


Addicted to Self-esteem: Understanding the neurochemistry of narcissism by using cocaine as a pharmacological model

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Abstract

There are pronounced behavioural and neuroimaging parallels between cocaine abuse and narcissism. Although the observed commonalities are not specific to cocaine as opposed to other types of addiction, we argue that the relatively constrained molecular actions of cocaine and, more importantly, the covariance of narcissism-like behaviours with cocaine use build a strong case for taking the known effects of cocaine as a starting point for addressing the hitherto under-investigated neurophysiology of narcissism. In this review, we discuss the potential relevance of cocaine abuse as a pharmacological model of narcissism. We outline previous research on the role of monoamines across several domains affected in narcissistic personality disorder and subclinical narcissism, namely, selected personality traits, social behaviour, emotional empathy and self-referential processing. We propose that dysregulation in dopamine signalling might underlie addiction-like features of narcissism and that altered serotonergic signalling may account for affective components of narcissism and, in particular, explain the differences between grandiose and vulnerable subtypes. In conclusion, we provide recommendations for future research.

Keywords

Narcissism, neurochemistry, cocaine, dopamine, right anterior insula, insular cortex, empathy, impulsivity, serotonin, norepinephrine

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Introduction

Narcissistic personality disorder (NPD) refers to the stable presence of pathologically exaggerated narcissistic traits. According to diagnostic manuals, NPD is characterised by the presence of certain features or symptoms such as impairments of personality functioning (identity and self-direction),

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interpersonal functioning (empathy and intimacy) or pathological personality traits (grandiosity and attention seeking) that make up rubrics of criteria in DSM-5 (American Psychiatric Association, 2013). In clinical populations, it is associated with considerable disability (Stinson et al., 2008), treatment resistance (Kacel et al., 2017), poorer treatment outcome (Spatz Widom et al., 2006), higher risk for relapse (Casillas & Clark, 2002) and suicide (Giner et al., 2013).

Literature usually differentiates between two manifestations of narcissism: the grandiose and the vulnerable subtype (Tortoriello & Hart, 2018). The grandiose subtype (also known as overt narcissism) is defined by fearless, confident and approach-oriented behaviour. People with the grandiose subtype have a strong desire for interpersonal dominance and dismissive attachment styles. They induce jealousy as a means of getting power and control over others, especially their romantic partners (Miller et al., 2012). The vulnerable subtype (or covert narcissism) is displayed in neurotic, fearful and shy behaviour. People with this subtype of narcissism often avoid pressure and stress and come across reserved at first. They also induce jealousy to attain security whilst gaining power from it (Miller et al., 2012).

However, according to more recent conceptualisations, rather than representing two distinct subtypes, grandiose and vulnerable aspects are better understood as dynamically changing, and often coexisting behavioural manifestations of narcissism that are expressed in response to environmental conditions and social context to varying degree according to an individual's predisposition. Engaging either reward- or threat-processing systems, grandiose and vulnerable manifestations of narcissism are dominated by behavioural patterns of approach and avoidance, respectively. According to the 'narcissism spectrum model' proposed by Krizan and Herlache (2017), at the core of what defines a narcissistic personality is 'a sense of oneself and one's needs being special and more important than others'.

Psychoanalytical theories claim that, narcissism originates in early preverbal object relations when parents fail to provide basic attention to the needs of a child (Auerbach, 1993). With the experience of being helplessly exposed to interoceptive sensations signalling existential threats such as abandonment, hunger or thirst, the child would create defence mechanisms for suppressing painful bodily feelings and feelings of abandonment, loss and rage. This results in constant and intense feelings of fear, abandonment and doubt, created by neglectful, overprotective or controlling parenting (Ronningstam, 2005; Ronningstam et al., 1995); derailed mechanisms of ego-defence from strong emotions may subsequently translate into narcissistic adult behaviour (Campbell, 1999; Kernberg, 1975).

