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Benefits of EEG-Neurofeedback on the Modulation of Impulsivity in a Sample of Cocaine and Heroin Long-Term Abstinent Inmates: A Pilot Study

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Abstract

The aim of this pilot study was to assess whether neurofeedback (NFB) can be useful in the treatment of impulsive behavior in long-term abstinent cocaine and heroin addicts. A single-blind sham-controlled NFB protocol was carried out to assess the effects of NFB on impulsivity in 20 (10 + 10) cocaine and heroin long-term abstinent addicts (*Diagnostic and Statistical Manual of Mental Disorders* [4th ed., text rev.; *DSM-IV-TR*]). Psychotic and neurologic diseases were excluded. Participants underwent 40 NFB sessions based on the very slow cortical potential range. Inhibitory deficits were specifically addressed through right and left prefrontal training. Clinical improvement was measured with Likert-type scales, the Hamilton Depression Rating Scale, and the State–Trait Anxiety Inventory, and impulsivity was assessed using the Barratt Impulsiveness Scale and the Continuous Performance Test. Although the results are preliminary due to the small sample size, the NFB-treated group showed a significant clinical improvement, including symptoms of anxiety and depression, with two differentiated time periods. No significant clinical improvement was found in the control group. A significant decrease in the post- versus pre-treatment measures

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of global impulsivity, nonplanning impulsivity, and error commission measures was found in the NFB-treated group; effect size (d_{Korr}) in the pre–post control design was moderate. No significant change was found in the control group. Despite the limitations of this study, the results suggest that NFB is better than placebo in improving impulsivity and clinical symptoms of anxiety and depression in long-term abstinent cocaine- and heroin-dependent individuals.

Keywords
impulsivity, anxiety, depressive symptoms, arousal, opiates, cocaine, dependence, neurofeedback

Introduction

Impulsivity plays a crucial role in cocaine and heroin dependence (Moeller et al., 2002; Ortal et al., 2015; Rodríguez-Cintas et al., 2016; Roncero et al., 2013; Valero et al., 2014), associated to poor treatment outcomes (Coffey et al., 2003; Poling et al., 2007) and relapses even after long-term abstinence (Laudet, 2007; Winhusen et al., 2013). Among substance-dependent individuals, poly-drug addicts are more impulsive than those dependent on single substances (Bornovalova et al., 2005; Boyle, 1993; Clarke et al., 2012; McCown, 1988; Verdejo-García et al., 2007). In drug-dependent individuals, impulsivity is usually both a premorbid characteristic that predisposes to addiction and a consequence of the consumption. In this regard, siblings of stimulant-dependent individuals also exhibit significantly decreased levels of inhibitory control, suggesting that impulsivity may be a trait predisposing to addiction (Ersche et al., 2013), a view that is also supported by animal models of cocaine addiction (Dalley et al., 2011) and neuroimaging studies in human beings (Makris et al., 2004). Impulsivity is also a risk factor among opiate-dependent people (Kirby et al., 1999; Rodríguez-Cintas et al., 2016; Tolomeo et al., 2016). On the contrary, impulsivity is a strong predictor of criminal offending (Loeber et al., 2012).

Impulsivity (or impulsiveness) is a tendency to act with a swift action, displaying behavior characterized by little or no forethought, reflection, or consideration of the consequences (Moeller et al., 2002). Impulsivity is a complex and multidimensional construct, which includes different trait and behavioral instruments. The majority of research in this field at a clinical level rely on self-reported questionnaires such as the Barratt Impulsiveness Scale (BIS), which is a measure of trait impulsivity that identifies three different components: attentional impulsiveness which refers to the tendency to make quick decisions and a diminished ability to focus on tasks, motor impulsiveness which refers to a tendency to act without thinking, and nonplanning impulsiveness referring to a lack of "futuring" or forethought (Barratt, 1967; Patton et al., 1995). On the contrary, different instruments to measure behavioral impulsivity have been developed, and these are thought to be better for neurobiological studies. Behavioral instruments include two dimensions: those measuring impulsive actions or disinhibition, and those measuring impulsive choice associated to impulsive decision making

(Winstanley et al., 2006, 2010). One of the most widely used behavioral tests of motor impulsivity is the Continuous Performance Test (CPT), which is also a test of attentional functions (Rosvold et al., 1956; Winstanley et al., 2010). It is not always possible to establish a direct relationship between the dimensions of trait impulsivity obtained from the BIS-11 and the psychological processes measured in behavioral paradigms. Despite that, there is agreement that the concept of impulsive action fits well into the BIS-11 factor of motor impulsivity. In contrast, the concept of impulsive choice appears to span both the cognitive/attentive and nonplanning domains of the BIS-11 (Patton et al., 1995; Winstanley et al., 2006, 2010), and it is related to lack of consideration of the consequences or "myopia for the future" (Damasio, 1994).

Emotional states such as anxiety, anger, sadness, or joy are associated with impulsivity (Chester et al., 2016) and linked to physiological arousal (Kreibig, 2010), both enhancing impulsive reactions. The relationship between impulsivity, anxiety, and depressive mood is complex and might be moderate by age (Moustafa et al., 2017). In addition, there is evidence that anxious individuals with high impulsivity (but not low impulsivity) experience strong craving after alcohol cue exposure (Adams et al., 2019). In opioid-dependent individuals, anxiety mediated the relationship between intolerance of uncertainty and impulsivity (Garami et al., 2017). From the neurobiological point of view, higher depressive symptoms and impulsivity were significantly associated with reduced cortical thickness in different regions of the prefrontal cortex (PFC), including ventromedial PFC/medial OFC (orbitofrontal cortex), although there was no significant association between anxiety symptoms and brain structures (Merz et al., 2018).

