Chapter 2

BRAIN WAVES IN IMPULSIVITY SPECTRUM DISORDERS

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ABSTRACT

Impulsivity has proven to be an important psychological construct reflected in both normal and pathological human behaviors and traits. However, the definition, measurement, and manifestations of the construct are varied and multifarious. Impulsive behaviors are observed in a wide range of psychiatric/behavioral disorders, including alcohol/substance use and abuse disorders (AUDs/SUDs), conduct disorder (CD), attention-deficit hyperactivity disorder (ADHD), antisocial (ASPD), narcissistic and borderline personality (BPD) disorders, pathological gambling, and eating disorders, and are variously termed as 'impulsive' or 'impulse-control', 'externalizing', or 'disinhibitory' disorders. Studies utilizing sensitive and non-invasive electrophysiological techniques to analyze brain waves in impulsive conditions and disorders have elucidated brain functioning associated with these conditions. These electrophysiological procedures primarily include electroencephalogram (EEG), event-related potentials (ERPs), and event-related oscillations (EROs). Major electrophysiological findings across the majority of the 'impulsivity spectrum disorders' include excessive beta power in the resting EEG, decreased P3 amplitude of the ERP, and decreased ERO delta and theta power. This chapter attempts to summarize, explain and synthesize key findings of studies that have used electrophysiological methods to elucidate and understand impulsivity in its normal and pathological manifestations.

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INTRODUCTION

> A true history of human events would show that a far larger proportion of our acts are the result of sudden impulse and accident than of the reason of which we so much boast.
> Peter Cooper

In the modern scientific literature, the term 'impulsivity' has assumed a central place to describe and understand a variety of behavioral and personality traits as well as pathological conditions. Although a growing body of theoretical and experimental studies has attempted to examine this concept, the construct of impulsivity has always posed a challenge because of its complexity in terms of its definition, dimensionality, factor structure, clinical manifestations, and biological bases. This lack of clarity may be due to several causes, including, 1) impulsivity is a multidimensional concept, 2) it is manifested in both normal and pathological conditions, 3) its biological bases have not been fully understood, and 4) a comprehensive and holistic model to explain the concept of impulsivity and its manifestations is still lacking.

The definitions and measurements of impulsivity vary widely in the research literature [1]. According to Dawe et al [2] impulsivity refers to a tendency to engage in behavior that involves rashness, a lack of foresight and planning, or a behavior that occurs without reflection or careful deliberation. Impulsivity is generally regarded to be a dysfunctional trait, associated with actions that may be criminal and/or violent, physically harmful to the self (such as suicide), or inappropriate in terms of accepted social standards [3]. This view is predominant in many of the definitions, including that of Daruna and Barnes [4], who defined impulsivity as "actions and tendencies that are poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation and that often result in undesirable consequences". Impulsivity and related constructs, such as sensation seeking, novelty seeking, disinhibition, response inhibition, delay discounting, risk taking, and extroversion, are often measured by self-report and/or laboratory measures. The most common self-report measures include the Barratt Impulsivity Scale (BIS; [5]), the Impulsivity-Venturesomeness-Empathy Scale (IVE; Eysenck et al.,1985), the UPPS Impulsive Behavior Scale (IBS; [6]), the Sensation Seeking Scale (SSS; [7]), the Tridimensional Personality Questionnaire (TPQ; [8]), and the Temperament and Character Inventory (TCI; [9]). On the other hand, direct measurement of inhibitory control processes using laboratory tasks include (i) measures of *response inhibition to* assess the ability to 'inhibit' or suppress automatic (prepotent) responses, namely in the Go-NoGo task, the Stop Signal task, the Stroop task, and measures of commission errors on Continuous Performance Tests (CPTs) and other tasks; (ii) measures of *delay discounting to* evaluate the choice preference for a small reward available sooner over a larger reward available at a later point in time); and (iii) measures of *cognitive impulsivity* to assess problems related to decision-making (cf. [3]).

Impulsivity has been a topic of great interest among both behavioral and neurobiological researchers. Studies aimed at understanding specific, biological underpinnings of impulsivity and impulsive conditions have used a wide range of methods, including electrophysiological, neuroimaging, molecular and genetic techniques. Among these, electrophysiological techniques, such as recording EEG, ERPs, and EROs with scalp electrodes provide non-invasive, sensitive measure of brain function in humans [10], and are unique in that they provide direct measures of brain activity while they are happening, with exquisite temporal resolution in the time scale of milliseconds. This chapter provides a review of

electrophysiological findings with regard to impulsivity in normal and clinical populations. Although the spectrum of impulsive disorders is wide and includes several clusters of disorders, only prominent disorders/conditions (aggression/violence, ASPD, ADHD, AUDs and SUDs, and mood disorders) with sufficient electrophysiological studies have been reviewed under separate sections, while other disorders/conditions (pathological gambling, eating disorders, kleptomania, pyromania, trichotillomania, and paraphilia) with a relative dearth of studies are dealt with together in a single section. Further, electrophysiological findings on normal as well as 'subclinical' individuals with high impulsivity are also discussed.

IMPULSIVITY IN NORMAL BEHAVIORS AND PATHOLOGICAL CONDITIONS

While subclinical 'impulsive' behaviors pervade and shape our decisions in everyday life, they are often not pathological in terms of their magnitude and appropriateness in a given context, situation or developmental stage. In fact, these behaviors add important color to our everyday life [1]. On the other hand, there are a wide variety of clinical conditions that involve impulsivity as a core component. Between these two spectrums, there is "a great deal of disagreement as to what differentiates socially acceptable impulsive behavior from the unacceptable; this varies from one culture to another, from one era to another, and depends upon the age of the person involved" [1, p.348]. Therefore, the definitions of impulsivity have taken a two-track approach. While most of the authors have attempted to paint a clinical picture of the concept, there are other explanations that argue that impulsivity is both normal and adaptive. For example, according to Dawe et al. [11], impulsivity refers to the tendency to engage in behavior that involves rashness, a lack of foresight or planning, or as a behavior that occurs without reflection or careful deliberation. In contrast, Jones et al. [12] defines impulsivity as an organism's quick response to environmental cues while not considering alternative responses to the cues. The authors further argued that impulsive traits have an evolutionary advantage for an organism in being quick and agile. In the authors' own words, "the organism that does not quickly pounce on a potential prey or dodge a potential predator may not get another chance" [p.1674]. In distinguishing these two streams, Dickman [13] classified impulsivity into two categories: 1) 'dysfunctional impulsivity' which is the tendency to act with less forethought than most people of equal ability when this tendency is a source of difficulty, and 2) 'functional impulsivity', which is, by contrast, the tendency to act with relatively little forethought when such a style is optimal. Dickman also found that these two tendencies are not highly correlated and that they bear different relations to other personality traits and cognitive processes. Furthermore, there are also developmental patterns of change in impulsivity, which show a steady decline in impulsivity scores as age increases from late adolescence to adulthood as the brain matures [14]. Therefore, age norms for impulsivity scores have been developed [15].

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) [16] has listed impulsivity as a core feature in the diagnostic criteria of many psychiatric disorders, including ASPD, CD, BPD, SUDs, paraphilias, adjustment disorder, eating disorders and mood disorders along with other conditions such as intermittent explosive disorder, kleptomania,

pyromania, pathological gambling, and trichotillomania. Several of such disorders have been classified as either disinhibitory disorders [17] or externalizing disorders [18-20]. Recently, a rubric of impulsive and compulsive disorders has also been proposed to include a range of conditions that have common neurobiological mechanisms [21]. In this chapter we focus on the application of various electrophysiological methods and studies to this spectrum of impulsivity disorders, in order to characterize the brain wave patterns and (sometimes subtle) aberrations in neural processing that are related to these disorders. In the following sections, electrophysiological studies, both with and without the direct assessment of impulsivity, have been included, based on the assumption that the disorders grouped as "impulsivity spectrum disorders" are, by virtue of their clinical and biological nature, associated with impulsivity as a core construct or symptom.

ELECTROPHYSIOLOGY IN IMPULSIVITY SPECTRUM DISORDERS

Electrophysiological techniques using non-invasive scalp recordings provide sensitive measures of ongoing brain function in the form of neuroelectric activity (i.e. brain waves). The first human EEG was recorded by the German physiologist and psychiatrist Hans Berger (1873–1941) in 1924 [22]. These brain waves can be recorded and analyzed using three distinct methods: 1) EEG—frequency-dependent, spontaneous and continuous neural activity during a restful or specific mental state, 2) ERPs—time-locked, trial-averaged, and task-specific neuroelectric activity in the time domain during a specific sensory/motor/cognitive event, and 3) EROs—time-frequency measures of brain electrical activity during a specific sensory/motor/cognitive event.

ELECTROPHYSIOLOGICAL METHODS

EEG

Recording of the ongoing brain waves in a continuous fashion represents the human scalp EEG. EEG can be recorded during various mental states: eyes-closed relaxed state, eyes-open steady state, meditation, hypnosis, sleep, coma, and other normal/altered states of consciousness [23]. Human EEG can be decomposed into different frequency components; traditionally, these are delta (0-4 Hz), theta (4-8 Hz), alpha (8-12 Hz), beta (13-30 Hz) and gamma (30-50 Hz), and each of these bands reflects different brain activity. These frequency bands have been aligned with a continuum of consciousness, with delta associated with sleep, theta associated with drowsiness and low levels of alertness, alpha associated with relaxed wakefulness, and beta and gamma associated with alertness, vigilance and cognitive processing [23]. The resting EEG during wakefulness in healthy adults primarily consists of medium (8–13 Hz) and fast (14–30 Hz) frequencies, while low frequencies (0.3–7 Hz) and high frequencies (>30 Hz) occur sparsely [24]. Measures of asymmetry (comparison of EEG activation across hemispheres) [25], power spectrum analysis (computation of power within a specific frequency band) [24], relative power (ratio of one frequency band to another) [24] and coherence (association between two EEG signals) [26,27] have been considered very

useful methods of EEG analysis to understand the underlying neurocognitive functions and neural connectivity in the brain.

ERPs

ERPs are time-locked voltage fluctuations in the brain in response to a sensory, motor, or cognitive event. ERPs can be recorded from the human scalp and extracted from the ongoing EEG by means of filtering and signal averaging [28]. These electrical potentials are obtained by averaging several EEG epochs, and represent large numbers of neural elements acting in synchrony during information processing, from early sensory perception to higher cognitive processing. While later components of the ERP reflect higher cognitive function, early components, occurring before 100 ms reflect sensory processes, and these are often termed evoked potentials (EPs); examples of EPs include, Brain Stem Auditory Evoked Potentials (BAEPs), sensory gating (P50), and auditory, visual and somatosensory EPs [29]. ERP components are generally identified and interpreted based on their eliciting conditions, polarity (positivity or negativity), timing (latency) and scalp distribution (topography). The voltage deflections as represented in these components reflect the reception and processing of sensory information as well as higher level processing that involves selective attention, memory updating, semantic comprehension, and other cognitive activity [30]. The latency (time of occurrence in milliseconds) of an ERP component reflects neural processing time, while the amplitude (or the magnitude of a waveform in microvolts) has been related to the neural resources available to process a stimulus or event [31]. ERP components that are frequently studied include P1, N1, P2, N2, P3 (P300), N4 (N400), Mismatch Negativity (MMN), Contingent Negative Variation (CNV), and Bereitschaftspotential (BP) or readiness potential. These components are obtained using specific ERP tasks, such as Oddball tasks, Go/NoGo tasks, Continuous Performance task, Stop-Signal tasks, Gambling tasks, Decision-making tasks, Memory tasks, and Tapping tasks [31].