Several theories claim that narcissism is related to fluctuation between high and low self-esteem, or unstable self-esteem: specifically, while people with narcissism have positive self-esteem and an independent self-construal, they

show a strong desire to maintain a pretentious self-image and strong need for admiration of others (Rohmann et al., 2012). Although the assumption that people with grandiose narcissism conceal personal insecurity behind a veneer of confidence due to a need for admiration is sometimes questioned, it is endorsed by many publications on narcissism (Bosson et al., 2008). When romantic partners try to break down this façade, consequences often result in emotional annihilation and occasionally end in homicide or suicide (Powell, 2010). To underline this risk, research has confirmed an overlap between psychopathy and grandiose aspects of narcissism: in addition to grandiosity, both conditions are associated with manipulation, low anxiety and a lack of empathy (Tortoriello & Hart, 2018). Vulnerable narcissists tend to alternate between feelings of superiority and inferiority, have fragile self-confidence due to low self-esteem and have an interdependent self-construal (Rohmann et al., 2012). Theories show that in NPD patients the amount of self-esteem discrepancy correlates with self-related anxiety displayed (Rohmann et al., 2012).

Despite its clinical relevance, studies on the neurobiological mechanisms of narcissism as of yet are scarce. Genetic studies in conditions related to narcissism (e.g. antisocial or borderline personality disorders) (Caspi et al., 2008; Langley et al., 2010; Silberschmidt & Sponheim, 2008) and imaging studies on the relationship of monoamine or opiate receptor binding to dimensional personality measures (Breier et al., 1998; Caravaggio et al., 2017; Farde et al., 2018; Moresco et al., 2002; Rodman et al., 2017) have provided significant evidence for the existence of specific biological correlates of personality disorders and the quantitative expression of personality dimension. In order to explore new ways of thinking about narcissism, we searched for pharmacological models that could inform future research into the neurobiology of narcissism. While the prevalence of substance use disorders in NPD is high (Stinson et al., 2008), a study relying on a much smaller dataset (Yates et al., 1989) describes particularly high rates of co-occurrence with alcohol and cocaine abuse. Additionally, acute and chronic use of cocaine induces a behavioural syndrome that emulates narcissism in several aspects (Gawin, 1991; Vonmoos et al., 2019). In particular, there seems to be a strong relation between acute and chronic effects of cocaine and the grandiose manifestations of narcissism. Thus, in this review, we report on behavioural and imaging findings in cocaine abuse and narcissism and discuss to what extent the mechanisms of action of cocaine could inform on some of the neurobiological mechanisms supporting narcissistic behaviour.

Cocaine

Cocaine is a naturally occurring alkaloid purified from extracts of the coca leaves. Besides central nervous effects,

it is a potent local anaesthetic and as such still used in medicine today. Cocaine readily penetrates the blood–brain barrier, has fast pharmacokinetics and pronounced reinforcing properties that are mediated primarily by subcortical dopamine (DA) $D_{2/3}$ receptors. These properties account for the high abuse liability of cocaine (Wang et al., 2019). Cocaine acts primarily by blocking the facilitated exchange-diffusion of the brain monoamines DA, serotonin and norepinephrine through their respective transmembrane-transporters. By blocking reuptake into the presynaptic neuron (and glial cells), cocaine induces a rapid and pronounced increase in extracellular monoamine concentrations and thus greatly enhances signal transmission at postsynaptic monoamine receptors (Carboni et al., 2001). In the periphery, systemic administration of cocaine has sympathomimetic effects and induces vasoconstriction, an increase in blood pressure, heart rate and plasma-concentrations of stress-related hormones such as cortisol (Baumann et al., 1995). Abstinence after prolonged abuse of cocaine may induce a state of anhedonia, low mood and fatigue that lasts for some days. Physical cocaine withdrawal symptoms are rather unspecific; yet, in some patients, withdrawal or DA depletion during cocaine binges can lead to severe depression and even acute suicidality (Lerner & Klein, 2019). Still, electroencephalographic markers show that hyper-reactivity to cocaine-associated cues persists for more than six months after desisting from the drug (Parvaz et al., 2016).

Cocaine is frequently consumed in a bingeing pattern of abuse, where days of intense and repeated use alternate with periods of abstinence. Cocaine pharmacokinetics are rapid: smoked as free-base, delivery to the brain occurs within seconds, when snorted, within 15–45 minutes and elimination-half-lives are approximately 30 and 120 minutes, respectively (Jeffcoat et al., 1989). Along these temporal dynamics, fluctuations in plasma cocaine levels lead to rapidly waxing and waning concentrations of extracellular monoamines in the brain resulting in the characteristic alternations between euphoria and a state of psychomotor tension, impulsiveness, dysphoria and an intense urge for consuming more of the drug, a state also known as craving (Howell & Kimmel, 2008).

