Making the Case for 'Power Abuse Disorder' as a Nosologic Entity

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Abstract

The development of societies and cultures arguably is based on the ability of human primates to form hierarchies in which some individuals acquire and wield power, that is, control resources and influence and control the behavior of their conspecifics. In the following, we focus on the type of human primate power wielding that (a) harms and (b) produces excessive negative emotions in (1) the victim(s) of the power wielder and (2) the power wielder her/himself. If such a harmful behavior of the power wielder is not accompanied by an ethically justifiable benefit for the involved human primate groups, it can be considered "power abuse." We propose to term the associated behaviors, cognitions, and emotions of the power wielder as "power abuse disorder" (PAD). This behavior results from what we consider addictive behavior of the power abuse disordered (PADed) power wielder. PAD can be diagnosed on the basis of the World Health Organization's criteria for "dependence syndrome" as listed in the International Classification of Diseases version 10. We will demonstrate that many PADed individuals may very likely carry the Zeitgeist diagnosis "burnout." This article reviews the current understanding of the neural correlates of PAD and suggests future research. Based on the available evidence, PAD seems to be associated with a dysfunction of the mesocorticolimbic dopamine system, rendering PADed individuals vulnerable for psychostimulant abuse/dependence, and suggesting specific pharmacotherapeutic approaches to treat PAD.

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Introduction

The development of societies and cultural achievements is arguably based on the ability of humans (i.e., human primates) to form hierarchies, in which some individuals acquire and wield power, that is, control limited resources and influence and/or control the behavior of their conspecifics. An animal experimental operationalization of social dominance with very high face validity for our (i.e., human primate) minds is winning a fight [1–10]. Maybe of even higher impact for the evolutionary selection process, dominance can be seen and has been operationalized as competitive control over access to vital resources such as water [11], food [12], space for advancement in its literal sense [11], or access to avoidance of unpleasant/harmful stimuli [12], which are

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all helpful for the survival of the individual, and for its control over reproduction within a group [7, 13, 14], which in turn is beneficial for the spread of such an individual's genes.

But are the neurobiologic mechanisms underlying the fight of two mice over a scrap of leftover cheese comparable to the travails of 21st-century Woman striving toward self-realization in a professional career? We will show in the following that they are. For one, we human primates are able to covet objects at such a degree of abstraction that the visual presentation of status symbols like luxury cars [15] or even the expectation of a monetary reward [16], with money arguably being one of the most salient status symbols, is able to activate the same neurocircuits (i.e., the so-called reward pathways) that are activated by "natural" or "physiologic" reinforcers (such as food, water, or sex) in rodents or nonhuman primates, in particular a brain region that is central to motivated behavior, that is, the nucleus acumbens (called "ventral striatum" in human imaging studies; see, e.g., [10, 17–23]). To paraphrase, the same neuronetworks that mediate motivated behavior for "natural" or "physiologic" rewards (e.g., food, water, sex) are also involved in drug seeking and consumption, that is, measures of substance dependence, in all species mentioned above. Not only is the seeking and consumption of physiologic reinforcers and drugs of abuse mediated by the same neuronetworks: the non-drug addictive disorder "pathological gambling" (code F63.0) of the International Classification of Diseases version 10 (ICD10) [24], which is essentially identical to the "gambling disorder" (code 312.31) of the Diagnostic and Statistical Manual 5th edition (DSMV, DSM5) of the American Psychiatric Association [25], is considered to involve essentially the same neurocircuits as the seeking and consumption of drugs of abuse, having prompted clinical trials in which the same medications that had been evaluated for the treatment of substance dependence syndromes/substance-use disorders were tested for their effectiveness against gambling disorder [26]. By the same token, we think that the nondrug addictive disorder proposed here, power abuse disorder (PAD), shares the same neurobiologic basis as well. Another nonpharmacologic stimulus that may engender addictive behavior, internet gaming, is already on its way to be incorporated into mental disorder compendia, as evidenced by its inclusion as a "condition for further study" in the DSM5 [25]. To emphasize, *any* motivated behavior, including food seeking and consumption [27], carries the risk of becoming addictive, with the nucleus acumbens (see, e.g., [10, 17–23]) and,

possibly, the whole accumbens corridor [10, 28, 29] as the central neuroanatomic hot spot of the motivational neuronetwork (reward pathways) and dopamine as the major neurotransmitter driving motivated behavior. Interestingly, food and power share the paradox of being both essential for our survival as well as becoming extremely harmful for us if abused.

We all have experienced that losing control over a resource to another conspecific is associated with negative emotions, an experience we share with other species, for example, rodents. However, even in the absence of competition for resources, social dominance (an alternative term for "power wielding") significantly decreases the attractiveness of dyadic social interaction for the subordinate animal, even if the likely hierarchic difference is modest, that is, only consists of a maximum of 2-fold weight difference during 4 consecutive 15-min dyadic social encounters between otherwise singly housed male rats [30]. There is a plethora of animal studies demonstrating that social dominance, at least in despotic species/genus like nonhuman primates, rats, or mice [31], can actually be harmful for the subordinate individual [13, 14, 31]. Even in humans, the constant proximity to an anxiety-provoking dominant member of one's own species, with dominance being maintained in despotic species through repeated intimidation rather than full-blown aggression, results in dendritic atrophy, impaired neurogenesis and synaptic plasticity, enhanced GABAA receptor antagonist-inducible anxiety ("enhanced endogenous benzodiazepine tone" [32]), elevated basal levels of glucocorticoids, sluggish response to and recovery from stress, basal immunosuppression and decreased immune responsiveness to challenge, basal hypertension, a pathologic cholesterol profile, testicular atrophy, decreased gonadal hormones, and increased risk of anovulation and miscarriage [31].