Each ERP component has been attributed to specific functions. In a broader perspective, according to Kotchoubey [32], a positive ERP component (e.g., P1, P2, and P3) is a manifestation of neural functioning of a given brain area and utilization of already prepared resources, while a negative components (e.g., N1 and N2) reflect further preparation for those events or features which are to be encountered next. Specifically, the P1 component reveals an attentional priming effect on early sensory responses [33]. The N1 component, first described as a measure of attention and vigilance by Haider et al in 1964 [34], is generally thought to represent the initial extraction of information from sensory analysis of the stimulus [35], or the excitation associated with allocation of a particular channel for information processing among others, and is at the level of the primary cortex [36-38]. The P2 component, in general, may represent inhibition of sensory input from further processing [39] through automatic stimulus identification and discrimination/classification [40], or inhibition of other channels of information competing for attention and further processing [37,38]. Under active attend conditions, the N2 component is thought to represent an endogenous mismatch detection process and to be related to stimulus discrimination [41,42]. The most widely used, debated and interpreted component of the ERPs is the large prominent positive P3 component that occurs between 300-600 ms, and can be derived from many different cognitive tasks [30]. Since the P3 or P300 component was first discovered by Samuel Sutton in the 1960s [43,44],

it has been widely used to investigate both normal and dysfunctional cognitive processing, and has served as one of the most effective and useful biological markers and endophenotypes in several behavioral and brain disorders [45]. An influential view is that it represents context updating when stimulus events require that an individual's model of the environment must be revised or updated [46]. The extent to which this updating process is activated depends upon the value, significance or relevance of the stimulus [47]. This view has been criticized, with alternative perspectives proposed, such as context closure [48,49] and event categorization [50]. Interpretations of a deficient P3 include abnormal capacity allocation [51], an attentional deficit [52], a context or short-term memory updating deficiency [53], or a memory-related processing deficit [54]. The N400, first discovered by Kutas and Hillyard in 1980 [55], is a negative-going deflection that peaks around 400 milliseconds post-stimulus onset, and is typically maximal over centro-parietal electrode sites. The N400 is elicited in response to visual and auditory words and other meaningful stimuli, such as sign language, pictures, faces, environmental sounds, and smells (See Kutas and Federmeier [56] for review). The mismatch negativity (MMN), discovered in 1978 by Näätänen et al [57], is thought to arise from automatic detection of stimulus deviance or irregularity, and represents a memory trace of physical or abstract environmental irregularities [58]. The contingent negative variation (CNV), first described in 1964 by Walter et al [59], occurs as a result of cognitive processes such as orienting, expectancy, and response preparation for an imperative stimulus [60]. The Bereitschaftspotential or "readiness potential", first recorded and reported in 1964 by Kornhuber and Deecke [61], is a slow negative activity preceding voluntary motor activity, and represents the pre-motor planning of volitional movement [62]. This review is restricted to an examination of the ERP components that have proved to be most significant in the study of various impulsivity spectrum disorders, primarily focusing on later ERP components, but also including EP studies of earlier components when few or no ERP studies are available.

EROs

Until recently, ERPs have been the traditional, well-established electrophysiological indices of cognition, providing valuable insights into human brain processes. More recently, there is an increasing literature that indicate that some ERP features may arise from changes in dynamics of ongoing EEG oscillations of different frequency bands that reflect ongoing sensory and/or cognitive processes ([63-65]). Hence EEG oscillations in the resting state became organized, amplified and/or coupled during mental activity, or the mental activity induced by an event or stimulus may trigger specific oscillatory responses; this gives rise to "evoked" (strongly phase-locked to the stimulus/event) or "induced" (not phase-locked to the stimulus/event) oscillations [66], termed event-related oscillations (EROs). Thus, EROs represent the basic mechanism of neural communication in the brain, providing links to associative and integrative functioning. ERO activity patterns are measured in terms of time (time course of the oscillations with respect to an event), frequency (number of waves per unit time), amplitude/power (height or magnitude of the waveform), phase (the initial angle of a sinusoidal function at its origin as well as the polarity in terms of positivity or negativity with reference to a baseline) and coherence (relatedness between two signals) measures [63,64]. Specific frequency rhythms of oscillatory responses have been attributed to underlie various cognitive processes [63,67-70], although the interpretation is often task-specific. Event-

related desynchronization and synchronization (ERD/ERS) is one of the valuable techniques to measure amplitude changes within a given frequency band due to a sensory, motor or cognitive processing in the brain [71,72]. Event related coherence (ERcoh) is another useful method to reveal brain functioning during cognitive processing as well as specific neural circuits underlying these processes [73-77]. Using the methods of coherence or synchrony, neural communications and functional connectivity can be studied. It has been shown that high frequency oscillations (fast beta and gamma bands) are involved in local, short range neural communication, while the low frequency oscillations (delta, theta, and alpha bands) represent long range communications between different brain regions [78]. Many studies have employed one or more of these methods to examine the electrophysiological correlates of impulsivity in normals and in several impulsive disorders as discussed in the following sections.

NORMAL / SUBCLINICAL INDIVIDUALS

Impulsive phenomena have been studied among healthy normals, 'subclinical' groups, and clinical samples. The term subclinical is used in the chapter to refer to the individuals who did not meet the criteria for a specific diagnosis but left with one or more symptoms that are relatively less debilitating for normal functioning. Major studies that have attempted to elicit the neuroelectric correlates of impulsivity among normal or subclinical individuals are discussed below.

EEG Studies

The quest to empirically understand the brain wave patterns in impulsive states in otherwise normal subjects through the use of scalp recorded EEG dates back to 1950s and 60s [79,80]. A turning point in EEG research was the implementation of frontal EEG asymmetry as a valid technique to understand mental and emotional phenomena (see the review by Coan and Allen [81]). Since the 1980s, many EEG studies on impulsive states, emotion, motivation, and personality have been based on the phenomenon of anterior asymmetry [25,82-88], a concept that represents relatively more activity in one cerebral hemisphere over another. Davidson and Fox [89] observed that infants who cried in response to maternal separation had greater right frontal activity at rest than those who did not. Using a sample of young healthy university students, Hewig et al [88] found that subjects with greater bilateral (left and right) frontal cortical activity showed higher behavioral activation scores. In a recent study, Lansbergen et al [90] examined the relation between subjective impulsivity, theta/beta EEG ratio, and inhibitory control in healthy individuals and found that the low theta/beta ratio group showed longer stopping reaction times. Given that an increased theta/beta ratio may reflect reduced cortical inhibition over subcortical input, the authors proposed that healthy individuals with relatively high theta/beta ratios are more motivated to maximize inhibition-related performance. Although previous studies have reported that neurofeedback enhances attention and decreases impulsive behavior in ADHD children [91], a recent study with a

sham-control group failed to find the effectiveness of neurofeedback on normals who had high scores on impulsivity/inattention questionnaires [92].

ERP Studies

Later studies in the 1980s and 90s primarily used ERP paradigms to understand impulsivity in both normal and subclinical samples [93-97]. For example, a study examining the association between psychometric impulsiveness and contingent negative variation (CNV) in a Go/NoGo signaled avoidance task reported that Go-CNV recorded at the vertex (center of the cranium or scalp) was related to a variety of impulsiveness-related measures, while the NoGo-CNV appeared to index subjective arousal [94]. Another study reported that action-oriented personality traits, such as sensation seeking, extraversion, and impulsivity, were related to the auditory evoked response pattern (N1/P2-component) of the superior temporal plane, including primary auditory cortex [96]. Harmon-Jones et al [98] reported that attentional impulsiveness and non-planning impulsiveness were related negatively to the parietal P3 amplitude of the visual oddball task and visual continuous performance task (CPT), while motor impulsiveness was related positively to parietal P3 in the visual oddball task.

There are a few ERP studies of people who engage in extreme sports. A study of skydivers reported that they manifested more sensation seeking and larger frontal P3 amplitudes than controls [97]. The authors postulated that the augmented P3 amplitude reflects the capacity to use some risky behaviors which improve automatic attentional processes, to obtain arousing stimulation that could counterbalance the emotional deficit already present in the skydivers. Fjell et al [99] reported that P3a habituated significantly more in extreme sporters than in the other two groups (high impulsive non-sporters and low impulsive group). Taken together, these studies suggest that extreme sporters require the stimulation of extreme sports to maintain automatic attentional resources.

Recent ERP studies have examined impulsivity using novel paradigms and analysis methods. Fallgatter and Herrmann [100] reported that impulsivity in healthy subjects was correlated with a more anterior location of the Go and the NoGo centroids of the global field power in the P3 time window. Pailing et al [101] proposed that error trials are associated with faster responses than correct trials in simple discrimination tasks; response time (RT) differences between incorrect and correct trials can be indices of impulsive responding. Martin and Potts [102] suggested that the P2a component, which was time locked to feedback signals in a simple gambling task, can be taken as an index of orbitofrontal reward processing and of reward hypersensitivity due to impulsivity. On the other hand, Ruchsow et al [103] reported that normal subjects with higher impulsiveness showed smaller amplitudes than subjects with lower impulsiveness for the ERN/Ne (error-related N2) component and the error positive (Pe, or error-related P3) component. Another study by the same investigators [104] reported that high impulsive subjects had significantly reduced NoGo-P3 amplitudes compared to low impulsive subjects. Another ERP component, the mismatch negativity (MMN), was found to be larger in high impulsive individuals [105]. Delorme et al [106] established that the positive pre-response ERP peak (P3f), which is maximal at forehead sites in visual selective attention tasks, may index cortical activity related to impulsive motor responses. Russo et al [107] found that high impulsive subjects had lower P3 amplitudes as

well as reduced cognitive performance on intelligence testing than low impulsives. Further, De Pascalis et al [108] reported that high impulsive compared to low impulsive participants displayed more pronounced N400 peak amplitudes over fronto-central sites, and made more errors and had longer reaction times in identifying incongruent words. Herrmann et al [109] investigated the association between the amount of inattention and hyperactivity/impulsivity symptoms in a non-clinical population of healthy students and found reduced amplitudes of error-positivity (Pe) with an increasing number of symptoms. In a study of decision making, Martin & Potts [110] found that the high impulsives had a larger P3 than the low impulsives when making a low-risk choice. Recently, using a sample of university students, Venables et al [111] reported that both aggressiveness and impulsivity had negative correlations with P3 amplitudes to both target and novel stimuli over frontal-central scalp sites. Taken together, a number of studies using various tasks and measures indicate these ERP components are reduced in high impulsive individuals.

Contrary to the prevalent findings that there is an attenuation in the ERP components in high impulsive individuals, Dimoska et al [112] reported that the N1/P3 complex in stop-signal trials was enhanced in the high impulsive compared to the low impulsive group, a rare finding shared by very few studies [97,113]. The authors postulated that in order to have comparable performance to the low impulsivity group, the high impulsive group can counteract an impulsive response style with enhanced inhibitory activation. An index for developmental lag in response inhibition in children was suggested to include higher false alarm and impulsivity scores and the absence/attenuation of the fronto-central NoGo P3- [114]. In summary, most studies find that individuals with high impulsivity and subclinical impulsive traits showed dysfunctions in several ERP components, especially showing reduced P3 amplitudes in several task paradigms.

ERO Studies

Relatively few ERO studies have examined impulsivity in normals. Using a time-frequency analyses in a task involving speeded response selection for visual target stimuli, Delorme et al [106] established that quicker responses are associated with larger slow theta complexes peaking near the mean P3f latency, and further suggested that frontal slow theta complexes (and concomitant mean P3f positivity) may index impulsive motor responses to rewarding or goal-fulfilling stimuli or events. Kamarajan et al [115] analyzed event-related theta power (4.0-7.0 Hz) following outcome stimuli in the time window of the N2-P3 complex (200-500 ms) in healthy normals during a gambling task that involved monetary loss and gain. It was reported that fast theta (5.5-7.0 Hz) was associated with impulsivity measures of self-reported BIS score and task-related impulsivity scores. Using a stop-signal task in normal subjects, Knyazev et al [116] reported five major ERO findings in high impulsive subjects: 1) higher baseline delta, theta and alpha power, 2) higher magnitude of induced responses in low frequencies, 3) lower phase-locking in low frequencies to auditory stimuli, 4) higher phase-locking to the overt behavioral response onset, and 5) higher alpha desynchronization. Recently, Bernat et al [117] used time-frequency analysis in a simulated gambling task and found that delta-P3 amplitude was reduced among individuals high in externalizing proneness, while theta response related feedback negativity (N2 activity during feedback or outcome) was unrelated to externalizing. In summary, several promising ERO

correlates of impulsivity and related traits have been reported with various paradigms, though the number of studies is relatively few.

ANGER, AGGRESSION AND VIOLENCE

Anger is a natural and mostly automatic response to pain of one form or another (physical or emotional) [118]. The emotional experience of anger does not always lead to an aggressive course of action, and by contrast, aggression or violence does not always occur within the context of angry affect [119]. Aggression can be categorized as instrumental (or proactive) aggression and hostile (or reactive) aggression [120,121]. Instrumental/proactive aggression involves a relatively nonemotional display of aggressive behavior that is directed toward obtaining some goal. Hostile/reactive aggression, on the other hand, involves aggressive behavior that takes place within the context of associated anger and high emotionality (cf. [119]). Reactive aggression often occurs in conjunction with anger and in response to the experience of negative affect, and often ends up in violent acts and crimes. Impulsivity is a major factor in all three constructs. Electrophysiological studies in this regard are discussed below.