Similarities between cocaine effects and narcissistic behaviour

Acute behavioural effects of cocaine include elevations in mood, energy and libido, and an increase in self-confidence and ‘sense of agency’, the feeling of being in control of one’s actions (Gawin, 1991). Dampening the perception of physical and mental distress while increasing the incentive salience of positive stimuli, cocaine reduces avoidance behaviour and shifts the motivational balance towards approach and risk-taking behaviour (Bartzokis et al., 2000; Fillmore et al., 2002; Verdejo-Garcia et al., 2008).

In many aspects, behavioural alterations in cocaine abuse resemble those observed in NPD, especially with regard to its grandiose manifestation. Narcissists show increased confidence and self-esteem (Watson et al., 1984), report being less depressed, anxious and lonely (Sedikides et al., 2004; Watson et al., 1984), exhibit heightened approach behaviour and tend to pursue rewards rather than avoiding undesired outcomes (Foster & Trimm, 2008). Narcissism is further associated with increased impulsivity (Vazire & Funder, 2006) and a bias towards taking higher risks in decision-making (Bosson et al., 2008). And similar to what is found with cocaine (Baumann et al., 1995), narcissistic subjects show increased plasma cortisol levels, at baseline and in response to stress (Edelstein et al., 2010; Reinhard et al., 2012). One of the few prospective studies addressing the neuropharmacology of narcissism showed that chronic cocaine abuse is also associated with an increase in narcissistic traits and deficits in social cognitive functions such as emotional empathy (Vonmoos et al., 2019). Since many of the behavioural alterations in cocaine users were reversible with abstinence, the findings of the one study strongly suggested that the transient increase in narcissistic behavioural traits is due to a direct pharmacological effect of cocaine (Vonmoos et al., 2019).

Imaging in narcissism and cocaine abuse

Imaging narcissism

Studies using structural and functional magnetic resonance imaging (MRI and fMRI) in NPD, although limited in number, have identified consistent alterations in specific brain networks. Brain structures repeatedly associated with NPD include the insular cortex (in particular the right anterior insular cortex; right AIC), the dorsal anterior cingulate cortex and frontal cortical areas such as the dorsolateral prefrontal cortex and the ventromedial prefrontal cortex (Cascio et al., 2015; Fan et al., 2011; Jankowiak-Siuda & Zajkowski, 2013; Scalabrini et al., 2017; Schulze et al., 2013). The relevance of these brain structures for narcissism is further supported by imaging studies on psychological domains or behavioural traits involved in narcissism such as empathy (Lamm & Decety, 2007; De Vignemont & Singer, 2006; Jackson et al., 2006; Leigh et al., 2013; Rankin et al., 2005; Sturm et al., 2006), impulsivity (Frost & Rickwood, 2017), anticipation of social or physical pain (Eisenberg & Eggum, 2009), anxiety (Paulus & Stein, 2006) or interoceptive awareness (Craig, 2009).

Imaging cocaine

A large proportion of the MR-based imaging literature on cocaine concerns the effects of long-term abuse on brain function, connectivity or structure. Comparably, few studies

address the acute cocaine effects. Functional changes after acute cocaine-administration occur in extended neuronal networks comprising dopamine-rich areas in subcortical reward-circuits (such as ventral striatum, substantia nigra and ventral-tegmental area) and cortical regions, including, among others, the ventromedial, latero-medial, dorsolateral prefrontal cortex, the inferior frontal gyrus and the cingulate cortex (Breiter et al., 1997). Aiming at cocaine effects in dopamine-rich nuclei of the subcortical ‘reward-circuit’, an early fMRI study shows clear and significant blood-oxygen level dependent (BOLD) signal changes in the right AIC (Breiter et al., 1997). A study investigating primates exposed to chronic cocaine confirmed functional changes in the ventromedial prefrontal cortex and ventral striatum (Porrino et al., 2007) while a rodent study found decreased metabolic activity in the anterior cingulate cortex, the insular cortex and the dorsolateral striatum and increased metabolic activity in the mesencephalon, amygdala and hippocampus (Nicolas et al., 2017). Subsequent studies have repeatedly demonstrated reduced grey matter volume and functional alterations in the bilateral insula and especially the right AIC in acute and prolonged cocaine use (Cisler et al., 2013; Garavan et al., 2008; Geng et al., 2017).

