

Relations among Delay Discounting, Addictions, and Money Mismanagement: Implications and Future Directions

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Background: Delay discounting is a reduction in the subjective value of a delayed outcome. Elevated delay discounting is a type of impulsivity that is associated with harmful behaviors, including substance abuse and financial mismanagement. **Methods:** Elevated delay discounting as related to addiction and financial mismanagement was reviewed from psychological, neurobiological, and behavioral economic perspectives. **Results:** Addiction and financial mismanagement frequently co-occur, and elevated delay discounting may be a common mechanism contributing to both of these problematic behaviors. **Conclusions:** Future research on the relationships between delay discounting, substance abuse, and financial mismanagement can provide important insights for developing improved prevention and treatment strategies.

Keywords: delay discounting, impulsivity, substance abuse, financial mismanagement, addiction, neurobiology

INTRODUCTION

Impulsivity involves a tendency to act promptly with diminished regard for future consequences (1). Temporal or delay discounting is a reduction in the subjective value of a delayed outcome. Elevated rates of delay discounting are a manifestation of a type of impulsivity, impulsive choice, that has been associated with adverse functioning (2,3), including substance abuse and financial mismanagement. Addiction and financial mismanagement frequently co-occur, and elevated delay discounting may be a common mechanism contributing to both of these problematic behaviors. The overlapping nature of neurobiological systems that appear to mediate delay discounting, financial mismanagement, and substance abuse supports this possibility. This review will propose that

elevated delay discounting is a common mechanism that contributes to both substance abuse and financial mismanagement.

DELAY DISCOUNTING

The field of behavioral economics provides insight into impulsive behavior. This field is a branch of economics that combines microeconomic principles with experimental psychology to predict human behavior ((4); also see articles in this issue). Behavioral economic principles can be used to explain and understand why people might behave irrationally. Discounting of delayed rewards involves a behavioral economic principle which states that people overvalue immediate rewards and undervalue future rewards, which may correspond to choosing immediate over delayed rewards despite the relative sizes of the rewards (5). People with high levels of choice impulsivity discount delayed rewards at an escalated rate.

Delay discounting, considered a component of choice impulsivity (6), has been associated with measures of adverse functioning (2,3). The preference for immediate rewards in elevated delay discounting may result from a steep decrease in the subjective value of a reinforcer as a function of time (7). Elevated delay discounting is related to substance abuse (8–10) and money mismanagement (11), suggesting that higher delay discounting may be a mechanism that contributes to both problematic behaviors.

SUBSTANCE ABUSE AND FINANCIAL MISMANAGEMENT

Substance abuse and dependence are associated with financial mismanagement, and the two problematic behaviors frequently co-occur. The procurement of drugs

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requires money, and the availability of money can signal the opportunity to obtain drugs to a person with a substance use disorder. In a phenomenon called the “check effect,” drug use tends to be elevated in the first part of the month, when people receive their monthly supplementary security income checks (12–14). Receiving a large amount of money may trigger relapse to drug use in substance abusers (15) and make individuals more likely to precipitously terminate their participation in a residential substance-abuse treatment program (16). Financial mismanagement in substance abusers may result from spending large amounts of money to procure drugs. People with substance dependence have approximately twice the likelihood of carrying debt when compared with those without substance dependence, although they may not have less income (17). Interestingly, Rosen and colleagues (18) found that providing substance abusers with a money management intervention not only improved their money management but also improved some substance-abuse outcomes. These findings suggest that, in addition to financial mismanagement and substance abuse frequently co-occurring, they might represent part of a constellation of problematic behaviors, at the center of which is elevated delay discounting. Higher levels of delay discounting may drive financial mismanagement and substance abuse, leading individuals to devalue long-term goals, such as saving money or achieving sobriety, for immediate rewards, such as the thrill of impulse purchases or intoxication.