EEG Studies

EEG Asymmetry: Anger and aggression are shown to be associated with EEG asymmetry (see Harmon-Jones [122] for a review). In a series of studies, Harmon-Jones and colleagues [123-127] established the relationship between frontal asymmetry and anger. Stewart et al [128] explored the possibility that different styles of anger expression could presumably involve different brain mechanisms and/or interact with psychopathology to produce various patterns of brain asymmetry by comparing EEG asymmetry in participants high in trait anger who differed in anger expression style (high anger-in, high anger-out, both) and participants low in trait anger. They found that trait anger, not anger-in or anger-out, predicted left-biased asymmetry at medial frontal EEG sites, suggesting that motivational direction is not always the driving force behind the relationship of anger and left frontal asymmetry.

Although the relationship between anger and aggression is not clear, Hortensius et al [129] recently posited that anger associated with greater relative left frontal cortical activation would be more likely to result in aggression. Pillmann et al [130] studied the relationship of EEG abnormalities and violent criminal behavior in 222 defendants referred for psychiatric evaluation and found that impairment of left hemisphere functions may enhance the propensity for violent behavior in a subgroup of offenders. Rybak et al [131] reported frontal alpha power asymmetry in aggressive children and adolescents with mood and disruptive behavior disorders, suggesting that impulsive aggression is related to left frontal hyperactivation. In a study by Peterson et al [132], frontal asymmetry could also be elicited in a simulated experimental condition of aggression, and greater relative left frontal activity was associated with greater aggression. In a study exploring the link between stress-induced asymmetric frontal brain activity and aggression risk, Verona et al [133] found that participants in the stress conditions showed more left than right frontal EEG activity, and this

asymmetry in response to stress exposure also predicted increases in subsequent aggressive behavior. In summary, studies of EEG asymmetry show that greater left frontal activity is associated with aggression, violence and criminal behavior.

EEG band power: Several studies have reported changes in the EEG band power in individuals with aggressive traits or tendencies. For example, aggressive subjects had more delta activity and less alpha activity in the spontaneous EEG, as has been typically observed in psychopaths and criminal offenders [134]. In a study on male psychiatric inpatients with violent behavior, Convit et al [135] reported that the number of instances of violence as well as the number of staff interventions were related to increased delta band activity and to decreased alpha band EEG activity in the temporal and the parieto-occipital areas. Bars et al [136] showed that individuals with high delta band absolute power in the right frontal lobe were more likely to exhibit explosive behaviors. In a study of sleep research, Lindberg et al [137] reported that the subgroup with intermittent explosive disorder (IED) had higher delta and theta power in stage 4 compared to males without this diagnosis. Taken together, these studies indicate that relative increases in slow wave resting EEG (i.e., delta) are associated with aggressive traits or tendencies.

EP / ERP Studies

Abnormalities in ERP and EP components in different task conditions have been shown to be linked with anger, aggression and violence. Fishbein et al [134] measured auditory brainstem evoked response (BAER) among drug users and found that overtly aggressive subjects had significant delays in BAER latency. Bars et al [136] observed that patients who exhibited explosive behaviors characterized by violent outbursts were significantly more likely to produce high-amplitude P1 waveforms than patients who did not exhibit such behaviors. Tarkka et al [138] reported altered frontal lobe function suggested by source analysis of event-related potentials in impulsive violent alcoholics. Recently, Fisher et al [139] conducted a retrospective chart review of 80 male and female juveniles undergoing inpatient treatment for pathologically impulsive aggression, and observed that decrements of mid-latency potentials and ERPs were more highly predictive of aggressive behavior. In a Go-NoGo task, N2 amplitude was significantly lower in impulsive-violent offenders than in matched controls [140,141], suggesting difficulties with inhibition of prepotent behavior. Koelsch [142] found the P3a component, which reflects involuntary attentional mechanisms and receives main contributions from the frontal lobes, was significantly smaller in individuals with moderate Intermittent Explosive Disorder compared to the control subjects. Branchey [143], in a study of P3 voltages in subgroups of alcoholics with disorders in mood and aggression control, observed that patients with histories of incarceration for crimes involving physical violence had the lowest P3 amplitudes. Drake et al [144] reported significantly delayed latency of P3 during an auditory oddball task in prisoners with impulsive, aggressive, and violent behavior. Barratt et al [145] examined the effects of the drug ‘phenytoin’ on aggression using ERPs during drug/placebo conditions. The amplitudes of P3 waveforms among impulsive aggressive subjects were increased significantly during the phenytoin condition but not during the placebo condition, suggesting phenytoin decreases impulsive aggressive acts. Studying the relationship among impulsiveness, aggression, reading, and the P3, Harmon-Jones et al [98] reported that attentional impulsiveness and non-

planning impulsiveness were positively correlated with physical aggression, negatively with reading level, and negatively with the amplitude of the parietal P3 in an oddball and continuous performance task (CPT). In contrast, motor impulsiveness was not related to aggression or reading level, but to the amplitude of the parietal P3 in the oddball task. In summary, individuals with anger, aggression and violence showed various deficits, but predominantly showed reduced P3 amplitudes in several task paradigms.

ERO Studies

ERO studies examining anger, aggression and violence in clinical samples are quite rare. However, many studies have investigated correlates of brain oscillations related to impulsive aggression and emotions in healthy subjects. For example, Salminen and Ravaja [146] examined ERO activity during affective processes related to a violent digital game consisted of wounding and killing events. It was found that the wounding event evoked increased occipital fast theta (6-8Hz) response and the killing event evoked slow alpha (8-10Hz) asymmetry over the central electrodes, both relative to the pre-event baseline. The same authors, in an earlier study [147], examined oscillatory brain responses evoked by video game events consisting of win (success) and loss (failure). Success events evoked decreased theta activation at central electrodes, decreased fast alpha activation at frontal electrodes, and increased beta activation at frontal electrodes, while loss events evoked decreased central theta activation and increased fronto-central beta activation. In another study, Sheikholeslami et al [148] used high resolution EEG to examine dynamic brain activity during video game play in healthy human subjects, and found that the frontal midline theta-wave activity increased over time while the parietal alpha-wave activity initially decreased followed by a slow increase relative to baseline resting condition. Further, Houston et al [149] assessed EEG activity at rest and during photic stimulation in an impulsive group and a non-aggressive control group, and observed consistently lower frontal delta and theta activity during both conditions. During visual and auditory stimulation conditions, Koelsch et al [150] observed that individuals with moderate intermittent explosive disorder (mIED) showed increased beta power and decreased theta power. In summary, increased theta ERO seems to be an index of aggression during a "game" situation in normal subjects, while decreased low frequency (delta and theta) activity and increased beta activity represents aggressive individuals during stimulus processing.

ANTISOCIAL SPECTRUM DISORDERS

The term "antisocial spectrum" indicates a constellation of behaviors that include aggression, psychopathy/sociopathy, CD, oppositional-defiant disorder (ODD), and ASPD (cf. [151]). Impulsivity is one of the core features of antisocial personality [152], conduct disorder [153,154], and criminal behavior [155-157]. Although there is good agreement regarding the assessment and behavioral correlates of psychopathy, relatively little is known about the neurocognitive processes implicated in the disorder. Psychopathy and aggression are shown to involve attentional and cognitive processes (for reviews, see [158-160]).

Electrophysiological studies, as discussed below, elucidate our understanding of neurocognitive processes underlying these disorders.

EEG Studies

EEG abnormality in psychopathy[1] and criminality has been reported since the 1940s [161]. McCord and McCord [162] defined a psychopath as "an asocial, aggressive, highly impulsive person, who feels little or no guilt, and is unable to form lasting bonds of affection with other human beings (p.3)." Most of the early studies reported that the incidence of EEG abnormalities among psychopaths was between 47% and 58% [163-172] (cf.[161]). The abnormalities reported in these studies are slow activity [167,168,171,172], very slow activity [167,168], and paroxysmal activity [168,169]. Although it is possible that most of the subjects with psychopathy and criminality were impulsive, only one study by Hodge [168] has mentioned the term 'impulsive psychopath' as the study group. Summarizing these early EEG findings on psychopaths, Ellingson [161] concluded that a significantly high proportion (about half) of patients with psychopathic diagnoses have abnormal EEG. In later studies, for example, Winkler and Kove [173] reported that of 55 subjects charged with murder or manslaughter had abnormalities in the EEG. Sayed et al [174] reported that 66% of the subjects with murder backgrounds were classified as having some EEG abnormality. Hill [175], in his review, stated that the incidence of abnormality in the EEG of criminal subjects was probably much the same as that found in admissions to the acute wards of a mental hospital (about 50 per cent) [cf. 176]. In a longitudinal study, Mednick et al [177] examined the possibility of EEG as a predictor of antisocial behavior and found that slower alpha patterns proved to be characteristic of later development of delinquent behaviors. Despite these findings, an association between EEG abnormalities and violent behavior was not found by all investigators [176,178].

While earlier studies were generally more qualitative, EEG technology has become increasingly more advanced, allowing for detailed quantitative computerized analyses instead of clinical visual inspection [179]. Further, EEG studies in comparison with ERP studies have become far less common in recent years, and EEG studies have predominantly reported region specific impairments. For example, Pillmann et al [130] examined the relationship of EEG abnormalities and violent criminal behavior in 222 defendants referred for psychiatric evaluation and found focal abnormalities, especially of the left hemisphere, were related to a significantly higher number of violent offenses. Gatzke-Kopp et al [180] investigated both EEG and positron emission tomography (PET) in fourteen murderers and reported that the EEG revealed significant increases in slow-wave activity in the temporal, but not frontal lobe in murderers, in contrast to prior PET findings that showed reduced prefrontal, but not temporal glucose metabolism; taken together, these studies suggest that resting EEG measures offer empirical utility in terms of topographic brain activation distinct from PET. A recent study by Reyes et al [179] investigated two groups of violent offenders (with and without the diagnosis of ASPD) and observed high incidences of EEG abnormalities, such as electrogenesis alterations, attenuated alpha rhythm and increased theta and delta activities in

[1] The term 'psychopathy' was replaced by a more inclusive term 'antisocial personality disorder' since the publication of DSM-III in 1980.

the frontal lobe. In summary, individuals with history of psychopathy, criminality/violence, and antisocial personality disorder showed several EEG abnormalities, predominantly qEEG findings showing increased slow wave (delta, theta and alpha) activity.

ERP Studies

Several studies have examined the ERP components (especially P3 and N2) and found them to be abnormal in antisocial and psychopathic individuals. Reduced amplitude of the P3 component has been postulated as a neurobiological marker for "antisocial spectrum" [151], a constellation of behaviors that include aggression, psychopathy, conduct disorder, oppositional-defiant disorder, and antisocial personality disorder [181,182]. Although impulsivity is a core concept within the definition of psychopathy and antisocial personality, very few ERP studies have attempted to associate P3 amplitude reduction with psychometrically measured impulsivity. Barratt et al [183] found that prison inmates with antisocial personality disorder showed reduced P3 amplitude and increased impulsivity compared to a matched non-inmate control group. Recently, Carlson [184] evaluated the relationship between P3 and psychopathic personality traits in normal undergraduate students, and found that 'Self-Centered Impulsivity' factor was related to P3 reduction, especially at the frontal loci. Although many studies have found reduced P3 amplitudes among individuals with violent offenses [144,185], conduct disorder [186-188], and aggression [98], studies have also observed significantly enhanced amplitudes in psychopaths when compared to non-psychopaths [189,190], or no difference between psychopaths and non-psychopathic controls [191,192]. Possible reasons for the inconsistent findings may be related to differences in the task paradigm and modality used in these studies. In summary, although the findings showing P3 abnormality in psychopathy are inconclusive, under certain task conditions, there is evidence to suggest that the P3 is reduced in psychopaths [151,193,194].