Cocaine effects

Dopamine

Here, we briefly review positron emission tomography (PET) and single-photon emission computed tomography (SPECT) studies on monoamine dysregulation in cocaine abuse. While cocaine has approximately the same affinity for dopamine, serotonin and norepinephrine transporters (DAT, SERT and NET), its effects on DA neurotransmission are by far the best characterised. Studies consistently show low D2 type receptor availability in the striatum, with evidence pointing to receptor downregulation as the most likely cause (Schneier et al., 2009). A concomitant increase in D3 receptor availability has also been shown in all studies employing a D3 preferring tracer (Boileau et al., 2015). Other findings include blunted striatal and cortical DA release in response to behavioural and pharmacological challenges, as well as lower availability of D2/3 receptors in cortical areas (Trifilieff & Martinez, 2014). Cox and co-authors found that even before the onset of manifest addiction there is a dorsal dopaminergic response as cocaine-related cues increased extracellular dopamine in the dorsal striatum of recreational cocaine users (Cox et al., 2017). Results of Ashok et al. (Carlson (1998) showed inconsistencies with regards to DAT as few studies showed no significant difference in DAT availability in cocaine users, whereas others revealed elevated DAT availability in short-term abstinent cocaine users (Proebstl et al., 2019). Moreover, low availability of D2 receptors and blunted

release has been observed with most substances of abuse (Volkow & Baler, 2014). Higher D3 availability also seems to be a feature of addiction in general and has been linked to impulsivity and the motivation for using drugs (Boileau et al., 2015). Even after cessation of cocaine use, changes of peripheral hormonal indicators related to dopaminergic function are evident (Satel et al., 1991).

Serotonin and norepinephrine

There is limited research available on SERT and NET in association with cocaine. An early SPECT study found higher SERT binding in chronic cocaine users (Jacobsen et al., 2000), but a recent postmortem study could not find any differences between protein levels of SERT (Tong et al., 2020). Matuskey et al. (2014) showed lower serotonin 1B receptor availability while (Ding et al., 2010) studied NET and found it upregulated. While the sparsity of research on serotonin and norepinephrine in cocaine use prevents from drawing any conclusions about the level and directions of alterations in these two neurotransmitter systems, changes are extremely likely given cocaine’s mechanism of action.

Linking monoaminergic function to personality and behaviour

Neuromodulators, DA, serotonin and norepinephrine are involved in regulating a wide array of cognitive processes, ranging from very basic functions to highly complex behaviours. Furthermore, the three systems are tightly intertwined through complex patterns of mutual regulation. This makes it very difficult to link a particular aspect of monoaminergic signalling to specific behaviours or traits. However, as summarised below, correlational evidence and findings from interventional PET and SPECT studies on personality dimensions (for recent review see Farde et al., 2018) could have implications for narcissism.

Serotonin

Studies investigating the role of the serotonergic system in personality and personality disorders show considerable variability in methods and results. However, the following studies provided relevant insights. Individual differences in 1A receptor availability were found to be linked to trait neuroticism, which in turn is tightly linked to vulnerable narcissism in particular (Hirvonen et al., 2015; Maciantowicz & Zajkowski, 2020). Neuroticism has also been linked to higher SERT availability in the thalamus (Takano et al., 2007).

Though small in effect, there seems to be an inverse relationship between serotonin and aggression (Duke et al., 2013). Serotonin has a beneficial effect on emotional empathy specifically, as amply demonstrated by studies using methylenedioxymethamphetamine (MDMA) and psilocybin,