In the following, we focus on the type of human primate power wielding that (a) harms and (b) produces excessive negative emotions in (1) the subordinate(s) of a socially dominant individual and (2) the power wielder her/himself. By "excessive negative emotions" we mean negative emotions of an intensity that exceed what an average empathic observer would expect under the circumstances. If such a harmful behavior of the power wielder is not at least accompanied by an ethically justifiable benefit (see our suggestions for appropriate diagnostic settings below) for the involved human primate groups, it can be considered "power abuse." We propose to term the associated behaviors, cognitions, and emotions of the power wielder PAD. This behavior results from what we

consider addictive behavior of the power abuse disordered (PADed) power wielder. PAD can thus be diagnosed according to the World Health Organization's criteria for "dependence syndrome" as listed in the ICD10 [24]. A detailed description of the ICD10-based PAD symptoms is given below.

Mirroring the behavioral similarity of PAD and other abuse/dependence syndromes, PAD, a non-substance addictive disorder, should have a considerable number of neuroanatomic and neurobiologic commonalities with drug abuse and drug dependence (substance abuse disorders). This article discusses data on power wielding–induced changes in the dopamine system because we think that the dopamine neuronetwork is heavily affected by both substance–dependence syndromes (especially by psychostimulant use disorders) and PAD. A discussion of other involved neurotransmitter systems, unfortunately, is beyond the scope of the present article. The translational power of possible animal models of PAD will be discussed. Suggestions for future research will be given.

It should also be borne in mind that this article focuses on the less severe forms of PAD, which are well known to essentially all of us and have caused all of us discomfort and, possibly, harm. It is beyond the scope of the present article to discuss severe forms of PAD, for example, torture or the actions of power wielders in totalitarian political systems.

The Clinical Presentation of PAD: Suggested Diagnostic Criteria

At the level of human primate behavior, PAD fulfils all classic diagnostic criteria of a dependence syndrome according to the ICD10 issued by the World Health Organization (www.who.org).

The ICD10's diagnostic guidelines (http://www.who.int/substance_abuse/terminology/definition1/en/, accessed March 29, 2017; symptom numbering by the authors) are given below in the left column of the Box 1, while the corresponding symptoms of PAD as suggested in the present article can be found in the right column.

Additional Behavioral Symptoms of PAD

PAD is characterized by behavioral symptoms that have not been covered by the ICD10 criteria for dependence syndrome, symptoms that can be helpful for a better diagnosis of PAD, and should be the focus of future

clinical research because they present targets for developing coping strategies for the victims of PAD superiors (PADS) as well as therapeutic interventions for the PADS themselves. The following behavioral symptoms of PADed power wielders are just a tiny (albeit highly relevant) sample of the symptoms that have anecdotally been observed by us:

a. A characteristic pattern of communication consisting of indirect, vague, noncommittal and/or explicitly threatening verbal and nonverbal behavior leading to fears, hopes, and confusion in the subordinate, ultimately harming the victim and sabotaging the victim's productivity.

b. Resistance by the PADed power wielder to any form of objectifiable documentation of her/his interaction with her/his victim, for example, video or audio recordings.

c. Resistance by the PADed power wielder to any form of communication that may reduce her/his perceived power, for example, team supervision. This resistance may also be in the form of apparent acceptance of the subordinate's suggestions and/or demands followed by sabotaging them.

Diagnosing PAD

So how can we arrive at a diagnosis of PAD? In everyday clinical practice, the diagnosis could be made by a trained and hopefully intervised and/or supervised professional using the diagnostic criteria detailed above. For research purposes, we suggest that a case of research interest in an anonymized form be submitted to a panel of at least 10 persons who explicitly must NOT belong to the same societal/institutional/professional system in which a putative PAD incident has occurred. Such a panel must be composed, in equal parts, of women and men and, preferably, have members from all walks of life. For a definite diagnosis of PAD, 8 of 10 panelists (i.e., 80%) or more must come to such a conclusion. For a definite diagnosis of the absence of PAD, 8 of 10 panelists must vote accordingly. We base the "8 of 10 votes" requirement on Fisher's exact test (Prism 7, www.graphpad.com), which states that an 8-to-2 vote vs. a 2-to-8 vote for a PAD diagnosis is statistically significant ($p = 0.023$, 2-sided). A 4-to-1 vs. 1–4 vote, that is, a smaller panel, would not have the necessary statistical power ($p = 0.21$, 2-sided). Our proposal is, of course, arbitrary and debatable, and should be tailored to the statistical needs of the respective scientific investigation.