Regarding the N2 component, Kiehl et al. [193] demonstrated increased N2 amplitude during an auditory oddball task in psychopathic subjects. In contrast with Kiehl's results, Munro et al. [195] detected a reduced N2 component in psychopathic individuals in a Go-NoGo task. However, In a recent study using an auditory oddball paradigm, Perdeci et al [194] found no difference in N2 amplitude between the impulsive antisocial group and the healthy control group. The results for the N2 components are contradictory possibly due to methodological differences across studies, and further studies are required. Taken together, although N2 findings are not robust compared to P3 findings, they suggest specific neurocognitive impairments associated with psychopathy and aggression.

ERO Studies

To our knowledge, there are as yet no ERO studies on psychopathic and antisocial individuals. However, a few studies have investigated correlates of brain oscillations related to impulsive aggression and emotions in healthy subjects. See the previous section for details.

ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD)

ADHD in children and adolescents is characterized by excessive restlessness and an extremely poor concentration span, resulting in impulsive and disruptive behavior [196]. Researchers have more often linked the construct of impulsivity to ADHD than to any other disorders. In the recent decades, considerable effort has attempted to delineate the electrophysiological underpinnings of ADHD and associated impulsivity in terms of EEG, ERP and ERO measures, as discussed below.

EEG Studies

EEG abnormalities in ADHD have been summarized by Barry et al [197]. Early qualitative studies used visual evaluation of paper recordings of the EEG, and many reported percentage differences in abnormalities between clinical and control groups rather than identifying the exact nature of the underlying abnormality [197]. On the other hand, later quantitative studies used computer-aided analysis and identified increased activity in the delta and theta bands and lower levels of beta activity in children and adolescents with ADHD than normal controls. For example, Mann et al [198] found increased theta and decreased slow beta in boys with ADHD. Matsuura et al [199] reported that children with ADHD had more delta and fast theta activity than age matched controls and a group of children with deviant behaviors. Ratio coefficients such as theta/alpha and theta/beta ratios have been analyzed to differentiate ADHD from control subjects [200,201], especially showing higher theta/beta ratio in ADHD subjects [202-205]. Recent studies have also shown EEG asymmetry in alpha [206], beta band [207].

Studies on EEG coherence in ADHD subjects have yielded some interesting findings. For example, Montague [208] found reduced interhemispheric coherences and elevated intrahemispheric coherences in hyperkinetic children. Chabot and Serfontein [209] found that ADHD was associated with increased coherence in frontal and central regions and reduced coherence parietally. Barry et al [210,211] found differences in intrahemispheric and interhemispheric coherences in ADHD, suggesting compromised neural communication across brain regions in ADHD. Interestingly, Barry et al [211] found that coherence was globally elevated in children with the ADHD combined subtype compared with both the ADHD inattention subtype and control children, and suggested that this elevation in coherence may be directly related to the hyperactivity and impulsivity unique to the combined subtype. In a recent study, Barry et al [212] found negative correlations between ADHD coherence anomalies and ADHD symptoms, and suggested that ADHD may reflect anomalous frontal right-hemisphere linkages that help compensate for functional brain anomalies in the left frontal regions in this disorder.

ERP Studies

Since the early 1970s, ERP studies have revealed deficits in information processing in ADHD [213-216]. Recently, Barry et al [217] reviewed the ERP studies that explored various

aspects of brain functioning in ADHD, and concluded that a complex range of ERP deficits has been associated with the disorder; these deficits were identified in preparatory responses, auditory and visual information processing, and in the frontal inhibitory system. Barry et al [217] also pointed out that there are many contradictory findings across studies due to differences in sample characteristics, task details, and data analysis techniques. For example, while studies showed that CNV amplitude was reduced in ADHD subjects compared to controls in some studies [33,218-220], others failed to find differences [221,222].

Several studies have examined the preparatory processes in ADHD, showing reduced CNV amplitude [33,218-220,223], atypical lateralization and topography [222,224,225], impaired attentional orienting to primary task stimuli or cues [219], and dysfunctional pre-motor processing [226]. Further, a large number of studies have reported ERP abnormalities in both early and late components, and in auditory and visual modalities [217]. For example, the predominant findings in auditory tasks are that ADHD subjects showed decreased N1 amplitude [54,227,228], increased N1 latency [229], larger P2 amplitude [54,229,230], shorter P2 latency [229], reduced N2 amplitude [52,54,231-233], increased N2 latency [53,231,234], reduced P3 amplitude [52-54,228,230,235-238] and longer P3 latency [239]. In visual paradigms, ADHD subjects have shown reduced P1 amplitude [226,228,240], longer P1 latency [241], larger N1 amplitude [214], delayed N1 latency [241,242], larger P2 amplitude [214,243-245], delayed P2 latency [242], larger N2 amplitudes [243,244], delayed N2 latency [242], reduced P3 amplitude [202,221,226,228,236,244-250], and delayed P3 latency [221,246,251,252]. In summary, the predominant ERP findings in ADHD subjects are increased P2 amplitude and reduced N1 and P3 components in both the auditory and visual modalities, while the latency findings are equivocal.

Recent studies have focused more on frontal inhibitory systems and executive functions in children with ADHD [253,254] by using specific paradigms, such as the Go-NoGo task and the stop-signal task. Using the stop-signal task with an auditory stop signal, frontal N2 amplitude to the stop stimulus was dramatically reduced in the ADHD combined subtype [225]. Using a visually-presented stop signal, topographically-altered P2/N2 potentials were reported in a delayed Go task, with controls showing additional differential processing between Go and NoGo stimuli [224]. Similar results have been reported using the standard Go-NoGo paradigm, showing that ADHD children had a smaller NoGo-minus-Go N2 effect than controls [255]. These results support the hypothesis of an inhibition-regulation problem rather than a central inhibition deficit in ADHD [217].

A caveat to the above findings is that there are studies showing negative or even no findings. Differences between studies may be due to task specific results, differences in subject sample characteristics and the interpretation of results for a specific finding may vary according to the disorder being studied. For example, contrary to the finding of delayed latency, shorter latencies in P2 [54,252] and N2 [252] were also reported. Sunohara et al [252] postulated that reduced P2 latency in ADHD reflects rapid and atypical stimulus-feature detection, generally reflecting impulsivity, while a shorter N2 latency in ADHD suggests rapid and atypical stimulus classification, or attention to stimulus features. A detailed discussion of ERP findings in ADHD can be found in the review by Barry et al [217].

ERO Studies

There are only a few ERO studies on ADHD. Sukhodolsky et al [256] reviewed physiological, imaging, and neuropsychological data of tic disorder (TS) and symptoms of ADHD, and suggested that abnormal neural oscillations may have a prominent role in the co-occurrence of TS with ADHD. Lazzaro et al [234] found that compared with controls, ADHD patients showed increased prestimulus EEG theta activity, which was interpreted to contribute to their ERP differences. Alexander et al [257] examined ERO measures during both an auditory oddball and a visual continuous performance task (CPT) found that ADHD subjects had less activity at low frequencies (approximately 1 Hz) during both tasks. For auditory oddball targets, this effect was shown to be related to smaller P3 ERP amplitudes. During CPT, the approximately 1 Hz wave activity in the ADHD subjects was inversely related to clinical and behavioral measures of hyperactivity and impulsivity. The authors concluded that low frequency wave activity (at 1 Hz) associated with levels of hyperactivity and impulsivity may be a new marker for ADHD. Further, a couple of studies have reported abnormal evoked gamma activity in ADHD. Yordanova et al [258] analyzed phase-locked gamma band (31-63 Hz) responses between 0-120 ms poststimulus using an auditory selective-attention task in ADHD children and matched healthy controls, and found that ADHD children produced larger and more strongly phase-locked gamma activity than controls, suggesting impaired motor inhibition. In another study, Lenz et al [259] reported enhancement of gamma band activity in ADHD subjects at parieto-occipital areas in response to the task stimuli in a visual recognition task, but this enhancement was not correlated with correct recognition performance as evidence of enhanced excitation levels and unspecific activation of processing resources in ADHD patients. However, more studies are needed to confirm this phenomenon. It is therefore suggested that future studies focus on ERO measures to further establish frequency specific contributions to characterize neurocognitive dysfunctions as correlates of impulsivity in ADHD.

ALCOHOL USE DISORDERS (AUDS)

Alcohol addiction is a major health problem affecting millions worldwide, thus causing health, economic and sociocultural problems. Alcoholism is a disorder of dysregulation and poor impulse control ([260,261]). Recent studies have revealed that alcohol/drug addiction has been strongly linked with the concept of impulsivity [262,263]. Many researchers have considered impulsivity as the key vulnerability marker for substance-use disorders, especially alcoholism [264,265](see Verdejo-Garcia et al [3] for a review). Recently, it was also found that impulsive behavior in humans predicts the onset of drinking during adolescence and alcohol use disorders (AUDs) in adulthood [266]. In this regard, electrophysiological findings relating alcohol dependence as part of the cluster of impulsive disorder, as discussed below, may offer a better scope of understanding both tenets of alcoholism and impulsivity.

EEG Studies

A considerable number of EEG studies have been performed in alcoholic subjects, and the primary findings have been in theta, alpha, and beta bands. For example, a number of studies have reported increased resting theta power in abstinent alcoholics [267-269]. The theta power increase (in the resting EEG) is considered to be an electrophysiological index of an imbalance in the excitation–inhibition homeostasis in the cortex (cf. [10]); increased theta in alcoholism may be a marker for disinhibition and impulsivity. A related finding is that elevations in theta power have been observed during administration of alcohol in proportion to increasing blood alcohol level [270,271]. With regard to alpha band, findings showing changes in alpha power in alcoholics are often contradictory. However, low-voltage alpha (LVA), has been shown to be associated with a subtype of alcoholism that is associated with anxiety disorder [272]. Ehlers and Phillips [273] found that the presence of the LVA variant was not associated with drinking status, family history of alcoholism, or a personal history of anxiety disorders, but was associated with significantly higher extroversion scores. In other words, LVA may be related to extroversion related traits such as impulsivity and disinhibition associated with alcoholism and possibly other externalizing disorders. Beta rhythm is considered to be a neurophysiological index in the study of predisposition to alcoholism [10]. Increased beta power in the resting EEG of alcoholics has been well documented [268,274-277]. Studies have also reported increased beta power in the EEG of relatives of alcoholics [278-281], suggesting that increased beta power is not a direct effect of alcohol use, but may antecede the development of alcoholism [10]. A genetic link of a $GABA_A$ receptor gene with the EEG beta band [282] and diagnosis of alcohol dependence [283] has also been established. Further, findings showing specific deficits in GABA benzodiazepine receptors in the brains of alcoholics [284,285], and individuals at risk [286] suggest neural disinhibition (hyperexcitability) that may be involved in the predisposition to develop alcoholism [10]. Taken together, increased beta band in the resting EEG is a primary characteristic feature in alcoholics and high-risk subjects; increased theta band increases in the alcoholics may also be significant, while the findings on alpha power are equivocal.

ERP Studies

Most studies investigating ERP deficits in abstinent alcoholics and their offspring at high risk (HR) to develop alcoholism have focused on the P3 component. Studies have found that alcoholics and HR subjects manifest significantly reduced P3 amplitude, especially in visual oddball tasks [287-300]. Recently, low P3 findings in alcoholics and HR subjects were reported in other tasks such as Go-NoGo [260,261,301-307] and gambling tasks [308,309]. Other ERP components in which alcoholics showed aberrations are N2 and mismatch negativity (MMN), although the findings are not consistent. MMN is reflective of a mental mechanism for automatic stimulus-change detection [310], and was found to be larger in alcoholics [311,312] and in HR [313]. On the other hand, while several studies reported reduced N2 component in alcoholics [308,309,314-322], others have reported either a larger N2 (e.g., [323-325]) or no difference in N2 amplitudes (e.g., [326,327]), or delayed N2 latency (e.g., [315,328].

Regarding the association among impulsivity, P3, and alcoholism, there are a few studies worth a discussion here. Chen et al [263] assessed the relationship between impulsivity and alcohol dependence, and the correlations with P3 amplitude. They found significant negative correlations between impulsivity scores (assessed by BIS) and P3 amplitude in a sample of alcoholics and controls. Further, low resolution brain electromagnetic tomography (LORETA) showed significantly reduced activation in the frontal regions in alcoholic subjects and highly impulsive subjects, suggesting that impulsivity may be a central factor that underlies the pathogenesis of alcohol dependence, and that this may involve deficits in frontal regions. In another study, Kamarajan et al [309] showed that alcoholics, as compared to normal controls, showed 1) significantly lower N2 and P3 amplitudes for the outcome stimuli, 2) decreased P3 current density at cingulate gyrus and less N2 current density at sensory and motor areas, and 3) higher levels of impulsivity and risk-taking features, suggesting that alcoholics may have a dysfunctional reward circuitry in the brain. Taken together, as summarized by Porjesz et al [10], one of the most consistent and robust findings in the ERP literature is the reduced P3 amplitude in alcoholics and in offspring at risk prior to alcohol exposure.