In chronic cocaine-dependent individuals, there is consistent evidence of structural (Tanabe et al., 2009) and functional abnormalities in prefrontal regions, especially in orbitofrontal areas (London et al., 2000; Stapleton et al., 1995; Volkow et al., 1992; Volkow & Fowler, 2000). Increased activity of the striatum and limbic regions relative to prefrontal areas (Hu et al., 2015) has also been reported. Similar deficits have been found in heroin-abstinent addicts (Tolomeo et al., 2016). These abnormalities are at least partially neurophysiological in nature and involve changes in the electroencephalogram (EEG; Fingelkurts et al., 2006; Franken et al., 2004; Prichep et al., 1996; Roemer et al., 1995) and evoked potentials (Cadaveira et al., 1994). As most of the brain areas associated to drug addiction overlap with those related to impulsivity (Bechara et al., 2000; Dalley et al., 2011; Winstanley et al., 2006), therapeutic approaches addressed to improve brain function can be useful in ameliorating impulsivity in difficult populations, such as cocaine and heroin addicts.

One of these approaches is EEG-neurofeedback (NFB), a form of biofeedback designed to learn how to enhance certain types of EEG activity and to decrease others, aiming to improve brain activity and hence behavioral, cognitive, and emotional self-regulation (Hammond, 2010; Johnstone et al., 2005). So far, different NFB approaches have been used to improve impulsivity in distinct populations. In children and adults with attention deficit hyperactivity disorder (ADHD), reinforcement of the sensorimotor rhythm (SMR; Fuchs et al., 2003) and the theta/beta NFB protocol (Bluschke et al., 2016) have shown to be useful in improving impulsive responses, with effects lasting

more than 6 months (Leins et al., 2007). Arns and coworkers used quantitative EEG (QEEG)–based NFB to improve clinical symptoms in people with ADHD (Arns et al., 2012; Arns, Feddema, & Kenemans, 2014). Birbaumer and colleagues (Birbaumer et al., 1990; Heinrich et al., 2007) developed a different kind of NFB based on slow cortical potentials (SCPs), which also improve behavioral symptoms of ADHD, including impulsivity (Gevensleben et al., 2009; Strehl et al., 2006). In addition, psychopathic offenders can also improve their impulsive behavior by training the SCP (Konicar et al., 2015). NFB has also been reported to be useful in the treatment of drug addiction. In this regard, Scott et al. informed an improvement of impulsivity, an increase in abstinence rates, and treatment retention in a group of poly-substance-dependent patients (Scott et al., 2005). In cocaine-dependent patients, a combination of NFB and motivation treatment lowered EEG reactivity to drug-related images (Horrell et al., 2010). NFB was also useful in the treatment of methamphetamine abuse (Rostami & Dehghani-Arani, 2015) and in opiate-dependent patients (Dehghani-Arani et al., 2013).

A relatively recent NFB design is the Othmer method, which is founded on evidence-based NFB protocols and combines three components: (a) the classic frequency band with individual inhibits that work up to 40 Hz; (b) very low frequencies, the so-called infralow frequencies (ILFs), which work in the very slow cortical frequency range; and (c) the bipolar approach which gives feedback to the brain on how the two regions work relative to each other. In addition, in this method, the learning process is not guided by the EEG changes but by improvement of clinical symptoms (Othmer, 2015, 2016; Othmer et al., 2013). The aim of this pilot study was to assess whether NFB, based on the Othmer method, can be useful for the treatment of impulsive behavior in cocaine and heroin long-term abstinent individuals. Two components, physiological arousal and prefrontal regulatory control, were specifically addressed to improve impulsivity. We hypothesized that the improvement of clinical symptoms, including measures of arousal, anxiety or mood, and impulsivity, would be higher in the NFB-treated group relative to the control group.

Materials and Methods

Participants

The clinical sample consisted of 20 inmates who were recruited from a whole group of 43 inmates from a module for specialized treatment (MST) in Prison Center Brians-2. Selected individuals were poly-addicts with long-term abstinence (between 8 and 30 months without consumption) according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000), and all of them had failures in their previous attempts to be reinstated into society as a result of their addiction. The diagnosis of addiction and other psychiatric comorbidities were based on medical and psychiatric forensic records. The inmates were recruited by the educators in the prison, according to the information of the official forensic records (“RisCanvi”; Pueyo, 2013) and according to the inclusion and exclusion criteria set in this NFB protocol.

All participants were undergoing regular urine tests to assure abstinence, before and during the whole protocol. They were currently smoking between 15 and 30 cigarettes in a day. The Inclusion criteria were men, aged between 22 and 52 years old, with a history of cocaine and heroin addiction as well as a history of delinquent behavior, and associated to robbery with or without violence. Robbery behavior was chosen because it is the most frequent offense among cocaine and heroin addicts, and it is often driven by the need to get money for their consumption. Other kind of delinquent behavior was excluded to have the sample characteristics as homogeneous as possible. Other exclusion criteria were as follows: (a) a lifetime history of psychotic or bipolar diseases, (b) current personality disorders including psychopathic personality, (c) organic illness, (d) an IQ estimated to be under 90, (e) delinquent behavior different from robbery, and (f) unwillingness to sign the informed consent. The psychiatric diagnosis relied on the medical prison records and currently confirmed by a forensic psychiatrist.

The investigation was carried out in accordance with the ethical principles of latest version of the Declaration of Helsinki and was approved by the Ethics Committee of *Universitat Autònoma de Barcelona* (UAB). Written informed consent was obtained from all the participants after the nature of the procedures had been fully explained.