Box 1.

ICD10 dependence syndrome	Power abuse disorder (PAD)
A definite diagnosis of dependence should usually be made only if three or more of the following have been present together at some time during the previous year: 1. A strong desire or sense of compulsion to take the substance	A definite diagnosis of PAD should usually be made only if three or more of the following have been present together at some time during the previous year: 1. A strong desire or sense of compulsion achieve and hold a position within a hierarchy that allows the holder to influence and control the behavior of subordinate(s), which is harmful for and associated with negative emotions of the subordinate(s)
2. Difficulties in controlling substance-taking behavior in terms of its onset, termination, or levels of use	2. Difficulties in controlling power-wielding behavior in terms of its onset, termination, or levels of wielding
3. A physiological withdrawal state when substance use has ceased or have been reduced, as evidenced by: the characteristic withdrawal syndrome for the substance; or use of the same (or closely related) substance with the intention of relieving or avoiding withdrawal symptoms	3. A physiological withdrawal state when power has ceased or has been reduced, as evidenced by: the characteristic power-withdrawal syndrome; or use of the other forms of power with the intention of relieving or avoiding withdrawal symptoms
4. Evidence of tolerance, such that increased doses of the psychoactive substance are required in order to achieve effects originally produced by lower doses (clear examples of this are found in alcohol- and opiate-dependent individuals who may take daily doses sufficient to incapacitate or kill nontolerant users)	4. Evidence of tolerance, such that increased levels of power are required in order to achieve effects originally produced by lower levels
5. Progressive neglect of alternative pleasures or interests because of psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects	5. Progressive neglect of alternative pleasures or interests because of power use, increased amount of time necessary to obtain or wield power or to recover from its effects
6. Persisting with substance use despite clear evidence of overtly harmful consequences, such as harm to the liver through excessive drinking, depressive mood states consequent to periods of heavy substance use, or drug-related impairment of cognitive functioning; efforts should be made to determine that the user was actually, or could be expected to be, aware of the nature and extent of the harm	6. Persisting with power wielding despite clear evidence of overtly harmful consequences; efforts should be made to determine that the user was actually, or could be expected to be, aware of the nature and extent of the harm. Of note, the harm is not limited to the power-dependent individual but includes and is most likely more severe for the subordinate and the environment of the PADed individual

Why has PAD not been diagnosed before? Why is the clinical and basic science literature on PAD so scarce? We opine that there is considerable political resistance to subject power wielding individuals to scientific scrutiny. From the psychotherapeutic perspective, power wielders, especially PADed ones, must be considered extremely defensive about the negative symptoms of PAD because they most likely interpret these symptoms a sign of their "weakness" and believe that any mentioning of these symptoms may threaten their dominant position. We will

show below that many PADed individuals may very likely carry the Zeitgeist diagnosis "burnout" (PubMed, accessed on March 30, 2017, searching for "burnout" or "burn out" or "burn-out" and year of publication: 1990, 221 hits vs. 2016, 1,279 hits). The diagnosis "burnout" (see, e.g., [33–41]) has become largely socially sanctioned and culturally mainstream, removing some of the stigma attached to mental disorders, maybe even carrying, in some subcultures, an honorable connotation like the Japanese "karoshi" (from the English "crash," signifying "overwork death"). Interestingly, karoshi (see, e.g., [42, 43]) may also be a consequence of power struggles within a commercial institution and may, in some cases, be predominantly due to PAD.

Treating PAD

To emphasize, helping the victims of PADed individuals should be the first therapeutic priority, prevention and inhibitory interventions being the second. To effectively cope with PADed superiors, various techniques from a host of psychotherapeutic approaches (see, e.g., [44–46]) may be introduced to and trained with a PAD victim. It is to be expected that a PADed superior will not seek help during most of her/his disease progression (see above and below). Still, the third priority should be to offer her/him therapeutic assistance during abstinence/withdrawal ("Entzug" in German) and rehabilitation ("Entwoehnung"). At the societal level, raising awareness for PAD should help both victims and PADed superiors.

Labeling any individually and/or socially harmful behavior as a disorder/disease runs the risk of putting an individual emitting such a behavior beyond the reach of ethics and justice. To emphasize, PAD results in harmful behavior for which the individual emitting PAD-associated behavior should take full responsibility. We hope that PAD will evolve the same way as smoking did over the last 7 decades, from being perceived as an annoying yet unavoidable behavioral variant to a behavior that has been recognized as harmful for its victims and has therefore been socially sanctioned in many instances.

Animal Behavioral Experimental and Neural Correlates of Power Wielding

If, as discussed above, power wielding ("social dominance" or "dominance" for short) is a stimulus that has abuse liability, it should engage the same neuronetworks and neurotransmitter systems that drugs of abuse act upon. We will demonstrate in the following that it does. As we think that striving for and wielding power engages the dopamine system more than any other neurotransmitter systems of the reward neuronetworks (see below), we focus on the experimental evidence presented by 2 groups who not only studied the effects of power wielding on the dopaminergic system with the reward neuronetwork per se but also investigated the effects of power wielding on the consumption of cocaine, a psychostimulant; psychostimulants having been used since World War II by military personnel of various nations to boost flagging–motivated behavior and alertness (see, e.g., [47, 48]).

