of the auditory evoked potential (LDAEP) marker of central serotonin function. Human Psychopharmacological Clinical Experiments, 21, 47–52.

O'Neill, B., Croft, R., & Nathan, P. (2008). The loudness dependence of the auditory evoked potential (LDAEP) as an in vivo biomarker of central serotonergic function in humans: Rationale, evaluation and review of findings. Human Psychopharmacology: Clinical & Experimental, 23, 355–370.

Prinstein, M. (2008). Introduction to the special section on suicide and nonsuicidal self-injury: A review of unique challenges and important directions for self-injury science. Journal of Consulting and Clinical Psychology, 76, 1–8.

Uhl, I., Gorynial, J., Gallinatl, J., Mulert, C., Wutzler, A., Heinz, A., & Juckel, G. (2006). Is the loudness dependence of auditory evoked potentials modulated by the selective serotonin reuptake inhibitor citalopram in healthy subjects? Human Psychopharmacological Clinical Experiments, 21, 463–471.

Whitlock, J., Eckenrode, J., & Silverman, D. (2006). Self-injurious behaviors in a college population. Pediatrics, 117, 1939–1948.

Wutzler, A., Winter, C., Kitzrow, W., Idun, U., Wolf, R. J., Heinz, A., & Juckel, G. (2008). Loudness dependence of auditory evoked potentials as indicator of central serotonergic neurotransmission: Simultaneous electrophysiological recordings and in vivo in the rat primary auditory cortex. Neuropsychopharmacology, 33, 3176–3181.