Study Design

A single-blind sham-controlled design has been used and the inmates were blinded to the treatment group where they were assigned. For group assignment, psychopathological variables from the official forensic records ("RisCanvi"; Pueyo, 2013) that included the whole history in prison and had been updated 2 to 3 months before starting this protocol were used. These psychopathological variables included impulsivity, aggressive behavior, anxiety, depressive symptoms, and emotional instability. All these variables were defined in the forensic protocols and were scored in a 3-point scale (low, middle, high). The forensic psychologist was blinded to the NFB groups. On the contrary, a trained psychologist associated to NFB carried out a first clinical interview to assess the same variables using the same measure range. The inmates, who received the same value in all those mentioned variables, were paired together and then assigned randomly to one of the two treatment groups. Ten inmates were included in the NFB group, and 10 were included in the control group. The inmates were blinded to the treatment group during the whole process.

Each inmate in the NFB and control groups underwent 40 sessions of 30 min each at a rate of two sessions per week, and the treatment lasted for 5 months. The sham group was designed to incorporate all the elements of the NFB group, including the same NFB equipment and EEG devices.

Clinical Measures

Impulsivity was assessed with two different instruments: the BIS-11 which includes 30 items organized into three subscales (Attentional, Motor, and Nonplanning) and a global measure (global-BIS) (Barratt et al., 1997; Patton et al., 1995) for trait impulsivity, and the commission-error subtests of the computerized "continuous performance

test" (QIK Test CPT, Bee Medic GmbH, Technologies for Mental Health) as a measure of behavioral impulsivity. The State–Trait Anxiety Inventory (STAI) was used to evaluate anxiety (Speilberger & Vagg, 1984). Finally, the Hamilton Depression Rating Scale (HDRS) was used to assess depressive symptoms (Hamilton, 1960). All these measures were administered twice, before and immediately after the treatment, to support the assessment of clinical symptoms.

The Othmer method is entirely based on the improvement of clinical symptoms. Seven categories of symptoms were included and the questions were as follows: (a) Insomnia (Have you had any difficulties falling asleep and awakening in the middle of the night?), (b) Anxiety (Have you felt any of these symptoms: heart palpitations, digestive, respiratory, sweating?), (c) Depressive mood (Do you feel sad, hopeless, or helpless?), (d) Mood instabilities (easy changes of humor along the day), (e) Irritability (Do you feel angry, or have you argued or got angry with other inmates?), (f) Impulsivity (Have you acted without thinking, or have you done or said something that has had or could have had negative consequences for you?), and (g) Attention/concentration (Have you had any difficulties paying attention in any of your current tasks, in class, when reading, or when playing with other inmates?). These items were recorded throughout the NFB treatment at the beginning of each session, and the inmates were invited to report how they were feeling and to score their symptoms in Likert-type scales in a range from 10 to zero (Othmer, 2015).

During the development of the protocol, all the inmates were undergoing regular drug testing to assure abstinence.

NFB Instrumentation and Protocol

The NeuroAmp II® (CE Class IIa, FDA Class II) with two-channel EEG amplifier, integrated with the Cygnet® 2.0, working with an Infralow – HD (ILF-HD) module (frequencies range from 0.01 mHz to 40 Hz), has been used for NFB. The Cygnet software performed the acquisition and the analysis of the EEG for training purposes. The whole system ran by means of the Windows 8 operating system, using a standard personal computer desktops and high-resolution monitors. Bipolar montage, active and reference electrodes, and right mastoid as ground position were used in all cases for EEG recording. Electrodes were labeled according to the 10–20 system and placed consecutively at P4–T4, T3–T4, T4–Fp2, and T3–Fp1, as reported in the procedure. Impedances were kept under 5 kΩ throughout the sessions, and artifacts including eye movements electrooculogram (EOG) and muscle tension were automatically removed by the Cygnet software. Sintered silver/silver chloride electrodes were attached individually to the scalp after preparation with Nu-Prep and using standard paste (Ten20 Conductive).

The difference signal (active minus reference or equivalently reference minus active) was displayed as EEG and spectral on the screen of the professional. Two classic elements of the NFB have been used for feedback purposes, the *reward* and the *inhibits*. The reward frequency reflects the frequency-selective filtering of the SCP, and the optimal reward frequency (ORF) for training was operatively set at the

Figure 1. This figure shows (left image) the Cygnet screen with the recording electroencephalogram signal, and the inhibit band at the bottom of the figure. Superimposed to the screen, there is the image of the game which is part of the participant's training. This screen presents a thematic video material with the feedback signal encoded as the size of the video. The right image presents another thematic video the participants watch during the training in which a rocket moves at a velocity that is directly determined by the signal level. The inhibit bands are included at the bottom of the image.

beginning of the training sessions between 0.01 and 0.02 mHz, for all the inmates. The reward signal is displayed as changes in the speed or size of the elements used in the feedback display. The *inhibits* included eight to 10 separate filter blocks in fixed frequency steps in the range between 1 and 40 Hz that worked automatically and responded to rapid and inappropriate brief events of EEG amplitude increase. All the individual inhibit bands were combined into an overall *inhibit* that influenced the feedback game display. The effect of the *inhibits* was to disrupt or limit the game display (a black fog, the picture whites out, etc.) and works according to the conditioned reinforcing model in which the brain is expected to learn to keep amplitudes under the thresholds. The diversity of ways to give feedback is important to maintain the brain engaged and attending to all these fluctuations while the participant is engaged with the thematic material (see Figure 1 for an example of video game). For both the reward and inhibit schemes, the average percentage above/below threshold ("percent success") can be set; in this protocol, percent success was set at 95%. Both the reward frequency and the *inhibits* are integrated in the video games working with the Cygnet, so that continuous feedback is displayed on the clients' screen during the training.