Power Wielding Increases Accumbal D2/D3 Receptor Density

Dopamine D2/D3 receptor density/availability was shown to increase between +6% and +22% in the basal ganglia, including the nucleus accumbens, of socially dominant cynomolgous monkeys (macaca fascicularis) as compared to their subordinate conspecifics, both in males [3] and females [8]. This difference between dominant and subordinate conspecifics appeared only as the hierarchy developed [3, 8] and disappeared again when the hierarchy was dissolved by single housing [8]. Dominant male Lister Hooded rats (females were not tested) showed a mean increase of +19% (range, +6 to +28%) in D2/D3 receptor density in the left and right nucleus accumbens core and shell than subordinate animals when tested after a hierarchy had developed [11].

Interestingly, upon repeated testing of male cynomolgous monkeys in the intravenous (i.v.) cocaine self-administration paradigm, the difference in D2/D3 receptor availability between dominant and subordinate conspecifics disappeared [49], suggesting that voluntary consumption of cocaine counteracts the individually beneficial effect of power wielding with respect to the sensitivity for motivationally relevant stimuli.

Behavioral Consequences of a Power Wielding–Induced Increase in Accumbal D2/D3 Receptors

What are the behavioral consequences of a power wielding–induced increase in accumbal/striatal D2/D3 receptors? The pervasive role of accumbal dopamine (see,

e.g., [10, 17–23]) for motivated behavior (as opposed to the valence of the reinforcer, and to the contribution of dopamine to the attraction/aversiveness of a stimulus) is important for our neurochemic investigation of PAD, as the life of a PADed individual seems to be controlled more frequently and more pervasively by an escalation of their motivation to obtain power and avoid the loss of power than by actually enjoying power.

As recently as 2016, that is, 4 decades after the discovery that administration of drugs of abuse increases accumbal dopamine [17, 50], subsequent generations of researchers humbly concluded [51] that "*overall, the role of dopamine in motivation is still considered to be mysterious,*" quoting the title of a 2012 review of a different group [23]. However, a crude summary seems permissible: dopamine in the accumbens is currently thought to serve at least 2 functions: (1) baseline dopamine and slow, that is, minute-to-minute, changes in accumbal dopamine (tonic DA release) seem to correlate with motivational vigor, reward rate, motivational vigor, and deprivation, whereas (2) fast, that is, second-to-second, changes in DA dopamine (phasic release) encode reward intensity/value as demonstrated by combinations of the most recent techniques [51–54].

Accumbal dopamine release [55] and accumbal activation [22] are induced by a plethora of stimuli that are considered attractive/pleasurable, including drugs of abuse like cocaine [18], which in fact, through its inhibition of dopamine reuptake, increases dopamine in the accumbens by itself and can be used to overcome a slump in motivated behavior. Cocaine is therefore a very useful experimental tool because it is both a pharmacologic probe to test the sensitivity of a power-wielding individual to attractive stimuli but also an abused drug, allowing researchers to investigate if PAD is associated with cocaine abuse/dependence (i.e., if PAD and cocaine use disorder are comorbid) or if power wielding protects against a cocaine–dependence syndrome and may even render the individual to be more appreciative of nondrug stimuli.

Some researchers (e.g., [22]) suggest that accumbal activation is exclusively associated with attractive/pleasurable/rewarding/reinforcing stimuli, whereas other researchers have pointed out that aversive stimuli and conditioning per se also lead to accumbal dopamine release and accumbal activation [56, 57]. This is important for our discussion of the neurobiologic basis of PAD because we think that the expected loss of power (be it real or imagined) is highly aversive to a PADed individual, and consequently, a major motivator for him/her through the use of the dopamine system.

To conclude our brief overview of the current hypotheses regarding the role of the dopamine system (1) in motivated behavior, that is, in the seeking of attractive stimuli or avoidance of aversive stimuli; and (2) consummation/consumption of coveted stimuli, some researchers emphasize that accumbal activation can be separated into signaling a dopamine-independent "hedonic impact" or "liking" of a stimulus and signaling its "motivational salience," that is, the "wanting" such an attractive/appetitive/positively reinforcing stimulus elicits, in a dopamine-dependent manner [27]. For an illustration of the psychologic constructs involved in explaining "reward" and "reinforcement," the reader is referred to [21]. To complicate matters, recent research indicates that stimulation of accumbal medium spiny neurons expressing predominantly dopamine D2 receptors (D2-MSNs) is aversive [58] and decreases cocaine reward [59]. Employing designer receptors exclusively activated by designer drugs, it was demonstrated that D1-MSNs are necessary for the acquisition of cocaine-conditioned place preference (CPP), with D1-MSN activity peaking immediately before the mice entered the cocaine-associated compartment, whereas D2-MSN activity decreased once the mice entered the compartment [53]. In a similar vein, accumbal dopamine was shown to decline once a food reward was obtained in an operant task [51]. Others demonstrated that accumbal D1 receptor activation was necessary only for neural excitation by relevant (reward-predicting) stimuli, whereas D2 receptor activation was necessary for both reward-predicting and neutral stimuli [52]. To summarize, the above-cited experimental also indicates that both aversive and attractive aspects of stimuli are mediated by the accumbal dopamine neuronetwork, albeit by different subsystems.