ERO Studies

A consistent and robust ERO finding is that alcoholics and their high risk offspring show decreased delta and theta power during the post-stimulus P3 time window in visual oddball and Go-NoGo tasks [329-332]. Since several studies have demonstrated that P3 responses are primarily the outcome of theta and delta oscillations elicited during stimulus processing [333-338], the findings of decreased delta and theta power are in agreement with decreased P3 amplitude in alcoholics and HR subjects; furthermore, they but can tease apart the relative contributions of these frequency bands spectrally and topographically, which can further enhance our understanding. Reduced theta and low alpha reflect the failure of the inhibitory control at the cognitive level [339]. Since theta activity at the cellular level is produced by the inhibitory interneurons of the hippocampus [340], and theta frequency is often related to inhibitory functions [341], the findings assume critical importance regarding the association between theta band, inhibition/impulsivity and alcoholism. Further, reciprocal synchronization has been observed in the theta range between hippocampus and frontal and parietal regions in the brain during attentional tasks [78], suggesting theta band to have important “networking” properties in the brain during cognitive processing. In a recent study, Kamarajan et al [342] showed the relationship among the factors of ERO measures, impulsivity, and alcoholism. In this study, ERO theta power in the time window of 200-500 ms was compared across alcoholic and control groups. A gambling task used in this study involved outcomes of either loss or gain of an amount (10 or 50 cent) that was bet. The alcoholic group showed significantly decreased theta power during reward/outcome processing compared to controls. A strong association between reduced anterior theta power and impulsive task-performance was observed. Furthermore, alcoholics exhibited increased impulsivity and risk-taking on the behavioral measures.

Dysfunction in the early evoked gamma band (around 100 ms poststimulus) was also observed in alcoholics [343] as well as in HR subjects [344]. This early gamma activity occurs at the P1 and N1 time window and represents an important processing step related to

the selection and identification of target stimuli, indicative of a top-down mechanism involved in selective attention [345]. This phase-locked gamma is larger to attended compared to unattended stimuli, particularly over frontal regions [258,334]. Since gamma band is associated with inhibitory functions, any aberration in the gamma system may represent either disinhibitory and/or impulsive traits, and therefore these findings may assume importance to unify the factors of impulsivity, oscillations, and externalizing disorders such as alcohol/drug dependence. In summary, ERO abnormalities in alcoholism include decreased delta and theta activity in the N2-P3 time window, along with reduced early gamma activity in the P1-N1 time window, showing deficits in both early and late cognitive processing stages.

SUBSTANCE USE DISORDER (SUDS)

Drug addiction is a common mental and brain disorder that causes damage at multiple levels to the individual, family and to society. Drug addiction may be defined as a chronic, often relapsing disorder characterized by obsession, compulsion, or physical or psychological dependence [346]. Researchers have proposed that impulsivity is a major factor in drug addiction [347-349]. Bechara [347] suggested that addiction is the product of an imbalance between two separate, but interacting, neural systems that control decision making: an impulsive amygdala system for signaling pain or pleasure of immediate prospects, and a reflective prefrontal cortex system for signaling pain or pleasure of future prospects. Koob & Le Moal [348,349] theorized that drug addiction involves drug-taking behavior that progresses from impulsivity to compulsivity in a three-stage cycle: binge/intoxication, withdrawal/negative affect and preoccupation/anticipation. Further, Franken [350] showed that compared to control subjects, drug addicts had higher Behavioral Activating System (BAS) scores, which are known to be correlated with impulsivity. Altered intrinsic amygdala functional connectivity (iAFC) network connectivity has been suggested to contribute to the loss of impulsive control in heroin dependent subjects [351]. It has been empirically shown that oral methylphenidate normalizes cingulate activity and decreases impulsivity in cocaine addiction during an emotionally salient cognitive task, suggesting that impulsivity is a core component in drug addiction [352,353].

Although electrophysiological studies of substance use disorders have focused on alcoholism more than on drug addiction (for reviews, see [10,354-357]), there are some interesting studies regarding stimulants (cocaine, nicotine, and amphetamines), cannabinoids/hallucinogens (cannabis/marijuana, MDMA/ecstasy, and LSD), and opiates (heroin, morphine, and codeine), which are discussed below.

Cocaine, originally a derivative from coca plant *Erythroxylon coca*, is a strong central nervous system stimulant and causes increased energy, reduced fatigue, and mental alertness by increasing the levels of dopamine, a brain chemical (or neurotransmitter) associated with reward and pleasure. Nicotine or tobacco is one of the most heavily used addictive drugs, and has stimulating effects by activating reward pathways in the brain. Amphetamine, a synthetic compound related to the plant derivative *ephedrine*, is a psychostimulant drug which produces increased wakefulness and focus in association with decreased fatigue and appetite. Cannabis/marijuana, made from the hemp plant *cannabis sativa*, is the most commonly used

illegal drug in the U.S. Short-term effects of marijuana use include euphoria, distorted perceptions, memory impairment, and difficulty thinking and solving problems. MDMA (methylenedioxymethamphetamine) or 'ecstasy' is a synthetic drug with stimulant and psychoactive properties. Short-term effects of MDMA include feelings of mental stimulation, emotional warmth, enhanced sensory perception, and increased physical energy. LSD (lysergic acid diethylamide) is a semisynthetic psychedelic drug that can distort perceptions of reality, alter thinking process and produce hallucinations. Opiates such as heroin, morphine, and codeine are the alkaloids derived from the poppy plant *(Papaver somniferum)* and have been used for centuries to relieve pain. Opiates elicit their powerful effects (such as pleasure, reward, and pain relief) by activating opiate receptors in the brain and body. Electrophysiological studies in various drug categories are reviewed below. Studies of acute effects of drugs in humans and animals are also included when there are few (or no) studies available with respect to a specific AUD condition.

EEG Studies

EEG studies investigating drug addiction have mainly performed power spectral analyses in different frequency bands. A consistent finding across these studies is that drug dependent individuals have shown increased beta activity in the resting EEG. Often, other frequency bands showed opposing trends between different drugs. For example, cocaine dependent subjects showed decreased low frequency activity [358] while methamphetamine addicts showed increased low frequency activity [359], although both substances are stimulants.

Stimulants

Cocaine: Human electrophysiological research literature on stimulants has focused primarily on cocaine. The qEEG studies have reported increased alpha activity [360-362], decreased delta activity [360,362-364], and increased beta power [361,363,365] in cocaine dependent patients during eyes closed, resting conditions. These abnormalities, primarily found in anterior cortical regions, were shown to correlate with the amount of prior cocaine use [362,364,366].

Nicotine: In cigarette smokers, the resting EEG profile in response to acute nicotine intake by smoking is similar to that seen with other psychostimulants, i.e., increased power in high alpha and beta band and decreased power in delta, theta, and low alpha bands [367-370]. Further, it was shown that nicotine abstinence negatively affects cognitive functioning while nicotine administration or smoking restores it [371,372].

Amphetamines: Methamphetamine dependent individuals had increased EEG power in the delta and theta bands [359]. Further, increased theta power was associated with poorer performance on reaction time and for a working memory task in the methamphetamine-dependent sample, but not in the non-drug-using volunteers [373]. Comparatively, patients with cocaine dependence had reductions in low frequency activity [358,360,364] while methamphetamine dependent volunteers had changes in the opposite direction (i.e. increased low frequency activity)[359]. This difference may be due to the differential neurotoxicity of methamphetamine compared to cocaine [374].

Cannabinoid/Hallucinogens

Cannabis: Struve et al [375-377] demonstrated and replicated a significant association between chronic marijuana use and topographic qEEG patterns of persistent ‘‘alpha hyperfrontality’’ (i.e., elevations of absolute and relative alpha power, and interhemispheric coherence over frontal cortex) as well as reductions of alpha mean frequency. Following acute marijuana use, studies have reported transient increases in alpha power and decreases in beta power in simple or passive tasks [355,377-380]. Further, there was an association between duration of marijuana use and reductions in alpha and beta power at posterior electrode sites [381].

MDMA: Acute effect of MDMA/ecstasy on EEG is the decrease of theta and alpha power after MDMA administration [382,383]. MDMA users showed increased absolute delta power compared to marijuana abusers and controls [384]. MDMA users have greater slow wave activity, a finding that has been previously associated with a number of other psychiatric conditions (e.g., schizophrenia, aggression)(cf. [357]).

LSD: In 1952, Delay et al [385] first described the phenomenon of accelerated alpha frequencies in rabbits following the acute administration of LSD (cf. [386]). This phenomenon was subsequently verified in cats [387] and humans [388-391]. Recently, Abraham and Duffy [386] confirmed alpha acceleration in patients with LSD-induced hallucinogen persisting perceptual disorder (HPPD), and suggested that this might represent LSD-induced cortical disinhibition.

Opiates

Heroin: EEG changes in heroin addicts are decreased alpha rhythm, increased beta activity, and a large amount of low amplitude delta and theta activity in central regions [392]. Davydov and Polunina [393] found that length of heroin intake predicted the elevations of high alpha activity in right hemisphere. Franken et al. [394] found that abstinent heroin-dependent subjects have an enhanced fast beta power compared with healthy controls. Further, spectral power in heroin addicts strongly related to abstinence length [392,395]. Abnormal functional connectivity as detected by EEG synchrony across brain regions has also been reported in heroin addicts, especially in the beta frequency band [396].

Morphine: Studies on morphine use or morphine-related EEG changes in human subjects are rare. However, several animal studies that examined the acute morphine effects have reported that morphine produced dose dependent EEG slow-wave bursts [397-399] and an increase in the total power spectra [398,400,401].

In summary, a consistent EEG finding in drug addiction is increased resting beta band activity, suggesting a possible dysfunction in the CNS activation or reactivity. Further, power modulations in several frequency bands in several drug conditions may also indicate abnormal neural processing in the addicted individuals. However, findings related to acute effects of a drug yielded neither consistent findings nor association with any impulsivity phenomena.

ERP Studies

One of the most consistent and robust ERP findings in drug addiction is decreased P3 amplitude [402,403]. In general, reduced P3 amplitude has been observed in individuals who

abuse illicit substances [404,405] and in their offspring at risk for substance use disorders (SUD) [406]. Recent studies have confirmed that individuals with substance use and misuse have reduced P3 amplitude (e.g. Yoon et al [407]), and prospective studies have predicted that those who would eventually develop SUD show smaller P3 early in life [403,408]. Specific ERP (and evoked potential) findings regarding stimulants, cannabinoid/hallucinogens, and opiates are discussed below.

Stimulants

Cocaine: In a reward processing task, Parvaz et al [409] reported that P3 differentiations across reward conditions were absent in individuals with cocaine use disorders. Similarly, Goldstein et al [410] observed that individuals with cocaine use disorders showed a lack of modulation of the P3 between monetary reward versus nonreward conditions, and this impairment correlated with frequency of recent cocaine use. Evidence suggests that cocaine dependent patients may have a disruption in the brain's error processing system as indicated by reduced ERN [411,412] and error P3 amplitudes [411]. Further, ERP correlates of cocaine craving (i.e., augmented late positive waves) have been elicited by Franken et al [413,414].

Tobacco: Anokhin et al [415] reported decreased P3 amplitude in a visual oddball task among active and former smokers in comparison to never-smokers. Recent studies showed that smokers also showed deficits in a Go-NoGo paradigm with decreased NoGo N2 [416], and in ERN paradigms with reduced ERN and P3 amplitudes [417]. ERPs of smoking related cues have also been elicited [418,419].

Amphetamines: Prolonged P3 latency in the auditory oddball task was reported in methamphetamine dependent volunteers compared to normal controls [420,421]. McKetin and Solowij [422] found slowed reaction time and reduced early processing negativity during an auditory selective attention task in severely dependent amphetamine users, suggesting deficient sensory processing. Interestingly, in normal volunteers, acute amphetamine administration decreased the P3 latency [252,423] and blocked P50 (sensory gating) suppression [424].