Each training session lasted for 30 min, and the position of the electrode was changed along within this period, along the 40 sessions, as follows: (a) the starting placement was P4-T4 and lasted for seven sessions of 30 min each; (b) keeping the P4-T4 position, seven sessions of interhemispheric training T3-T4 were added, so that 15 min of each session were for training at P4-T4 and 15 min at T3-T4; (c) we introduced 14 sessions of the right prefrontal T4-Fp2, so that the training time was divided by the three electrode positions (P4-T4 7 min, T3-T4 7 min, and T4-Fp2 15 min); and finally, (d) we introduced the left prefrontal, T3-Fp1, during the rest of the protocol, and the training times were P4-T4 (7 min), T3-T4 (7 min), T4-Fp2 (7 min), and T3-Fp1 (7 min) (see Figure 2).

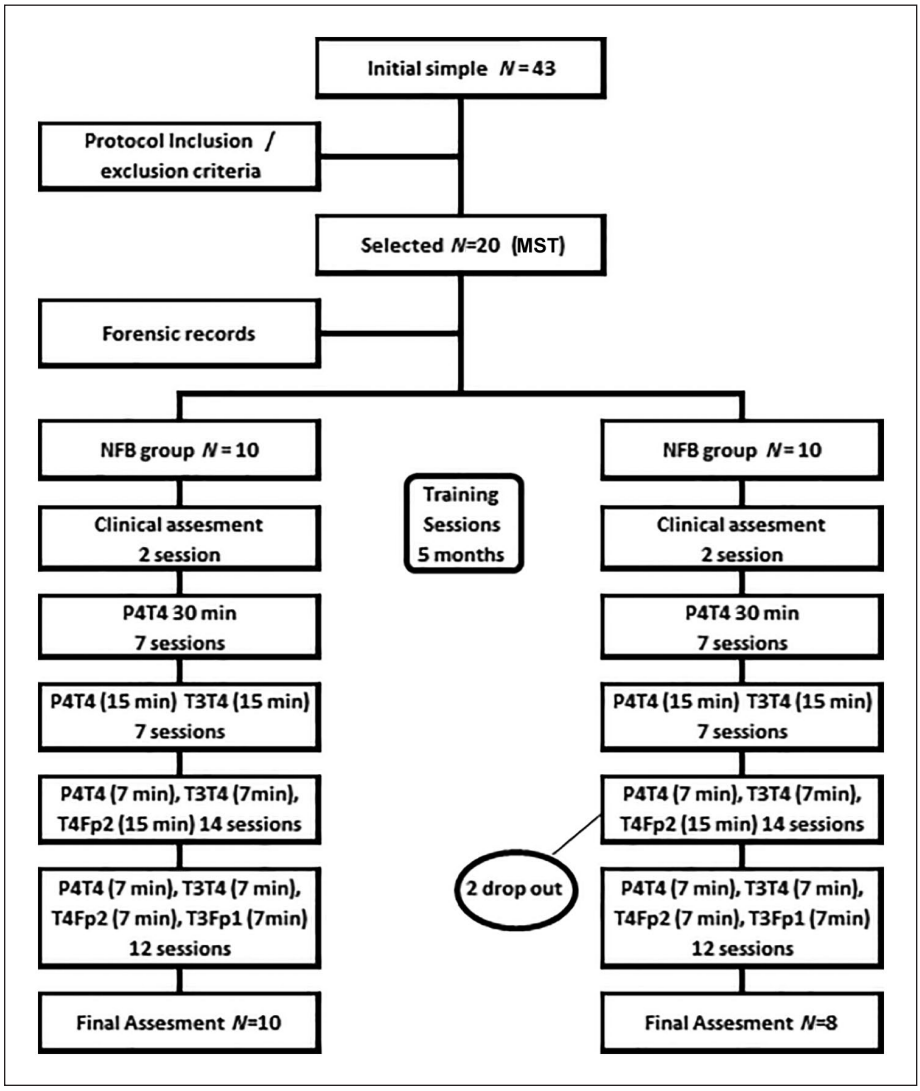

Figure 2. Flow diagram including participant selection and the different training sessions and electrode placements.
Note. MST = module for specialized treatment; NFB = neurofeedback.

For the sham feedback, a second NeuroAmp II® (CE Class IIa, FDA Class II) with two-channel EEG amplifier, integrated with the Cygnet® 2.0, was used. In this case, the EEG being recorded was not connected to the Cygnet software so that the feedback lacked the active core component associated to the EEG activity. The video games used were Dreamscapes series and a computer expert prepared the video games to be

used throughout the sessions in a similar way as the video games used in the real feedback. In actual fact, no manipulation of the video imagery was involved and the fluctuations appeared random. In the sham group, the electrodes were allocated at the same places and following the same sequence than in the NFB group (see Figure 2).

Procedure

This protocol has been carried out in a module for special treatment needs (MST). The clinical assessment lasted for 2 days immediately before the start of the NFB treatment. On the first day, the clinical questionnaires (BIS-11, STAI, and HDRS) and the CPT were administered. A clinical psychologist assisted the inmates in completing the questionnaires to guarantee the reliability of the information. On the second day, clinical symptoms including insomnia, physical anxiety, depressive mood, mood instabilities, irritability, impulsivity, and attention/concentration were assessed and scored with Likert-type scales. In addition, these clinical symptoms were also recorded at the beginning of each treatment session just before NFB training, throughout the study. Both the NFB and sham groups were carried out by the same psychologist in the same physical environment, and all of them received the same instructions. During the sessions, all the participants were seated in a comfortable chair within a quiet room. After the electrodes were attached to the scalp and impedances were checked, the instructions were given to the participants: "Please pay attention to the elements in the screen. Do not do any special effort; just pay a relaxed attention. Enjoy the session." The participants were also asked to pay attention to their feelings and sensations during the session and to inform the psychologist if they noticed any discomfort in their body or in their brain. At the beginning of the first session, they were allowed to see their brain waves and how they change after a muscular movement or eye blink; this information also helped in keep their attention and their interest in the sessions.