On pharmacologic principle [21, 60–64], an increase in receptor density should render an individual more sensitive to the effects of agonist ligands binding to these receptors. This phenomenon has been amply demonstrated for mu opioid receptors and several behavioral paradigms, that is, self-administration of the full mu opioid receptor agonist alfentanil and the partial mu opioid receptor agonist nalbuphine in rhesus monkeys [65] as well as mu opioid receptor agonist-mediated antinociception in rhesus monkeys [65], rats [66], and mice [67, 68]. Accordingly, an increase in D2/D3 receptor density should result in an increased sensitivity to stimuli that enhance dopamine in the accumbens (but see [69, 70] who, surprisingly, found a negative correlation between D2 receptor levels in the striatum and reports of "drug liking" for or positive and negative effects of psychostimulants).

The only robust, that is, consistent across-species finding, namely, that accumbal D2/D3 receptor density/availability increased upon power wielding was, however, not correlated with consistent effects on measures of dopamine sensitivity when using cocaine as a pharmacologic probe and i.v. cocaine self-administration [3, 11, 71] as a behavioral readout (as would be expected from pharmacologic principle, see above): dominant male cynomolgous monkeys sometimes show decreased [72] or increased [49] responding (i.e., "worked less hard or harder") for at least some of the available cocaine doses than their subordinate conspecifics, even if tested by the same laboratory that also found that dominance in female monkeys increased responding for i.v. cocaine [8]. Power wielding also increased responses to cocaine in male Lister hooded rats [11]. Of note, all these i.v. cocaine self-administration data were obtained once responding for cocaine became stable, that is, once power wielding-associated changes were reflected during the maintenance phase (as opposed to the acquisition phase of either dominance or cocaine self-administration, but see [8] for a different interpretation). Finally, response rate data obtained under the fixed-ratio schedules of reinforcement employed in the above-quoted experiments are extremely difficult to interpret due to the direct drug effect during the session (drug satiation, direct effects on motor behavior), especially if the drug under investigation is cocaine, that is, a drug of abuse that possesses aversive effects at the very same doses that are attractive for a cocaine consumer [21, 64]. For different interpretations of the quoted experimental findings, the reader is referred to the respective original publications.

Effects of Subordination on the Sensitivity to Psychostimulant Stimuli: Human and Animal Experimental Data

There are a number of studies (reviewed by [73]) demonstrating that an extreme form of subordination/submission, that is, social defeat, sensitizes the defeated animals to the locomotor and rewarding effects of psychostimulants, while reducing their self-administration. "*Social submission is a known risk trait for drug abuse in laboratory animals*" (p. 279 in [73]). The reader is also referred to the excellent work of Russo et al. [74–76] and Miczek et al. [77].

We are not aware of any study on human primates that investigated the effects of power wielding on the subordi-

nate under controlled experimental conditions. Epidemiologic studies and meta-analyses often, although not consistently, demonstrate an inverse correlation between socioeconomic status (SES) and consumption of illicit drugs and alcohol (see, e.g., [78–80]) including the use of cocaine [81, 82].

Effects of Psychostimulant Consumption on the Dopamine System

If subordinate individuals are vulnerable to psychostimulant (i.e., cocaine or methamphetamine) abuse and dependence, as the abovementioned data show, what are the consequences of psychostimulant abuse on the dopamine system and, hence, to the sensitivity of a subordinate and psychostimulant abusing individual? When compared to normal controls, humans who had abused cocaine for at least 6 months at a rate of at least a self-estimated "4 g" per week showed a –14% decrease in dopamine D2 receptor availability in their basal ganglia, which persisted 3–4 months after detoxification, the D2 receptor availability being negatively correlated with depressive symptoms as assessed with the Beck Depression Inventory [83]. Dysphoria and other depression-like symptoms are well known in psychostimulant withdrawal [25].

Neural Working Model of PAD

If, as detailed above, power wielding increases D2/D3 receptor availability, thus most likely increasing the sensitivity for rewarding stimuli, whereas subordination decreases D2/D3 receptor availability and whereas psychostimulant abuse itself decreases D2/3 receptor availability, then power-wielding individuals should be protected against dependence syndromes. Why then does power wielding seem to be a stimulus that has abuse liability, that is, why does power wielding carry the risk of rendering susceptive individuals dependent on it, resulting in PAD?