Cannabinoid/Hallucinogens

Cannabis: ERP changes have been observed in chronic users as well as during acute cannabis administration. For example, acute cannabis exposure has caused decreased P3 amplitude [425-427], enhanced word recognition after 250 ms poststimulus [428], and reduced MMN amplitude [429]. On the other hand, chronic cannabis users during the unintoxicated state exhibited reduced P3 amplitude [430,431], delayed P3 latency [430,432], reduced visual N160 amplitude [433], reduced MMN amplitude [434], larger late positivity [435], and amplified early processing negativity for irrelevant stimuli [436]. Further, the Stroop paradigm has established an association of poor Stroop performance with cannabis use in terms of earlier onset [437], longer duration [438], and higher frequency [439].

MDMA: Lower P3 amplitudes were also found in a Go-NoGo task among MDMA users [440]. Recently, in a memory task, Burgess et al [441] found that Ecstasy/polydrug users showed an attenuated late positivity (at about 400-800 ms) over left parietal scalp sites, a component associated with the specific memory process of recollection. Further, chronic MDMA users had larger N1 and P2 amplitudes as well as N1/P2 slopes than the control group [442]. In a visual oddball task, Mejias et al [443] reported that MDMA abusers

displayed reduced N170 amplitude reflecting deficient attentional resources, as well as longer P3b latency showing a delayed decision-making and premotor response compared to controls.

LSD: ERP studies on LSD use is rare, although a few evoked potential studies have been reported. Administration of LSD significantly attenuated the flash-evoked cortical potential in rats and thus produced deficits in neural conduction in the retino-geniculato-cortical system [444]. Similar neural damage in evoked responses has also been reported in cats [445,446]. In a study on human subjects, Abraham and Duffy [386] reported that LSD dependent patients showed shorter latency in visual evoked response, suggesting abnormally accelerated neural sensory processing.

Opiates

Heroin: There are only a handful of ERP studies on the effects of heroin. Using visual ERP recordings, Bauer [405] reported that opioid-dependent abstinent subjects exhibited decreased P3 amplitudes. Papageorgiou et al [447] reported that abstinent heroin addicts exhibited significant reduction of P3 amplitude compared to current heroin addicts and controls in a memory task. Interestingly, Kouri et al [448] reported that chronic heroin- and cocaine-dependent individuals under the influence of heroin or heroin and cocaine demonstrated normal P3 amplitudes in an oddball paradigm, while they manifested reduced P3 amplitudes during detoxification. In response to stimulus cues, P3s elicited by opiate-related stimuli were significantly larger than those elicited by neutral stimuli in the opiate addicted group [449,450]. A heroin-dependent group also demonstrated a startle-elicited P3 attenuation which further predicted future heroin use [451].

Morphine: While there are no ERP studies on the effects of morphine, a few studies have used evoked potentials. Sinitsin [452] reported that high doses of morphine enhanced most components of the visual evoked potential in cats, suggesting a change in the cortical excitability. Nowack et al [453] also showed that high-dose morphine increased the thalamocortical-augmenting response in cats. Kuroda et al [454] found that morphine administration caused a significant increase in the amplitude of early and late visual evoked potential components (P1–N1, N1–P2, P3–N3 and N3–P4). In humans, it was reported that morphine administration decreased the pain sensitivity and the pain related evoked potentials [455].

In summary, ERP studies on addiction (except for opiates) have demonstrated reduced P3 amplitude and increased P3 latency, coupled with changes in early components. In response to cues, larger P3 as well as late positive potential responses to drug pictures compared with neutral pictures, showing drug-dependent adults exhibiting much greater responses to drug-related pictures than healthy adults for several drugs including heroin, cocaine and marijuana.

ERO Studies

ERO studies in drug addiction are relatively few in number due to the fact the ERO techniques are more recent. Among these studies, many have reported changes in evoked gamma activity in drug dependent individuals and a few have examined the event-related (de)synchronization (ERD/ERS).

Stimulants

Cocaine: To our knowledge, there is only a single ERO study on cocaine. Horrel et al [456] reported that evoked gamma showed decreased power to non-target and to a lesser extent target drug-related cues at all brain topographic areas (left, right, frontal, parietal, medial, inferior), while induced gamma power decreased globally to both target and non-target drug cues in current cocaine abusers.

Tobacco: ERO studies on tobacco are also very rare. In humans, Crawford et al [457] found that smokers generated significantly larger gamma band oscillations compared to never-smokers. In rats, Phillips et al [458] found that acute nicotine increased the normal burst of evoked gamma oscillations following an auditory stimulus.

Amphetamine: There are as yet no ERO studies on amphetamine in humans. In behaving rats, administration of amphetamine caused a prolonged decrease in fast gamma power (about 50 Hz) and increase in ultra-fast gamma power (about 80-100 Hz) [459].

Cannabinoid/Hallucinogens

Cannabis: ERO studies on cannabis are very few in number. In 2004, Ilan et al [425] observed a spatially distributed decrease in task-related theta-band power after marijuana ingestion. These results were further confirmed by the same group of researchers [426]. Recently, Edwards et al [460] found that while cannabis users exhibited attenuation in poststimulus high frequency activity in the beta range (13-29 Hz) and in the gamma range (30-50 Hz) compared to control group, greater levels of cannabis use were positively associated with poststimulus gamma power.

MDMA: To our knowledge, there are as yet no ERO studies on MDMA.

LSD: To our knowledge, there are as yet no ERO studies on LSD.

Opiates

Heroin: To our knowledge, there are as yet no ERO studies on heroin.

Morphine: There are as yet no ERO studies on amphetamine in humans. In an animal study, Marczynski et al [461] found that administration of morphine in cats trained to press a lever for milk reward showed dose-dependent variations in postreinforcement EEG synchronization and the reward contingent positive variation.

Taken together, among the ERO studies on effects of drug and addiction, many have reported evoked gamma power changes in response to direct effect of the drug or in drug dependent individuals, and a few studies have reported changes in ERD/ERS. There is also a study on high risk offspring. Brigham et al [462] found alpha desynchronization and significantly longer latency of alpha synchronization in the young offspring of substance dependent individuals compared to control subjects.

BORDERLINE PERSONALITY DISORDER (BPD)

Impulsivity is a core feature in borderline personality disorder (BPD) [463-469]. In a detailed review of electrophysiological aberrations in BPD, Boutros et al [470] reported that electrophysiological research is scarce, and that many studies are hampered by

methodological limitations. On the other hand, some recent studies have used novel cognitive paradigms to examine the ERP correlates in BPD patients as discussed below.

EEG Studies

By the mid-1980s, the presence of significant EEG abnormalities in BPD patients was well documented (see Boutros et al [470] for a review). According Boutros et al [470], there were two types of standard EEG abnormalities in BPD patients. The first was the presence of epileptiform discharges (e.g., [471,472]) indicating decreased threshold for seizure-like activity or increased cortical excitability. The second was the presence of diffuse EEG slowing (e.g., [473-475]), suggestive of either a metabolic or a degenerative brain disorder. Further, the most frequently reported sleep architectural change in BPD patients is shortening of REM latency [476-479] (cf. [470]). A recent sleep EEG study reported that BPD patients showed a tendency for shortened REM latency and significantly decreased stage-2 NonREM sleep, coupled with increased delta power in both NREM and REM sleep [480].

ERP Studies

The majority of the ERP studies in BPD patients have reported decreased P3 amplitudes and delayed latencies compared to healthy control subjects [481,482]. In a visual Stroop task, Houston et al [483] reported a significant reduction in P3 amplitude in adolescents with BPD features. Studies have reported that BPD patients have also manifested reduced amplitude in ERP components of specific cognitive tasks, such as ERN/Ne (error-related N2) [484] and NoGo-P3 (P3 for the 'NoGo' condition) [485]. A recent study of emotional regulation [486] reported that borderline patients displayed larger late positive potentials (LPP, which generally begin around 400-500 ms post-stimulus and lasting for a few hundred milliseconds) to pictures with an unpleasant valence compared to a control group, indicating enhanced elaborative processing for negative emotions in BPD subjects. Further, specific deficits in P3a, a frontal lobe specific component, (such as a decreased age-related decline in P3a amplitude [487] and a right-sided focus of P3a at fronto-cental sites [488]) in BPD subjects has suggested possible failure of habituation of P3a, deficient inhibitory activity, and impeded maturation of the fronto-medial processing systems in BPD patients. This abnormality of brain maturation was further confirmed by another ERP study which reported that adolescent girls with borderline features failed to show normal age-related reductions in P3 amplitude [489]. Evoked potential studies have reported prolonged latencies in early and late components [490,491] and increased P50 sensory gating (i.e., pre-attentional habituation of responses to repeated exposure to the same sensory stimulus) [492], possibly reflecting aberrant sensory neuronal processing.

ERO Studies

There is only one ERO study of BPD, to our knowledge, which has dealt with condition-related EEG changes. Russ et al [493] reported higher absolute theta power in groups of BPD

patients compared to depressed and healthy control subjects during a laboratory pain procedure of the cold pressor test.

OTHER IMPULSIVE CONDITIONS/DISORDERS

Since electrophysiological studies on some of the impulsive disorders (e.g., pathological gambling, eating disorders, kleptomania, pyromania, trichotillomania, and paraphilia) are very few in number, they are dealt with together in this chapter rather than individually.

EEG Studies

EEG abnormalities have been reported in several other impulsive conditions, although findings are considered preliminary. For example, in eating disorders, Hatch et al [494] reported that underweight anorexic participants showed reduced relative alpha power and increased beta power in frontal brain regions of the eyes open resting EEG. In young subjects with history of fire setting (pyromania), Milrodand and Urion [495] found photoparoxysmal EEG responses to intermittent photic stimulation as well as temporal lobe abnormalities. In paraphilia, Flor-Henry et al [496] reported that pedophiles showed a pattern of increased frontal delta, theta and alpha power and a pattern of reduced interhemispheric and increased intrahemispheric-interhemispheric coherence. EEG of pathological gamblers showed a lack of switching between hemispheric EEG activity in tasks that typically involved left or right hemispheric activity [497] (cf. [498]).

ERP Studies

ERP studies have identified dysfunctional processing during cognitive tasks in several impulsive conditions. For example, patients of anorexia nervosa displayed larger P3 amplitudes and longer P3 latencies in a shape recognition task [499], while both anorexic and bulimic subjects showed increased N2 amplitudes to all face categories and decreased P3 amplitudes in response to unpleasant emotional faces [500]. As ERP responses to visual erotic stimuli, paraphilic men showed greater P600 responses to paraphilic stimuli than normal men [501]. Further, an ERP study of pathological gamblers showed reduced acoustic startle prepulse inhibition (PPI, a phenomenon in which a weaker prestimulus/prepulse inhibits the reaction of an organism to a subsequent strong startling stimulus/pulse) and prepulse-induced attenuation of the frontal P3, showing a disrupted brain functioning [502], while another study on cue reactivity showed a larger gambling stimulus-induced late positive potential in these subjects [503].

ERO Studies

To our knowledge, there are as yet no ERO studies on impulsive conditions such as pathological gambling, paraphilia, pyromania, kleptomania, etc. Possible reasons for the absence of an ERO study in these conditions could be that these disorders are often rare and do not receive as much clinical and research attention, and that the ERO techniques are relatively new, and have not yet been optimally utilized even to examine more prominent clinical conditions and disorders. It is suggested that future studies more often use ERO techniques to study several impulsive conditions.

SUMMARY AND DISCUSSION

The major electrophysiological findings with regard to impulsive conditions and disorders have been reviewed in the earlier sections. As indicated in the review, while there is a good deal of corroboration of findings, particularly for well-studied impulsive spectrum disorders, there are often contradictory findings in the literature; therefore it would be beneficial to enumerate and clarify possible reasons for these discrepant or negative findings. Further, it is also essential to indicate possible limitations of these studies so that future research might benefit from pitfalls in research design and methodology.