The task for the psychologist responsible for the training was to follow the participant's reactions, to assure they kept involved in the sessions. The psychologist was also responsible for changing electrode position at the right moment (see Figure 2). By attending the NFB protocol, all the inmates (NFB and sham groups) received 2 extra hours per week of treatment, relative to the rest of the inmates not included in this protocol. This gave the inmates an extra benefit and contributed to maintain the interest in attending the protocol, even those included in the control group. At the end of the treatment, the clinical questionnaires (BIS-11, STAI, and HDRS) and the CPT were administered again with the help of a clinical psychologist.

Statistical Analyses

To study the differences between the NFB and control groups in age and pattern of cocaine/heroin abuse, Mann–Whitney *U* test was executed to compare the differences between the NFB and control groups. The Likert-type scores for each clinical symptom (including insomnia, anxiety symptoms, depressive mood, mood instabilities, irritability, impulsivity, and attention/concentration) were averaged for each treatment session. The

resulting value "mean clinical state" was analyzed using repeated-measures analysis of variance (ANOVA) with two factors (the control and NFB treatment groups) as between-participant factor, and "mean clinical state" at each time point (Mcs; 10 levels) as within-participant repeated-measures factor. For this analysis, 10 time points were used, that is, only one in four data were included in the analysis and two values per month along the 5 months of the treatment were introduced in the analysis. The Greenhouse–Geisser method was used to correct the degrees of freedom when the assumption of sphericity was not met, and post hoc Bonferroni corrections for multiple comparisons were applied. The *mean clinical state* along the treatment period was used to calculate the slope to compare treatment-associated changes. To study differences in "mean clinical state" between two time periods—Mcs1 and Mcs4 and between Mcs4 and Mcs10—and changes after treatment in anxiety measures (STAI) and depressive symptoms (HDRS) in both the NFB and control groups, the Wilcoxon test was applied.

To assess the changes in BIS measures (pre- vs. post-treatment), the nonparametric Wilcoxon test was applied for each subscale of the BIS-11 and the commission-error measure of the CPT, in each treatment group. Mann–Whitney *U* test was executed to compare the differences between the NFB and control groups and applied to each subscale of the BIS-11 and the commission-error measure of the CPT. Effect size was calculated (Klauer, 2001). Confidence intervals for statistical significance were always set at 5%. In all cases, the statistical package for social sciences (SPSS), version 20, has been used.

Results

All the individuals completed the 40 sessions of the treatment except for two of the inmates in the sham group that were excluded from the protocol for behavioral reasons associated with their roles in the institution. The rest of the inmates behaved during the whole protocol according to what the institution expected from them, and all of them continued until the completion of the protocol.

Demographic and Clinical Characteristics

There were no differences between the NFB and control groups in age, sociodemographic, or educational status. From the clinical point of view, 40% of the individuals in the NFB treatment group and 30% of the inmates in the control group were HIV positive. None of the participants had any psychiatric disease other than remittent drug addiction. The inmates reported their first contact with drugs at a very early age (between 8 and 17 years old); they had been poly-drug users, including cocaine, heroin, amphetamines, alcohol, and cannabis; and some of them started their drug use with inhalants. One of them had declared as alcohol addict, but the rest of the inmates announced themselves just as alcohol users. Currently, all of the inmates reported to have being abstinent from any drug of abuse except tobacco. There were no group differences regarding the age of their first contact with drugs, the age of starting abuse/dependence of cocaine and/or heroin, or the number of years of cocaine/heroin abuse/

Table 1. Mean and Standard Deviation for Clinical Variables of the NFB and Control Groups Before the NFB Treatment. Z scores and significance values when comparing the NFB and control groups are also reported. NFB = neurofeedback; COC = cocaine; HER = heroin; STAI = State–Trait Anxiety Inventory; HDRS = Hamilton Depression Rating Scale.

Parameter	Control *M* (*SD*)	NFB *M* (*SD*)	*Z*	*p*
Age	39.05 (6.7)	37.1 (5.7)	−1.100	.272
Education	Primary school	Primary school	—	—
Sociodemographic status	Low	Low	—	—
Estimated IQ	>90	>90	—	—
First contact with drugs (age)	11.3 (3.6)	10.4 (2.8)	0.213	.763
Start abuse of COC/HER (age)	16.6 (2.4)	15.2 (3.1)	0.102	.914
Years of COC/HER abuse	17.2 (7.6)	16.35 (5.8)	0.112	.812
Preferential drug use				
COC (*n*)	2	3	—	—
HER (*n*)	2	2	—	—
COC + HER (*n*)	6	5	—	—
STAI				
State	30.40 (7.7)	28.60 (11.1)	−0.151	.880
Trait	28.1 (11.5)	29.40 (7.7)	−0.114	.910
HDRS	18.70 (6.0)	17.20 (8.0)	−0.495	.623

dependence. Although all the individuals have abused both cocaine and heroin, there were some differences in their preferred drug of abuse. Eleven individuals have abuse/dependence of both cocaine and heroin, and four individuals were heroin dependent and only later in life abused cocaine. The other five individuals were cocaine and only later in life abused heroin. Due to the small number of individuals per group (relative to the pattern of consumption), the results of the inmates within each treatment group, NFB or control, were averaged together to study clinical characteristics and impulsivity changes. On the contrary, no differences in anxiety or depression were present before treatment, between the NFB and the control groups (see Table 1).