Our neural working model of PAD (Fig. 1) tries to reconcile these seemingly discrepant findings. From the psychotherapeutic perspective, it is very plausible that a power wielder has to fear her/his loss of power because social circumstances render this likely and/or because the power wielder's fear is unrealistically enhanced due to her/his psychopathology. In our model, loss of power constitutes withdrawal, that is, ICD10 criterion #3. Interestingly, the Addiction Research Center Inventory (ARCI), a ques-
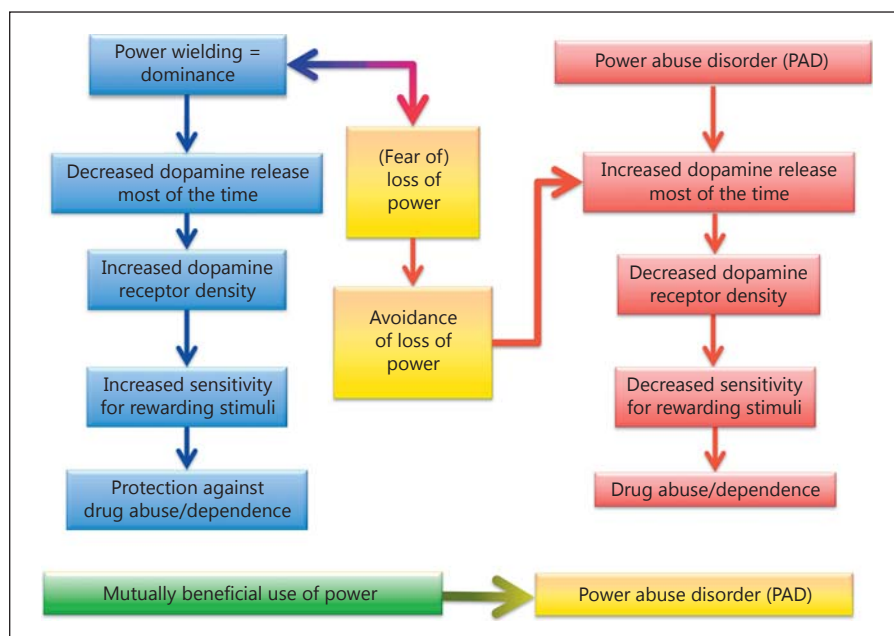
Fig. 1. Neural working model of power abuse disorder (PAD). Shown is the progression from the use of power that is beneficial for both the socially dominant and subordinate partner in a power dyad to PAD, which is harmful for both.

tionnaire published as early as 1963 and based on self-reports of drug users [84], lists the statement "I fear that I will lose the contentment that I have now" in the Morphine Benzedrine Group (Benzedrine® was a trade name for amphetamine). Similarly, when cocaine-dependent subjects received an infusion of 0.6 mg/kg i.v. cocaine over 30 s, after around 15 min, self-ratings of "low" and "craving" already reached a maximum when the "high" score had declined from its maximum but was still half-maximal (see Figure 2 of [18]). Thus, the fear of the approaching termination of the addictive stimulus is known to produce intense – and highly motivational – feelings in drug abusers. The same is to be expected from power abusers, as illustrated by a vast number of historic accounts of "paranoid tendencies" in despotic rulers in a number of different cultures.

Thus, the anticipated loss of power is expected to stimulate the power wielder's motivational neuronetworks, with increased accumbal dopamine as the major neurochemical correlate. The dopamine release induced by anticipation/fear and the resulting avoidance behavior, in turn, leads to a decrease in available D2/D3 receptors. Once this occurs, the power wielder is caught in an oscillation between (1) a state in which he/she finds him/herself under the influence of an increased dopaminergic tone most of the time, which decreases his/her DA receptor sensitivity (by the downregulation of receptor density and/or decrease in signal transduction efficacy (loss of power), and (2) a state in which dopamine may

be transiently (phasically) increased during power wielding but remains lower for most of the time, upregulating DA receptors and/or increasing signal transduction efficacy. Once the balance between the 2 states tips in favor of state (1), a downward spiral can result that is very similar to the allostasis model proposed for drug abuse ([19], compared to other models of addiction in [21]): as PAD progresses, the initial D2/D3 receptor sensitivity is never obtained again, resulting in the fulfillment of the well-known dependence syndrome diagnostic criteria of withdrawal (ICD10 criterion #3; [24]) and neglect of alternative stimuli (ICD10 criterion #5), which in the case of PAD would be all non-power stimuli. Our working model (Fig. 1) predicts that all drugs of abuse that are powerful dopamine releasers, that is, psychostimulants, remain the most attractive pharmacologic class of drugs of abuse for PADed individuals, especially if their imagined or real loss of power occurs at a time when their own dopamine resources are depleted, providing a much desired boost to a flagging motivated behavioral output.

Zeitgeist Diagnosis "Burnout" May Very Likely Hide PADed Individuals

As described above, many PADed individuals are expected to hide symptoms that they believe may be perceived as "weakness" and thus may threaten their pow-