a naturally occurring psychedelic compound (Hysek et al., 2014; Kuypers et al., 2017; Preller et al., 2016). Together, these seem to implicate action at 1A and 2A receptors. Psilocybin reduces the neural response to social exclusion (Preller et al., 2016). Cascio et al. (2015) inversely applied this paradigm to narcissism (exaggerated response to social exclusion) and one study reports similar findings in cocaine users (Hanlon et al., 2019). In another study, prolonged administration of the selective serotonin reuptake inhibitor (SSRI) escitalopram led to less frequent endorsement of negative adjectives in relation to self, which was accompanied by altered neural responses (Maron et al., 2016). Acute administration of the SSRI citalopram, however, had no effects on self-referential processing (Hobbs et al., 2020). Hobbs et al. (2020) also noted an increase in prosocial behaviour, something that was also shown in studies with psilocybin and MDMA (Gabay et al., 2018). This is supported by the fact that tryptophan depletion reduces cooperation (Wood et al., 2006). Interestingly, a study comparing the effects of a single dose of the SSRI citalopram with those of a single dose of reboxetine, a selective norepinephrine reuptake inhibitor (SNRI), found increased social engagement and cooperation and a reduction in self-focus after reboxetine administration (Tse & Bond, 2002), while in another study of the same authors, participants tended to be more self-confident and assertive after daily intake of the SNRI reboxetine over a period of 2 weeks (Tse & Bond, 2006).

Dopamine

Lower D2 availability in the striatum has been linked to trait personality detachment (Breier et al., 1998). Since receptor availability in PET and SPECT can be influenced by receptor density, affinity and also endogenous levels of the neurotransmitter (Laruelle et al., 1996), the interpretation of lower D2 availability in subjects scoring high in detachment is not straightforward. However, considering also the negative correlation between detachment and DAT availability described by Laakso et al. (2000), it is probably safe to say that greater detachment goes along with a dampened dopaminergic tone. Lower D2 availability has also consistently been linked to higher scores on the lie scale, indicating the tendency to present oneself in a better than realistic light and has also been linked to lower socialisation in (Caravaggio et al., 2017). Another study included the correlations between low D2 levels and higher novelty seeking (right insula and midbrain) and overvaluing social status, as well as the association between higher DA release in amygdala and rACC and lower trait anxiety (Berry et al., 2019). Caravaggio et al. (2017) showed higher D3 availability could be linked to lower attachment and social status.

Altogether, individual differences in dopamine signalling appear to be reflected in dispositional social traits. A

pattern of dopamine receptor availability seen in cocaine abuse as well as other types of addiction seems to be associated with lower proclivity towards getting involved with others, and a tendency to provide inflated self-descriptions. In relation to this, it is worth mentioning that Blackwood et al. (2003) associated self-serving bias with activity in the bilateral caudate. Inflated self-descriptions could be explained either in terms of intentionally projecting a socially desirable image (see e.g. (Egerton et al., 2010)) or as reflecting self-deception. Support for the association between DA and important features of NPD can also be found in genetic studies. For example, a genetic variation in an enzyme degrading monoamines such as DA and norepinephrine, catechol-O-methyltransferase (COMT), has been associated with narcissism scores in relatives of patients with schizophrenia (Silberschmidt & Sponheim, 2008).

All of this should be interpreted very cautiously since individual differences in receptor availability in PET/personality studies could be explained differently than in cocaine abuse. Concerning cocaine abuse, we have good reasons to believe dopamine signalling is attenuated in the absence of the drug, which could be a predisposition to addiction factor, or an adaptation to chronically elevated dopamine levels by regular consumption of cocaine.

Norepinephrine

Tse and Bond (2002) found that norepinephrine seems to foster prosocial behaviour with a single dose of reboxetine and assertiveness with two weeks of reboxetine (Tse & Bond, 2006). With regard to self-referential processing, Miskowiak et al. (2007) found no effect of acute reboxetine on self-referential processing fMRI signals, but did find it biases subsequent recollection towards positive words. Norbury et al. (2008) showed enhanced BOLD responses to positive self-referent words and better recall after seven days reboxetine intake. Involvement of the norepinephrine system in narcissism is further suggested by findings on an exaggerated stress response in highly narcissistic subjects, including higher levels of alpha amylase (Cheng et al., 2013), a marker of noradrenergic activity (Thoma et al., 2012) and hypervigilance to ego threat (Ditzen & Heinrichs, 2014).

In summary, both serotonin and norepinephrine appear to affect self-referential processing, in that sustained blocking of their reuptake induces a bias towards more positive self-descriptions during processing and subsequent recall. In the social domain, enhancing extracellular levels of the two neurotransmitters seems to have beneficial effects, in that it promotes cooperation while making people less submissive and more self-confident. Serotonin specifically seems to mediate emotional empathy, reduces responses to social exclusion and proneness to neuroticism (Carhart-Harris & Nutt, 2017). There is convincing evidence supporting the involvement of monoamines in the traits and behaviours

that are the most affected in narcissism. Some of these findings align well with results from neuroimaging studies in cocaine abuse. Still, more focussed research is required to draw resilient conclusions in this direction.