Clinical Follow-Up

Repeated-measures analysis of "mean clinical state" including 10 time points and comparing the two treatment groups revealed a significant effect of group ($F = 4.958$, $df = 1$, $p = .039$), and a significant effect of time ($F = 16.02$, $df = 3.438$, $p = .000$) and Group × Time interaction ($F = 3.32$, $df = 3.438$, $p = .021$). When analyzing group differences in each time point, there were no significant differences at point Mcs1 ($t = 0.77$, $p = .449$), Mcs2 ($t = 0.65$, $p = .522$), or Mcs3 ($t = 1.19$, $p = .248$). Symptom decrease in the NFB group relative to the control group was significant at Mcs4 ($t = 2.28$, $p = .035$), Mcs5 ($t = 2.56$, $p = .020$), Mcs6 ($t = 2.18$, $p = .043$),

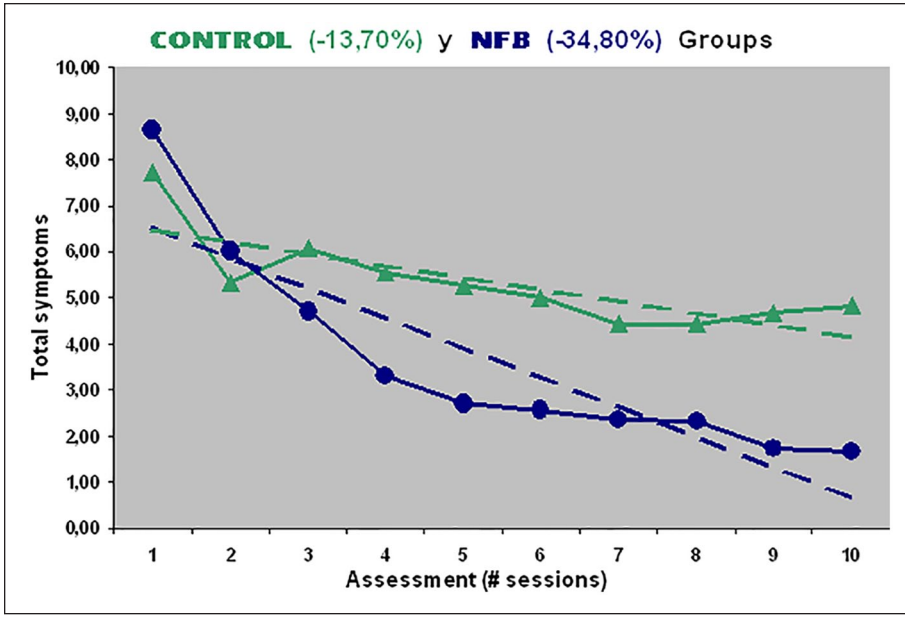

Figure 3. *Mean clinical state* over time for the NFB and control groups.
Note. The average of clinical symptoms for all participants at each session is depicted over the treatment period. NFB = neurofeedback.

Mcs9 ($t = 1.96$, $p = .015$), or Mcs10 ($t = 3.13$, $p = .006$). Quasi-significant differences were present at Mcs7 ($t = 1.96$, $p = .065$) and Mcs8 ($t = 2.05$, $p = .055$).

As group differences appear to be significant at Mcs4 and remain significant at Mcs10, the Wilcoxon test was applied to study "mean clinical state" differences between two time periods: Mcs1 and Mcs4 and between Mcs4 and Mcs10, in both the NFB and control groups. In the NFB-treated group, there were significant differences in the two time periods: Mcs1 to Mcs4 ($z = -2.81$, $p = .005$) and Mcs4 to Mcs10 ($z = -2.71$, $p = .007$). No significant differences were found between the same time points in the control group: Mcs1 to Mcs4 ($z = -1.60$, $p = .110$) and Mcs4 to Mcs10 ($z = -0.54$, $p = .588$). A significant improvement in depressive symptoms (HDRS: $p = .008$) and anxiety measures (STAI-state: $p = .038$; STAI-trait: $p = .019$) was observed in the NFB group at the end of the treatment, whereas no significant differences were found in the control group for depressive symptoms (HDRS: $p = .141$) or anxiety-trait measures (STAI-trait: $p = .495$). Nevertheless, a significant decrease was found in anxiety-state measures (STAI-state: $p = .017$) in the control group. Figure 3 shows that symptoms change along the treatment sessions for the NFB and the control groups, respectively; the average of clinical symptoms for all participants at each session is depicted over the treatment period (5 months). A trend line is included to show change over time for NFB (slope: –34.8%) and control (slope: –13.7%) groups.

Table 2. Number of Persons Being Under Medication in the Assessments Before (Pre-) and After (Post-) Treatment, in the NFB and the Control Groups.

Name	Drug group	Pre (*n*)	Post (*n*)
Control group			
Quetiapine	Antipsychotic	7	6
Mirtazapine	Antidepressant	0	1
Clonazepam	Benzodiazepine	1	1
Alprazolam	Benzodiazepine	2	1
Clorazepate	Benzodiazepine	4	3
Diazepam	Benzodiazepine	2	1
Methylphenidate	Psychostimulant	2	1
NFB group			
Quetiapine	Antipsychotic	2	1
Trazodone	Antidepressant	2	2[Hd]
Mirtazapine	Antidepressant	1	0
Fluoxetine	Antidepressant	1	1[Hd]
Clorazepate	Benzodiazepine	2	0
Diazepam	Benzodiazepine	4	1
Alprazolam	Benzodiazepine	1	0
Lormetazepam	Benzodiazepine	2	0
Methylphenidate	Psychostimulant	0	1

Note. NFB = neurofeedback; Hd = half dose.

At the beginning of the protocol, all participants were under psychotropic medication, whereas at the end of the treatment, the lowering of dosage medication was larger in the NFB group compared with the control group (see Table 2).