erful position. On the other hand, the Zeitgeist diagnosis of "burnout" or "burn out" (no separate diagnosis in the DSM5 yet, and listed under ICD10 code Z73) may offer PADed individuals a socially sanctioned diagnosis. The term "burnout" itself evokes the image of exhausted resources (i.e., low dopamine levels and/or a decreased signal transduction in the dopamine system, as suggested in the present article). Of note, most of the 9 symptoms of the subscale "emotional exhaustion" of the widely used Maslach Burnout Inventory (MBI) [33], that is, "I feel emotionally drained from my work," "I feel used up at the end of the workday," "I feel fatigued when I get up in the morning and have to face another day on the job," "working with people all day is really a strain for me," "I feel burned out from my work," "I feel frustrated by my job," "I feel I am working too hard on my job," "working with people directly puts too much stress on me," and "I feel like I am at the end of my rope" bear a striking resemblance to some symptoms experienced during stimulant withdrawal, that is, fatigue, hypersomnia, and psychomotor retardation, all of which are plausibly due to an exhaustion of the dopamine system (withdrawal generally carries the DSM5 code 292.0, without a specifier for (psycho)stimulant withdrawal; however, psychostimulant withdrawal symptoms are described in detail on p. 569 of [25]). Please keep in mind that we think that PAD is associated with an overstimulation of the dopaminergic system (Fig. 1) that leads to its exhaustion, thus producing symptoms that are due to psychostimulant (i.e., dopamine releaser) withdrawal. Interestingly, 2 burnout studies found that individuals who were hierarchically higher suffered more from these exhaustion symptoms than their lower-ranking coworkers within the same workplace, that is, Swiss industries of various sizes [34] or the Bavarian school system [35]. The researchers of the Swiss sample described this as a "reversed social gradient." In the Bavarian sample, a social bias toward overdiagnosing and overtreating the surveyed school principals as opposed to their lower ranking teaching staff seems unlikely, because in our opinion even "simple" Bavarian teachers (and not only principals) enjoy globally outstanding social security. A survey of anesthesiology chairpersons in the United States [36] found that 28% (i.e., 26 of 93) chairpersons met the criteria for high burnout and an additional 31% met the criteria for moderately high burnout. Among the aggravating issues reported by the chairpersons [36] were "faculty retention" (indicating power struggles) and "problems with departmental budget" (again, a power related is-

sue). Unfortunately, this study did not compare chairpersons to hierarchically lower personnel.

One burnout study found a higher rate of burnout in intermediate (as opposed to high or low) management positions [37], reflecting, in our opinion, the 2-sided power struggle of a "hierarchically sandwiched" individual. Accordingly, a Swedish study [39] found the smallest incidence of burnout in business owners (as opposed to white- and blue collar workers in companies), a finding that we would explain by the hierarchically supreme position of a business owner. Apparently, the financial stress of business owners seemed to impact on their incidence of burnout less than power struggles in companies [39]. In a similar vein, decision latitude, which increases with an increase in the hierarchic position within an institution, inversely correlated with burnout symptoms in a study of English civil servants (the so-called Whitehall II study, [38]). Subjective SES was also inversely correlated with burnout severity in an Israeli sample of long-term health care staff members [40]. Of great interest, the authors of a study of Finnish dentist – dental nurse dyads [41] suggest that the – hierarchically higher – dentist "*passes exhaustion*" to the nurse, another plausible reason why many humans are known to seek a hierarchically superior position in a group of conspecifics. To summarize, individuals in higher hierarchical positions report, to a large degree, burnout symptoms that we think can also be found in PADed individuals, with 2 studies [34, 35] reporting a higher percentage of burned out individuals in higher hierarchical positions than in lower hierarchical positions within the same work environment.

We therefore think that the Zeitgeist diagnosis "burnout" covers, at least in the MBI subscale "emotional exhaustion," symptoms that are a result of PAD. In PADed individuals, "burnout" can thus be considered a secondary diagnosis based on symptoms that are better explained and treated by correctly allocating them to PAD. To paraphrase, many PADed individuals may hide or may be misdiagnosed and suboptimally treated under the socially sanctioned diagnosis "burnout."

Future Clinical Research

We think it extremely worthwhile to investigate the clinical presentation of PAD and its neurobiologic correlates and to investigate if PAD is comorbid with psychostimulant use disorder, as the present article suggests. However, we opine that any scientific study systematically investigating the drug abuse patterns of

high-ranking individuals in hierarchic structures (e.g., the military, churches, political systems, businesses) would be extremely difficult to perform because (1) a PADed individual would not seek treatment during power wielding and (2) would likely be extremely reluctant to seek psychiatric/psychotherapeutic/psychologic help because of the social stigma of a diagnosis of a mental disorder that most likely is even more punitive for a high-ranking individual than a subordinate one (see also above). Most likely, PADed individuals of middle to lower ranks would seek therapeutic help during phases of foreseen, imagined and/or real loss of power, complaining about "burnout" (detailed above), "mobbing," "bossing," or presenting with depressive symptoms or conversion (psychosomatic) symptoms. We also think that these coping mechanisms are the reason why there is essentially no published clinical evidence on PAD.

A plethora of other questions have to be addressed with respect to the diagnosis, epidemiology, and comorbidity of PAD. The following questions are but a tiny sample: What is the prevalence and incidence of PAD in the general population? Is PAD prevalence and/or incidence proportional to the hierarchic position in a social system of interest, that is, is obtaining a higher hierarchic position the result of an underlying PAD or constitutes a higher hierarchic position an increased risk of developing PAD? In which ways do subpopulations or subcultures or various political systems differ with respect to the prevalence of PAD? Which mental disorders other than substance-use disorders is PAD comorbid with?