ELEVATED DELAY DISCOUNTING IN FINANCIAL MISMANAGEMENT

Across conceptual domains, a theme of balance between opposing systems has emerged to describe the determinants of irrational choice behavior. Strack and colleagues (19) proposed that consumer behavior is a product of the interaction between the reflective and impulsive systems. The reflective system contains schemata (such as urges, desires, and impulses) that may lie dormant in an individual’s mind until activated by stimuli in the environment. The reflective system’s schemata are centered around plans, goals, and rules. In a critique of the reflexive–impulsive model, Vohs (20) noted that while the impulsive system seems to be driven by its own energy, Strack and colleagues did not provide a mechanism to power the reflective system. To address this deficiency, Vohs proposed that the reflective system is powered by self-regulatory resources that govern controlled and regulated responses. It has been proposed that self-regulatory resources are limited in nature and can be depleted (20). Depletion of self-regulatory resources may diminish the ability of the reflective system to direct consumer behavior and allow the impulsive system to guide financial decisions (20). In a study by Vohs and Faber (21), depleting participants’ self-control resources by requiring them to engage in attentional, emotional, or mental self-control resulted in subsequent increases in four indexes of impulsive spending tendencies. According

to the reflexive–impulsive model, consumer choices are dynamic, with fluctuations that are influenced by the cognitive system in control of behavior at the moment. Individual differences in levels of impulsive choice and self-control may thus influence financial decisions.

Elevated delay discounting may influence financial mismanagement in several domains, including those involving credit card usage, credit card debt, income, and wealth. Credit cards allow consumers to obtain objects immediately and pay for them at a greater cost in the future. Because delay discounting involves a devaluation of future rewards, such as being debt free, and an overvaluation of immediate rewards, such as being able to purchase a desired object immediately, it follows that elevated delay discounting would be associated with credit card usage. Showing greater impulsivity on a measure of delay discounting was correlated with having credit card debt and with having higher amounts of credit card debt (11). Chabris and colleagues (22) measured delay discounting and determined its association with a variety of indices, including financial and behavioral measures. The authors reported that elevated delay discounting was associated with whether an individual pays his/her credit card bill in full at the end of a billing cycle, with failure to pay the bill in full associated with steeper discounting.

In the economics literature, “time preference” is a phrase used to describe a consumer’s preference for immediate utility over delayed utility (5), with “utility” referring to relative satisfaction. Individuals with high time preference emphasize their financial well-being in the present and immediate future while individuals with low time preference are more focused on their financial well-being in the distant future. Becker and Mulligan (23) conducted a series of analyses to determine the role of many variables, including wealth, on time preference. When discussing their model, they used the terms “rate of time preference,” “impatience,” and “discount factor” relatively interchangeably to refer to the extent to which an individual discounts their future financial well-being. Becker and Mulligan proposed that wealthier individuals have higher time preference (more patience) than the less wealthy individuals. From their analyses of income data from three generations in which there was a positive relationship between a person’s adult annual family consumption (implying more financial means) and his parents’ income when he was a child, they concluded that wealth leads to patience (i.e., lower rates of delay discounting). The immediate utilization of an asset represents less of a gain for a wealthy individual (i.e., there is a low marginal utility of wealth), and the future utilization (investment) of an asset represents more of a gain for the wealthy individual. Essentially, wealthy individuals have less of a “push factor” (i.e., less of an immediate need compelling them to choose a smaller immediate reward) and more of a “pull factor” (i.e., a greater need compelling them to choose a larger delayed reward), and this is reflected in a larger incentive to invest because the future rewards they anticipate receiving will be larger. The resulting low time preference (i.e., lower rate of delay

discounting) leads to financial decisions that increase the wealthy individual's future wealth, thereby creating a positive feedback loop in which wealth begets wealth via a mechanism of lower rates of delay discounting. However, perhaps a more plausible explanation for lower rates of delay discounting among wealthy individuals is related to their anticipated stream of consumption. It is possible that, for poorer participants, immediate money is more likely to affect what they expect they will purchase in the near future. The rich are less likely to anticipate an effect on their near-anticipated stream of consumption based on the reward they select. Therefore, it is possible that the association between greater wealth and lower rates of discounting is driven by higher rates of discounting in poorer individuals, based on their greater need.