Changes in Impulsivity After NFB Treatment

Barratt Impulsivity Scale. No significant differences were found in the control group between the pre- and post-treatment BIS-11 measures. Mann–Whitney *U* test showed significant differences between the NFB and the control groups after the NFB treatment, for Attentional subscale of BIS-11 ($p = .01$), and a tendency toward a significant effect for global-BIS ($p = .02$), after Bonferroni correction. Effect size for mean differences of groups within the pre–post control design showed a moderate to high effect for the attentional, nonplanning, and global-BIS (see Table 3).

Commission errors. In the NFB-treated group, there was a significant decrease in the commission-error scores, both in the H1 ($p = .017$) and the H2 ($p = .036$) stages of the test, when comparing the pre- and the post-test scores. After Bonferroni correction, only the decrease of the H1 stage remains significant. No significant differences were seen in the control group in any of these scores (see Table 3).

Table 3. Mean and Standard Deviation for the Cognitive, Motor, Nonplanned, and Global Scales for the BIS-11 and Err-Comm-H1 and Err-Comm-H2 Scores Obtained Before (Pre-) and After (Post-) Treatment in the NFB and the Control Groups (Wilcoxon's Test). Z scores and the significance value (p), comparing the NFB and the control groups (effect size dKorr for mean differences of groups has included Mann–Whitney U test), are also reported. BIS = Barratt Impulsiveness Scale; NFB = neurofeedback.

	Pre	Post	Pre–post	
Parameter	*M* (*SD*)	*M* (*SD*)	*Z*	*p*
Control group (*n* = 8)				
Cog-BIS	17.60 (4.48)	16.50 (2.39)	−0.91	.362
Motor-BIS	20.30 (6.13)	15.63 (6.84)	−1.52	.128
Nonplan-BIS	19.50 (5.63)	17.00 (6.00)	−1.02	.307
Global-BIS	57.40 (12.93)	49.87 (14.18)	−1.69	.091
Err-Comm-H1	7.44 (10.92)	5.14 (8.65)	−1.36	.175
Err-Comm-H2	9.11 (15.53)	5.00 (6.35)	0.94	.351
NFB group (*n* = 10)				
Cog-BIS	13.90 (6.08)	11.80 (4.80)	−1.12	.262
Motor-BIS	14.50 (5.76)	11.70 (4.55)	−1.52	.128
Nonplan-BIS	19.10 (5.02)	14.10 (4.41)	−2.41	**.016***
Global-BIS	49.10 (11.02)	37.90 (9.00)	−2.19	.028
Err-Comm-H1	3.90 (5.09)	1.66 (3.57)	−2.39	**.017***
Err-Comm-H2	5.33 (7.31)	1.66 (1.32)	−2.10	.036

NFB vs. control (post-treatment)

	p	Effect size d_{Korr} pre–post control
Cog-BIS	.01**	**0.49**
Motor-BIS	.23	0.30
Nonplan-BIS	.41	**0.48**
Global-BIS	.02[a]	**0.51**
Err-Comm-H1	.25	0.14
Err-Comm-H2	.35	**0.46**

[a]Quasi-significant.
*Significance level at .01 after Bonferroni correction.

Discussion

To our knowledge, this is the first study using an NFB protocol based on the Othmer method to treat impulsivity in offenders who have been abstinent for at least 8 months and also had symptoms of anxiety and depression. Both groups, the NFB and the control, were equivalent at the beginning of the protocol, that is, there were no significant differences in measures of anxiety, depression, or impulsivity, in age or in educational and sociodemographic status. All the inmates in the NFB group got benefit from the treatment according to the improvement of their clinical symptoms, and this benefit

was higher than in the control group. The current results are equivalent to those reported by a recent study reporting an improvement in behavior and impulsivity in a small group of forensic psychiatric individuals (Fielenbach et al., 2019).

When analyzing the evolution of the clinical symptoms from the temporal perspective along the period of treatment, there were two clearly differentiated periods: first one, from the beginning (Mcs1) to Session 16 (Mcs4), with significant between-group differences in the "mean clinical state." In addition, at Mcs4, there was a significant improvement in the NFB-treated group (Mcs1–Mcs4) relative to the control group. Another differentiated time period was found between Mcs4 and Mcs10, with significant differences between both treatment groups, and a significant improvement between Mcs4 and Mcs10 in the NFB but not in the control group. The decrease in depressive symptoms measured with the HDRS and in STAI-related anxiety measures supports the recovery of clinical symptoms and the enhancement in the well-being in this population, with the NFB treatment. This improvement is more clinically relevant when considering that people in the NFB group reduced the dosage or even withdraw their psychotropic medication at a higher rate than the control group during the treatment period. The improvement of clinical symptoms of anxiety and depression is also crucial in preventing relapse in cocaine- and heroin-abstinent patients (Corominas et al., 2010; DiGirolamo et al., 2017; Hasin et al., 2002). The current results are in line with a previous study reporting that the Othmer method was useful in restoring sympathetic imbalance in the general population (Altan et al., 2016).

Regarding impulsivity, the Nonplanning subscale of the BIS-11 showed an improvement in the NFB group with a middle effect size, similar to that of the Attentional subscale. The Nonplanning and the Attentional subscales are thought to be associated to impulsive choice or decision making (Patton et al., 1995; Winstanley et al., 2006), and from the neurobiological point of view, impulsive decision making has been linked to functional deficits in the orbitofrontal and the ventromedial PFC, including the frontal pole (see Winstanley et al., 2006, for discussion). This background is relevant because these brain regions have also been associated, at least in part, to neurobiological deficits underlying addiction (London et al., 2000; Stapleton et al., 1995; Volkow et al., 1992; Volkow & Fowler, 2000). Regarding behavioral tests, there was also a significant improvement in the Error Commission subscale of the CPT, a neurocognitive test associated to motor impulsivity (Dalley et al., 2011). The differential evolution between motor and nonplanning measures of impulsivity could be due to differences in brain areas underlying both kinds of impulsive measures or even to limitations of the study due to the small sample size. In this regard, there had been some interindividual differences in the addiction profile that might have contributed to differences in the evolution of motor and nonplanning measures, in response to the NFB treatment. It is also important to mention here that the modulation of impulsivity was present even taking into account that antidepressant or antipsychotic medication used to improve impulsive behavior was reduced or withdrawn during the treatment with NFB. On the contrary, and taking into account that by the time of the current protocol the inmates had been abstinent between 8 and 30 months, the decrease in impulsivity can be attributed to improvement of long-term consequences of drug abuse that