Narcissism as addiction to self-esteem

As previously stated, the most consistent findings in cocaine users concern a pattern of dopamine function that is present in most substances of abuse as well as some (though not all) types of behavioural addictions. This is particularly interesting, given influential theories that describe narcissism in terms of addiction to self-esteem. Baumeister (2001) compares fluctuation in narcissistic behaviours to the three hallmarks of addiction and found that a narcissists' personality traits are not stable, but rather a constant search for yielding their addiction to inner urges.

A recent DTI study (Chester et al., 2016) found weakened frontostriatal connectivity associated specifically with grandiose narcissism. Structural connectivity is associated with the trait self-esteem and functional connectivity is associated with the state self-esteem. This has been interpreted as a possibly underlying deficiency in integrating the representation of self with feeling of reward (Chavez & Heatherton, 2015).

It is hypothesised that this could lead people with narcissistic traits to compensate by pursuing self-affirmation. If deficient, fragile or unstable self-esteem is indeed a core feature of narcissism (Brown & Bosson, 2001). Evidence of negative affect as per fMRI during self-viewing (Jauk et al., 2017) may demonstrate an addiction-like state that requires constant external boosting. An addictive attitude to self-esteem could explain an excessive focus on self-image and relative disregard for other things – similarly to what is seen in addiction in general (heightened responses to drug cues, attenuated responses to natural rewards). As circumstantial support for this, Brailovskaia et al. (2020) found that both grandiose and vulnerable narcissism were associated with anxiety and addiction-like use of social media. We can infer that both grandiose narcissists, who tend to manage their condition successfully, as well vulnerable narcissist, who seem to have a more serious disruption in achieving their inner urges, are prone to have more anxiety. This anxiety then leads to addictive behaviour (e.g. in social media use) which assists in self-inflation. A potential difference between grandiose and vulnerable narcissism may then boil down to how efficient one is in managing “the addiction”, in other words, how good one is in achieving the desired self-esteem high.

Conclusion

While our attempt of understanding narcissism along the lines of cocaine neuropharmacology is supported by

findings in the literature on behavioural and physiological similarities, it needs to be mentioned that functional and volumetric alterations discussed in this review are not specific to cocaine but are found also with other drugs of abuse.

Still, behavioural and neuroimaging parallels between narcissism and cocaine abuse suggest that acute and long-term effects of cocaine may be sufficient to induce a behavioural phenotype exhibiting important characteristics of narcissistic behaviour, especially in aspects relating to narcissistic grandiosity. However, while quick to evoke the concept of shared causes and conditions, phenotypic similarities are not necessarily expressed on similar molecular backgrounds. Prospective experiments elucidating the relationship between narcissism and cocaine abuse are lacking as of yet. However, similar alterations in brain structure and function, together with findings on a temporal covariance in the expression of narcissism-related personality traits, social cognitive functioning and increases or decreases in cocaine abuse (Vonmoos et al., 2019) may justify further studies on monoamine functioning in the AIC, dACC and related brain networks. We have also reported on evidence suggesting that neurotransmitter systems directly affected by cocaine may be relatable to behaviours that are exaggerated in narcissism, with serotonin in particular mediating affective components. Evidence shows that serotonin could be involved in empathy, thus being one direct regulator between the feelings of grandeur and vulnerability. Whether this is indeed the case cannot be established without directly studying those neurotransmitters in narcissism. However, we think that as candidate explanation, a dysregulation in DA, serotonin and norepinephrine systems and their interactions is plausible enough to inform future research. This especially concerns the insular cortex, which seems to be a key brain area when it comes to modulating the severity of drug craving in general. Although the results from functional and structural imaging studies in narcissism and cocaine abuse are surprisingly convergent, alterations in the insular cortex are neither specific to narcissism nor to cocaine abuse but have been described in several psychiatric diagnoses.

Thus, to further the understanding the link between narcissism and cocaine, we suggest that future studies in narcissism should primarily investigate whether dopamine signalling in individuals with NPD or high narcissistic traits is disrupted along the lines of addiction-like alterations, whether serotonin signalling can be related to empathy deficits and whether it mediates differences between grandiose and vulnerable subtypes of narcissism.

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