EEG FINDINGS

EEG studies, especially the qEEG studies using computer-aided signal analysis methods, have teased apart the spectral power variations across impulsive conditions and disorders. A relatively robust and consistent finding is that the majority of impulsive conditions/disorders show increased EEG power in delta, theta and beta bands. Increased delta activity has been observed in individuals with psychopathy and criminal/violent offense [134,179], BPD [480] (sleep EEG), ADHD [199], methamphetamine dependence [359], MDMA use [384], and paraphilia [496]. Increased theta power has been found in several conditions such as violent offense [179], ADHD [198,199], alcohol dependence [267-269], methamphetamine dependence [359], and paraphilia [496]. These findings suggest that many of the impulsive states are manifested as excessive low frequency activity of the ongoing EEG rhythms. However, it is important to note that there are a few other conditions that showed opposite findings. That is, individuals with either impulsive aggression or intermittent explosive disorder and those with nicotine or cocaine dependence have showed decreased low frequency activity. Decreased delta power was observed in individuals with impulsive aggression [149], cocaine dependence [360,362-364], and nicotine use [367-370], while decreased theta power was reported in subjects with impulsive aggression [149], intermittent explosive disorder [150], and nicotine use [367-370]. Historically, decreases in the delta and theta frequency bands have been associated with excessive cortical activation (cf. [149]). For example, decreases in delta activity in response to nicotine administration have been interpreted as evidence of greater cortical activity and alertness [504,505], and suppressed theta has been repeatedly associated with hypervigilance and attention [506-508]. Studies

have also demonstrated that individuals scoring high on self-report measures of impulsivity show greater slow wave activity [509-511].

As the majority of the impulsive conditions are associated with increased slow wave activity, it is possible that these disorders share common features such as 'low cortical arousal' as well as neurobiological and genetic underpinnings. Since evidence of low cortical arousal in impulsive individuals has also been reported [512], it is possible that in such individuals and conditions, the state of 'low cortical arousal' is compensated by seeking several 'externalizing' activities such as substance use, aggression, violent sports and other impulsive and 'sensation-seeking' activities. This explanation is similar to the 'self-medication hypothesis' of substance use [513,514], which states that individuals use alcohol and other drugs to alleviate or cope with negative affect such as anxiety and depression [515]. This explanation may also apply to conditions/disorders that exhibit decreased low frequency activity. Another viable explanation is that conditions/disorders with increased low frequency activity—psychopathy and criminal offense, BPD, ADHD, alcohol dependence, methamphetamine dependence, MDMA use, and paraphilia—are possibly more severe in their psychopathology and symptomatology, while other conditions with decreased low frequency activities, viz., impulsive aggression, intermittent explosive disorder, cocaine dependence and nicotine use, may have relatively less severe psychopathology.

In terms of high frequency EEG activity, a major and consistent finding is that for most of the disorders, with an obvious exception of ADHD, individuals exhibited increased beta activity. These disorders include intermittent explosive disorder [150], alcohol dependence [268,274-277], cocaine dependence [361,363,365], nicotine use [367-370], heroin addiction [392], and anorexia [494]. Beta frequency band is considered to be an index of cortical arousal, and increased beta power may indicate hyperexcitability in the central nervous system [277]. According to Whittington et al [516], beta rhythm involves a balance in networks of excitatory pyramidal cells and inhibitory interneurons involving $GABA_A$ action as the pacemaker. Activation of $GABA_A$ receptors are most important for maintaining inhibitory tone in the mammalian brain, and activation of these receptors causes a depolarizing Cl^- efflux that can be either excitatory or inhibitory (cf. [517]). Further, beta rhythm has been shown to be associated with a $GABA_A$ receptor gene [282]. The GABRA2 gene, which encodes the protein gamma-aminobutyric acid receptor subunit alpha-2, has been found to be associated with adult alcohol dependence [283,518-521], illicit drug dependence [522,523], childhood conduct disorder [523], and adult antisocial behavior [524]. Further, GABRA2 was associated with self-reported externalizing behaviors in adolescents and young adults [525]. Therefore, it is reasonable to conclude that impulsive conditions/disorders with increased beta activity could underlie an imbalance of inhibition-excitation in the central nervous system, and may also share a common neurobiology and genetic predisposition. On the other hand, while many studies have reported decreased beta activity in ADHD, this phenomenon is not ubiquitous but could be a part of ADHD clusters. For example, Clarke et al [526] explored EEG defined subtypes by using cluster analysis in a large sample (n=184) of boys with ADHD, and identified 3 distinct EEG-defined subtypes. The first cluster indicated cortical hypoarousal with increased total power, relative theta and theta/beta ratio, and decreased relative delta and beta across all regions. The second cluster reflected maturational lag in CNS development, characterized by increased slow wave and deficiencies of fast wave activity. The third cluster had excessive beta activity, and was labeled an "over-aroused" group.

In addition to the prominent findings of increased power in delta, theta, and beta bands, distinct disorder-specific findings have been reported. For instance, indices of EEG asymmetry have been associated with many emotional states, including anger and aggression (see Harmon-Jones [122] for a review). Other disorder-specific findings include 'alpha hyperfrontality' in chronic marijuana users [375-377], 'accelerated alpha activity' in LSD users [386], and 'abnormal beta synchrony' in heroin addicts [396]. While these specific findings might offer valuable clues to understanding pathophysiological processes, such phenomena may not have been studied in other impulsive conditions/disorders, and therefore these findings may not be exclusive to a particular disorder or category.

ERP FINDINGS

The most robust and consistent ERP finding across a wide variety of impulsive conditions/disorders is the reduction of P3 amplitude measured in different paradigms in such disorders. As reviewed in earlier sections, reduced P3 amplitude has been reported in almost all the impulsive conditions/disorders, including high impulsivity [98,104,109,111,184], intermittent explosive behavior [142], aggression [98], criminality [143,183], violent offense [144,185], psychopathy [151,193,194], antisocial spectrum [151,181-183], conduct disorder [186-188], ADHD [52-54,202,221,226,228,230,235-238,244-250], borderline personality disorder [481-483,485], personal and/or parental history of alcoholism [260,261,287-307], personal and/or parental history of drug addiction [402-407], cocaine dependence [411,412], nicotine dependence [415,417], acute cannabis exposure [425-427], chronic cannabis use [430,431], MDMA use [440], opioid dependence [405], and heroin dependence [447]. Further delayed P3 latency was also observed in ADHD subjects [221,239,246,251,252], violent prisoners [144], BPD [481,482], methamphetamine dependent individuals [420,421], chronic cannabis users [430,432], MDMA abusers [443], and anorexics [499]. The explanations for P3 amplitude reduction are numerous depending either on the task paradigms and conditions or on the specific theory by which representation of P3 is explicated. For example, NoGo-P3 may indicate inhibition, while P3 in error-related paradigm may indicate the conscious detection of errors. Further, various theories have been proposed regarding the specific cognitive process(es) that the P3 is thought to reflect. According to Sokolov's 'working memory model' [527], a representation of the stimulus is initially stored in the brain's online memory buffer, and P3 involves detection in stimulus change in comparison to the existing mental "schema". According to the 'context updating model' [46], recognition of stimulus change involves the attentional allocation and subsequent ''update'' of the memory representation for the stimulus that elicits the P3. On the other hand, the 'context closure model' [48,49] suggests that P3 represents the completion or closure of the stimulus processing context. Polich [528] proposed a 'processing capacity model', which assumes that the amplitude of the P3 is considered an index of the allocation of neural resources and cognitive processing capability, with increasing size reflecting enhanced processing capability. As mentioned earlier, other interpretations of P3 include event categorization [50], capacity allocation [51], attentional allocation [52], short-term memory updating [53], and memory-related processing [54]. In addition, the latency of the P3 is thought to reflect stimulus evaluation time and classification speed, with shorter latencies indicating superior

cognitive performance in allocating attentional resources for cognitive processing [529-533]. In essence therefore, reduced P3 amplitudes and longer P3 latencies broadly reflect relatively poorer neurocognitive processing.

It is also important to mention other ERP findings, although they are not as robust and consistent across studies. For example, decreased N1 amplitude was observed in ADHD [54,227,228], chronic cannabis users [433], and chronic MDMA users [443], while reduced N2 amplitude has been reported in ADHD [52,54,225,231-233], impulsive-violent offenders [140,141], psychopaths [195], alcoholics [319-321], and smokers [416]. As mentioned in the earlier section, the N1 component may represent information regarding sensory registration and analysis of the stimulus [35], or the activation associated with information processing at or from the primary cortex [36-38]. On the other hand, the N2 component is thought to represent processes related to stimulus discrimination and endogenous mismatch detection [41,42]. Therefore, reduced amplitudes in N1 and N2 components in these conditions/disorders may indicate dysfunctional stimulus processing and related neurocognitive deficits. In addition to these deficits, some of the disorders do manifest specific ERP findings. Individuals with borderline personality disorder failed to show normal age-related reductions in P3 amplitude [488,489], suggesting abnormal brain maturation. Cocaine users showed no P3 differentiation between monetary reward versus nonreward conditions, and this deficit was associated with frequency of recent cocaine use [410], suggesting a compromised ability to advantageously modify behavior in response to environmental contingencies. Further, heroin-dependent subjects rated pleasant pictures as less arousing and showed decreased startle-elicited P300 attenuation while viewing pleasant pictures, and this P3 attenuation further predicted future heroin use [451], demonstrating a reduced brain responsiveness to natural reinforcers. Lastly, paraphilic men showed heightened P600 responses to paraphilic stimuli than normal men, showing that an innate preponderance to erotic arousal in such individuals [501]. These findings confirm the view that impulsivity spectrum disorders do have both common neurobiological abnormalities as well as disorder specific neurocognitive impairments as seen above.

ERO FINDINGS

Event-related oscillations represent a basic mechanism of neural communication, providing links to associative and integrative brain functions [534], and are elicited during stimulus/cognitive processing and measured in several parameters viz., time, frequency, amplitude/power, phase and coherence. Although ERO studies are more limited in number, as this is a recently developed method, several of the impulsivity spectrum disorders have shown abnormalities in several ERO bands. Major ERO findings include decreased activity in delta, theta, and gamma bands, while a few studies have also reported increased theta activity in some of the impulsive spectrum disorders. Specifically, decreased delta activity was seen in externalizing proneness [117], ADHD [257], and alcohol dependence [329-332]. Decreased ERO theta activity has been reported in individuals with high impulsivity [115], alcoholics and their high-risk offspring [329-332,342], and during marijuana ingestion [425,426]. At the high frequency level, decreased ERO gamma was observed among alcoholics [343], offspring of alcoholics [344], and cannabis users [460], as well as during the administration of

amphetamine in rats [459] and in response to cocaine-related drug cues in humans [456], while increased evoked gamma band responses were observed in ADHD patients [258,259]. In contrast, increased theta ERO activity has also been reported in BPD patients during a cold pressor test [493] and in individuals with ADHD during the prestimulus time interval [234]. While these abnormalities in ERO frequency bands may reflect gross dysfunction in neurocognitive processing, specific frequency bands of ERO activity have been associated with various cognitive functions [63,67-70]. For example, delta responses are believed to mediate signal detection and decision making (e.g., [63,535,536], while theta rhythms are linked with different cognitive processes, such as conscious awareness, recognition memory, episodic retrieval, and frontal inhibitory control [67,69,329,337,535,537]. The slow alpha rhythm (8–10 Hz) has been assumed to modulate as a function of attentional demands [538-540], and fast alpha activity (10–12 Hz) has been shown to mediate semantic memory processes as well as stimulus-related aspects [67,539,541,542]. Further, oscillatory gamma responses are involved in visual perception, cognitive integrative function such as "binding," and frontal input during sensory processing (top-down processing) [68,535,543-545]. It should be noted that these oscillations mediate neural communication. Higher frequency oscillations are involved in more localized neural networks, whereas the slower oscillations govern the long-range communication networks (e.g., posterior to anterior sites) [65,78,546-548]. Given the deficient ERO activity in delta, theta and gamma bands reported in this review, it is possible that impulsivity spectrum disorders may share many of these neurocognitive deficits ranging from signal detection, memory processes and decision making to visual perception, inhibitory control and cognitive integrative functions.

Furthermore, it has been proposed that common features across the externalizing disorders can be largely attributed to common genetic factors [19,549]. Electrophysiological endophenotypes, especially brain oscillations, are closely linked to genetic underpinnings. According to Begleiter and Porjesz [550], brain oscillations represent highly heritable traits that are less complex and more proximal to gene function than either diagnostic labels or traditional cognitive measures. For example, Jones et al [551] reported that frontal ERO theta band during visual oddball task was linked with a single nucleotide polymorphism (SNP) from the cholinergic muscarinic receptor gene (CHRM2) on chromosome 7. Specific genes have been identified in several externalizing spectrum disorders, including alcohol dependence and related disorders [283,552-557], other substance use disorders [522,558,559], childhood conduct disorder [523], adult antisocial behavior [524], and other behavioral disorders [560-563]. It is expected that as complex relationships among genes, neurophysiological processes and the various disorders are elucidated, it will lead to a better understanding of the common and specific underlying liabilities that determine the outcome of impulsivity spectrum disorders.