continue to take place even after long-term abstinence (Cadaveira et al., 1994), or even to a decrease in impulsivity itself, as have been also reported in people with ADHD (Gevensleben et al., 2009; Strehl et al., 2006). These results are relevant because the modulation of impulsivity is crucial to prevent relapse in cocaine (Bell et al., 2014; Economidou et al., 2009; McHugh et al., 2013) or heroin (Fareed et al., 2017; Li et al., 2013; Su et al., 2015) consumption. In addition, impulsivity and substance misuse are often associated to criminal offending, and reducing these symptoms might be important in reducing recidivism in offending behavior. It is important to take into account that the inmates had limited their access to drugs of abuse, and hence, the current results are not comparable with other authors such as Hulka et al. (2015), who assessed trait and behavioral impulsivity in cocaine addicts during withdrawal.

The results of the current study are in line with those by Scott et al., who reported a significant improvement in impulsivity after 10 to 20 sessions of combined theta/beta and SMR NFB protocol (Scott et al., 2005). Although the current study also included a group of poly-substance-dependent individuals, both samples have crucial differences because Scott included active drug abusers and the current protocol involves long-term abstinence. Other authors in this field, although reporting positive effects of NFB in drug consumption, do not specifically assess the effects over impulsivity (Dehghani-Arani et al., 2013; Horrell et al., 2010; Rostami & Dehghani-Arani, 2015). The results of the current study are also in line with those that assessed the effects of NFB over impulsivity in patients with ADHD (Arns, Feddema, & Kenemans, 2014; Bluschke et al., 2016; Gevensleben et al., 2009). The protocols including children and adults with ADHD are relevant to the current study because impulsivity is one of the core symptoms of this disorder (Biederman et al., 1996; Ramos-Quiroga et al., 2014) and ADHD is overrepresented in forensic populations (Woicik et al., 2017). ADHD characterizes by hypo-activation of the frontal cortex, low brain activation in the right inferior PFC, the precuneus and cingulate gyrus, and dysfunction of the fronto-striatal system (Castellanos et al., 1996; Rubia et al., 2005), most of the brain regions associated to impulsivity. Moreover, the results of our study are in line with the report from Konicar et al. who addressed impulsivity in a sample of psychopathic individuals. Konicar reported an improvement in impulsive behavior after training with 15 SCP-based NFB sessions (Konicar et al., 2015). Despite the differences between the two study samples, long-term abstinent inmates and psychopathic individuals have in common their impulsivity and, from the neurobiological point of view, also share deficits of the prefrontal function underlying inhibitory control (da Cunha-Bang et al., 2017).

Despite all these positive results, recent studies based on double-blind designs have reported that NFB was not superior to sham feedback in improving neurocognitive functioning and impulsivity, and suggested that unspecific factors might be driving the clinical improvement reported in previous studies (Bink et al., 2014; Logemann et al., 2010; Schönenberg et al., 2017). In the understanding of these discrepancies, in addition of the criticisms that double-blind designs have received (Arns, Heinrich, & Strehl, 2014; Lansbergen et al., 2011), the characteristics of the study population are also to be taken into account. Most of these studies were conducted in patients with

ADHD, a neurodevelopmental disorder that in most cases persists into adulthood (Bink et al., 2014; Logemann et al., 2010; Schönenberg et al., 2017), or primary insomnia (Schabus et al., 2017). The current study population included long-term abstinent addicts, and in these patients, impulsivity is not only a primary condition but also a consequence of the consumption. This leads to suggest that NFB might be more effective with acquired deficits that might be more susceptible of being reversed.

Some limitations of the current study are to be mentioned. First, due to the small sample size, these results are only preliminary. A second limitation is related with the use of Likert-type scales that are useful to assess very short-term repeated measures, although they are somehow unspecific. Third, those issues associated to the closed environment where the study has been conducted. On one hand, the fact that the inmates' life together would introduce some "contagion effect" might influence the evolution of both study groups, reducing their differences. On the other hand, this environment would affect the ecological validity of the study. The freedom of the inmates was limited, so we do not know the results of the treatment if the inmates would had to face their daily life in freedom. Finally, even taking into account that the EEG measures are not pathognomonic of any psychiatric disease and cannot be used as primary markers, to measure the NFB effects based on the EEG parameters would have been important. Nevertheless, authors such as Egner et al. (2004), Arns et al. (2012), and Mayer et al. (2015) only reported behavioral changes after NFB training, whereas changes in EEG frequencies were not significant. These data suggest that focusing on clinical improvement can be a good criterion to assess the effects of NFB training.

Conclusion

Our results suggest that NFB is better than placebo in the modulation of impulsivity in this population of long-term abstinent cocaine- and heroin-dependent individuals, and the improvement of impulsivity is crucial to prevent relapse, not only in substance misuse but also in offending behavior. In addition, it is also important to mention the improvement of clinical symptoms of anxiety and depressive mood, suggesting that NFB is also useful to improve the general well-being in this population. The nonplanning measures of impulsivity, closely associated to the PFC function, showed larger improvement than motor measures. On the contrary, the clinical profile is also very important to understand the results of an NFB study. Future studies with larger sample sizes to study the long-term NFB effects over impulsive and addictive behavior would be needed.

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