Possible Pharmacotherapeutic Approaches to Treat PAD

If, as discussed above, PAD is associated with a dysfunction of the mesocorticolimbic dopaminergic system, then medications that stabilize this system may be useful for the treatment of PAD. On pharmacologic principle, stabilization of a dysregulated (i.e., oscillating) dopaminergic system (with power craving associated with a hypodomanigergic state and power wielding associated with a hyperdopaminergic state, see above) could be a obtained with the partial dopamine D2 receptor agonist aripiprazole, which has proven efficacious in the treatment and prophylaxis of manic episodes in bipolar patients [85, 86] ((http://www.awmf.org/leitlinien/detail/ll/038-019.html). However, according to these reviews and guidelines, aripiprazole did not significantly improve depressive episodes in bipolar patients and resulted in extrapyramidal effects, akathisia in particular.

For hypodopaminergic episodes in the course of PAD, the dopamine transport inhibitor bupropion [87] may be effective. Bupropion is registered in some countries both as an antidepressant and as a smoking cessation aid [88]. Effective antidepressant plasma levels for bupropion and its active metabolite hydroxybupropion have been established [89, 90]. However, precisely because of its psychostimulant-like molecular target, neurochemic effects, and behavioral experimental effects (reviewed in [91]), bupropion possesses abuse liability for some patients [91, 92]. In that respect, and on the same pharmacologic principle, bupropion shares the fate of other substitution (maintenance) medications used for the treatment of cocaine–dependence syndromes [93, 94] and opioid–dependence syndromes [95–98]. Therefore, antidepressants without evidence of abuse liability despite widespread and long therapeutic use may be more safely used to treat the depressive symptoms of PAD.

Future Basic Research

As detailed above, the experimental evidence obtained so far [3, 8, 11] suggests that power wielding (social dominance) is associated with an increase in accumbal D2/D3 receptor density. In cynomolgous monkeys, it was shown that this increase in D2/D3 receptor density is a consequence of achieving power and not a trait of powerful individuals. Respective data in male and female rats or mice are still lacking, with the mouse being an especially important experimental genus due to the plethora of transgenic models available in mice [10, 99]. The most striking apparent discrepancy so far is that male and female individuals, despite showing the same increase in D2/D3 receptor availability, show an apparently opposite pattern of i.v. cocaine self-administration obtained by the same group using the same schedule of reinforcement [3, 8]. This apparent discrepancy needs to be resolved, again preferably in a widely accessible experimental species/genus (mouse and/or rat), and by quantifying cocaine reinforcement/reward in operant schedules of reinforcement that (1) optimally, avoid direct drug effects on responding, that is, second-order schedules of reinforcement with the cocaine stimulus delivered at the end of the session, or (2) at least, minimize a direct drug effect on responding, that is, progressive ratios of reinforcement (for detailed methodological discussions see, e.g., [21, 64]) and are complemented by rate-free measures of drug reward/

aversion, for example, CPP/conditioned place avoidance (CPP/CPA; see, e.g., [10, 100, 101]). Cocaine as (1) a dopamine transport inhibitor and, hence, an enhancer of extracellular dopamine in mesocorticolimbic brain regions, as well as (2) a drug of abuse with high abuse liability, seems an excellent tool to test (a) the consequences of the well-documented dominance-induced increase in D2/D3 receptors in reward neuronetworks with respect to the sensitivity of the power-wielding individual to reward in general and (b) with respect to the increased risk of PADed individuals for cocaine dependence, a comorbidity that seems well known anecdotally and which the findings in female cynomolgous monkeys [8] and male Lister Hooded rats [11] seem to support. Nader and coworkers have already contributed a great deal of data obtained in a food-cocaine choice experimental paradigm suggesting, in our interpretation, that, overall, the sensitivity to physiological stimuli (i.e., palatable food pellets) is not changed by the acquisition of power/social dominance [8, 71, 102]. Respective data obtained from widely used and easily accessible experimental paradigms and species (e.g., CPP/CPA in C57BL/6J mice or Sprague Dawley rats) would be desirable. Concurrent CPP paradigms (see, e.g., [99, 103]) could allow a direct comparison with the monkey food-cocaine choice data described above. As already stated in the introduction, animal models that operationalize a power struggle [1–10] with opposite consequences for the participants (i.e., winning/losing a fight) constitute a sound and well-validated animal experimental basis to study PAD. As stated above, research has focused exclusively on the subordinate/loser of such a fight. By singling out individuals with an ex-treme degree of power wielding under these controlled experimental conditions (with measures of situation-inappropriate power wielding to be developed by the field) and making them the focus of investigation, a lot could be learned about the neural correlates of PAD.

Conclusions

In conclusion, we have tried to establish the case that power wielding is a stimulus that may be of considerable abuse liability in some individuals. The resulting PAD can be considered a nondrug form of a dependence syndrome, sharing neuronetworks and pathophysiology with substance-use disorders/drug dependence. In this article, research avenues have been suggested to increase our understanding of PAD in an effort to help the victims of PADed individuals to overcome this socially harmful disorder and the PADed individuals themselves.

Disclosure Statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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