CRITIQUE AND CAVEATS

It is clear from the above findings that electrophysiological methods are very useful in elucidating the common and specific deficits associated with impulsive spectrum disorders. As noted earlier, the prominent electrophysiological findings among individuals with high impulsivity and impulsive conditions/disorders were differentiated frontal asymmetry,

increased resting beta power, decreased P3 amplitude, and decreased ERO delta, theta and gamma power. Notwithstanding these highlighted findings, it is also crucial to note that quite a few contradictory as well as equivocal findings have also been reported. For example, in resting EEG, while many of the impulsive spectrum disorders had increased delta activity [134,179,199,359,384,480,496], a few conditions showed decreased delta activity [149,360,362-364,367-370]. Interestingly, it was striking when this contradiction was within a single category. For example, among stimulants, methamphetamine dependence caused increased delta [359], while a decrease in delta activity was observed in cocaine dependence [360,362-364] and nicotine use [367-370]. Further, a highly equivocal finding in resting EEG was the role of alpha activity that was found to be increased in many conditions (cocaine dependence, nicotine use, chronic marijuana use, and paraphilia) [360-362,367-370,375-377,496] as well as decreased in several conditions (psychopathy, criminal / violent offense, nicotine use, and heroin addiction) [134,179,367-370,392,494]. Similar equivocal findings were also seen in ERP as well as ERO studies. For example, several conditions (ADHD, impulsive-violent offense, psychopathy, and alcoholism, and nicotine use) [52,54,140,141,195,225,231-233,319-321,416] were marked with decreased N2 amplitudes in several paradigms, while a few conditions (ADHD, psychopathy, alcoholism, anorexia and bulimia) [193,243,244,323-325,500] showed increased N2 amplitudes. Among ERO findings, an obvious but contrary finding was in theta band, which was found to be decreased in a few conditions (high impulsivity, alcoholism, high-risk status with parental/family history of alcoholism, and acute marijuana ingestion) [115,147,329-332,342,425,426] and increased in a few of the disorders (borderline personality disorder and ADHD) [234,493]. Further, while decreased ERO gamma was observed in individuals with certain conditions, such as alcoholics [343], offspring of alcoholics [344], and cannabis users [460], ADHD patients, in contrast, showed increased evoked gamma band responses [258,259].

On the one hand, variations in electrophysiological findings could be specific not only to a disorder but also to testing and/or task conditions and methodologies. There may be limitations in the study design, methodology and scope as well. However, interpretation of these seemingly contradictory findings may vary depending on the research context and methodology. As a typical example for a 'state-dependent' finding, Kouri et al [448] reported that chronic heroin- and cocaine-dependent individuals demonstrated normal P3 amplitudes under the influence of heroin or heroin and cocaine, while they manifested reduced P3 amplitudes during detoxification. An explanation for these findings might be that under the influence of a drug, i.e., during a 'self-medicated' state, the brain is no longer hypo- or hyper-aroused, but during the 'non-drug' state, it becomes a disinhibited, hyper-aroused cortical state, as manifested by reduced P3 amplitudes. Similar findings have been observed in high-risk offspring of alcoholics who manifested low P3 amplitudes that increased or "normalized" with the ingestion of alcohol [564]. Alcohol may actually be acting to "normalize" P3 amplitude in some individuals with low P3 amplitudes at baseline (cf. [565]). Similarly, in another P3 study, skydivers were shown to have more sensation seeking and larger frontal P3 amplitudes than controls [97], and this finding of augmented P3 was explained as a mechanism for counterbalancing the emotional deficit already present in the skydivers. In yet another example, while the delayed ERP latency is generally considered to reflect a deficit, Sunohara et al [252] found faster P2 latency in ADHD and explained that this reflected a rapid and atypical stimulus-feature detection, reflecting impulsivity. The major reasons for discrepancies in findings are differences in the study sample and methodologies across

studies. Sampling variables such as ascertainment of subjects and groups, instruments for diagnosis or classification, and sample characteristics such as age, gender, onset, duration and severity of the disorder determine the quality of the study and affect the results and findings. Further, differences in electrophysiological methodologies may further contribute to the discrepancy in findings across studies. ERP components may be interpreted differently depending on the type of paradigms, cognitive processes involved in the task, and stimuli used to elicit the components. For example, a P3 component is different across paradigms and modality, i.e., the P3 from an oddball paradigm will be different across its conditions (e.g., rare target, rare non-target), modality (auditory, visual, and somatosensory), and other paradigms (Go-NoGo, error paradigm, gambling paradigm, etc.). These issues are not limitations in the methodology, but differences in study designs are tapping different aspects of brain function. Hence, while results look discrepant, they may not be tapping the same brain processes. Furthermore, it should be noted that although all the 'impulsivity spectrum disorders' have been treated as a unitary spectrum based on impulsivity, electrophysiological findings reviewed here may not be directly related to the psychometric or behavioral measures of impulsivity as such, but rather may be related to the nature and symptomatology of a disorder wherein impulsivity is considered to be a core feature. In this context, several of the reported studies in this chapter deal with the disorder per se, rather than with the psychometrically measured impulsivity. Further, these disorders vary in terms of the nature of psychopathology, pathophysiology, manifestations and outcome, as do the components or factors of impulsivity as a multi-dimensional construct. In this regard, antisocial spectrum disorders may be vastly different from substance use disorders or borderline personality disorder or eating disorders, and different disorders may have different effects on the brain. For example, increased beta power of the resting EEG was observed in most of the impulsivity spectrum disorders [150,268,274-277,361,363,365,367-370,392,494]; yet children with ADHD have shown decreased beta power [198]. Further, various dimensions of impulsivity may be related to different electrophysiological components and tasks. For example, Harmon-Jones et al [98] reported that non-planning and attentional impulsivity were related negatively to the parietal P3 amplitude of the oddball task and continuous performance task (CPT), while motor impulsivity was related positively to parietal P3 in the oddball task.

Given the above mentioned critique and caveats, future studies may aim at bridging the gap in the existing research trend, methods and findings. An obvious limitation in this regard is the small number of studies that investigate the same diagnosis with the same electrophysiological procedures. Hence, due to the small number of studies in a particular disorder with the same electrophysiological measures, there is a lack of replication studies for many of these disorders (excepting alcoholism, psychopathy and ASPD). A suggested first step would be to perform meta-analyses across studies that use the same procedure to establish the findings that are robust. For example, a low P3 amplitude has been established as a marker of risk in offspring of alcoholics, and this meta-analysis also determined that this finding was strongest for visual paradigms in younger males [300]. Since that time, these findings have been replicated by numerous studies throughout the world. Similarly, in a meta-analysis on P3 impairments in antisocial and psychopathic individuals by Gao and Raine [151], it was established that compared to non-psychopathic offenders, psychopathic offenders showed P3 amplitude impairments in standard oddball tasks, but not in other tasks. However, other disorders in the impulsivity spectrum disorder lack such meta-analyses that

are vital to characterize the markers or endophenotypes for the disorder due to the paucity of studies. Therefore, more electrophysiological studies need to be done in those disorders where there is a dearth of studies in order to determine general/specific aberrations that are markers for specific disorder(s). Once key findings (i.e., markers) are established with a substantial number of studies, they need to be confirmed by replication using identical methodologies. Moreover, in order to further characterize the genetic risk for developing a disorder, studies have to explore whether the specfic markers present in the affected individual is also present in their offspring prior to the onset of the disorder and in other first-degree and/or second-degree relatives. Such approaches will pave the way for the understanding of endophenotypes for the entire rubric of impulsivity spectrum disorders.

CONCLUSIONS: ELECTROPHYSIOLOGY, IMPULSIVITY, AND FRONTAL LOBES

The terminology 'impulsivity spectrum disorders' has been employed to connote a wide variety of disorders, such as antisocial personality disorder, conduct disorder, borderline personality disorder, substance dependence, paraphilias, adjustment disorder, and eating disorders, along with other conditions such as aggression/violence, intermittent explosive disorder, kleptomania, pyromania, pathological gambling, and trichotillomania. However, this classification closely resembles the existing terminologies such as disinhibitory disorders [17] and externalizing disorders [18,19,566]. According to Gorenstein and Newman [17], the general rubric of disinhibitory psychopathology refers to a range of conditions marked by a failure of self-control, such as hyperactivity in children, antisocial behavior in adolescents, and psychopathy and primary alcoholism in adulthood. The unifying themes across these phenotypically related yet distinct conditions include deficits in inhibition and excesses in rule-breaking or norm-violating behavior (cf. [515]). Similarly, in Krueger's model of externalizing spectrum disorders [18,566], externalizing phenomena are organized hierarchically [566] and poor delay of gratification may be a specific risk factor [18]. These externalizing disorders may share a common structure of genetic etiology. Kendler et al [19], in a large empirical study, identified 2 genetic factors. The first had its strongest loadings on externalizing disorders such as alcohol dependence, drug abuse/dependence, adult antisocial behavior, and conduct disorder; the second, on internalizing disorders such as major depression, generalized anxiety disorder, and phobia. While each of the externalizing disorders were found to have specific factors involved, the single most important factor for all these externalizing disorders was an underlying common factor comprising disinhibitory or impulsive traits [19].

There is a great deal of evidence indicating that heightened impulsivity and disinhibitory disorders involves frontal lobe pathology as a major underlying neurobiological substrate that could explain all the pathological manifestations of all these disorders [468,567-573]. In addiction disorders, Goldstein and Volkow [574] conceptualized alcohol/drug addiction as a syndrome of impaired response inhibition and salience attribution, and summarized the involvement of the frontosubcortical circuits in addiction disorders. Concepts of impulsivity, disinhibition, and risk propensity underlie the vulnerability, not only for addiction disorders, but for the entire rubric of disinhibitory or externalizing psychopathology [356,575]. It was

also proposed that the primary motivation circuitry involving cortical–striatal–thalamic-cortical loops were putatively involved in impulsivity, decision making and the disorders of alcohol/drug addiction and pathological gambling [576,577]. Hall et al. [578] connected the dimensions of electrophysiology, impulsivity, and markers of psychopathology, by suggesting that neurobiologically based deficits (such as dysfunctional brain oscillatory activity) in endogenous action monitoring systems may underlie a generalized risk for an array of impulse-control problems and disinhibition. Further, at the neurophysiological level, the P3 which may serve as a robust neurophysiological marker for the impulsivity spectrum disorders; it is assumed to be the outcome of an interaction between the frontal cortex and the hippocampal system for memory updating, and the locus coeruleus-norepinephrine (LC-NE) system (cf. [151]), which contributes to the initiation and maintenance of behavioral and forebrain neuronal activity states and modulates the collection and processing of salient sensory information through cortical and subcortical sensory, attentional, and memory circuits [579]. Brain imaging studies have observed abnormalities in frontal regions in several impulsive disorders including antisocial personality (e.g., [580,581], alcoholism [582], ADHD [583-585], borderline personality disorder [572,580,586,587], etc. Electrophysiological findings in these disorders can also highlight problems in neural communications between/among brain regions. Consequently, specific electrophysiological findings such as P3 abnormalities may reflect abnormalities in brain structures and/or functions that are linked to impulsive spectrum disorder through possible causative factors such as behavioral disinhibition, poorer regulation of aggression, lack of fear and moral decision-making [588]. Newer studies are also increasingly using localization techniques, such as LORETA, in order to delineate specific activity patterns and communications across brain regions in many of the impulsive spectrum disorders such as alcoholism [261,263,309,321,589], ASPD [590], and ADHD [219], and brain region/structure related electrophysiological phenotypes have also been used to elicit genotype differences in these disorders (e.g., Williams et al. [590]). Finally, it may be concluded that electrophysiological methods have effectively identified distinct biological markers in several impulsivity spectrum disorders (such as alcoholism, ADHD, and ASPD), while this elucidation was not possible in some of the other disorders mainly due to the paucity of studies. It is suggested that future studies can test and confirm the possibility that impulsivity, electrophysiological phenotypes, frontal lobe development/functioning may share common as well as specific genetic mechanisms and vulnerability in producing the range of disorders that are hierarchically structured as a common spectrum, as well as a specific entity with respect to several behavioral, clinical and neurobiological manifestations and outcomes.

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