Several lines of research support Becker and Mulligan's proposed relationship between wealth and low rates of delay discounting, although the research is correlational and does not necessarily suggest a causal relationship. In an experiment by Green and colleagues (24), delay discounting in a hypothetical monetary task was inversely related to the level of income. Participants with lower incomes had higher rates of delay discounting than did individuals with higher incomes. The evidence for a relationship between delay discounting and financial mismanagement crosses cultures. In a sample of Indian participants, Pender (25) reported that delay discounting for hypothetical amounts of rice to be received immediately or in the future was inversely associated with household net wealth per capita. Interestingly, there was no relationship between delay discounting and wealth in Tsimane' Amerindians of the Bolivian rainforest (26), although there was an inverse relationship between delay discounting and quarterly income, an index that would contribute to later wealth. Research by Plunkett and Buehner (27) more fully characterized individuals with high and low monetary discount rates, revealing that individuals with an external economic locus of control (LOC) have higher monetary discount rates than the individuals with an internal economic LOC. Just as the devaluation of future rewards and preference for immediate rewards associated with elevated delay discounting contribute to poor financial decisions, they also contribute to drug use.

ELEVATED DELAY DISCOUNTING IN SUBSTANCE ABUSE

Similar to financial mismanagement, the use of illicit drugs, such as cocaine and heroin, involves the discounting of delayed rewards (e.g., health and freedom from incarceration) and preference for immediate rewards (e.g., intoxication). A relationship between high levels of delay discounting, or steep temporal discounting, and substance abuse has been demonstrated. Delays are discounted more rapidly by abusers of many drugs, including alcohol (28), cigarettes (29), cocaine (10,30), and heroin (30), than by non-abusing control subjects. Similar findings have been reported in individuals with pathological gambling (PG) (31,32). Cocaine-dependent

participants have shown higher discounting rates regardless of cocaine-use status (currently using vs. recently abstinent) (10). Individuals dependent on cocaine and those dependent on heroin discount hypothetical monetary rewards and hypothetical health and freedom outcomes more rapidly than do non-addicted controls (30).

In addition to illicit drugs, impulsivity is also associated with excessive use of licit substances. Petry (28) reported that individuals with alcoholism discounted delayed rewards more rapidly than did control subjects. Impulsivity is associated with early alcohol use, current use, early indicators of alcohol problems, and alcohol abuse and dependence (33). Individuals reporting cigarette smoking had higher levels of delay discounting than did non-smoking subjects (34) and were more impulsive on a behavioral choice task (35). Additionally, frequency of smoking and chronic level of nicotine exposure were both associated with more impulsive behavior in a delay-discounting task (36). The relationship between cigarette smoking and impulsivity also exists in adolescents. Smoking adolescents were more impulsive than the non-smoking ones on measures of delay discounting (29). Caffeine use was associated with impulsivity in men, but not in women (37), although Hewlett and Smith (38) reported no relationship between impulsivity and caffeine use. Additionally, caffeine use correlated with impulsivity in a sample of 60 university students (39), although the relationship may have been accounted for by the association between caffeine use and sensation seeking.

Not only does substance abuse occur more frequently in individuals with high levels of delay discounting, substance abuse itself may lead to an increase in delay discounting. Current smokers discounted delayed rewards more than never, occasional, or ex-smokers (9, 40). Current smokers' delay-discounting rates were higher than never-before smokers' rates across various hypothetical money magnitudes (41). While these results suggest that smoking may increase delay-discounting rates, it is also possible that high delay discounting predated cigarette smoking. Inclusion of the former smoker group by Sweitzer et al. (40) and Bickel et al. (9) helped to better characterize the relationship between smoking status and delay discounting. This approach was taken by Petry (28) with alcohol and delay-discounting research. Petry reported that the rate of delay discounting varied by drinking status, with currently drinking alcoholics discounting delayed rewards more rapidly than the abstinent alcoholics, who discounted delayed rewards more rapidly than the non-alcoholic controls (28). The relationship between drinking status and delay discounting among alcoholics and non-alcoholic controls suggests that alcohol-related delay discounting may result from an interaction between genetic predisposition and alcohol use. Inclusion of a currently drinking non-alcoholic control group may have provided additional support for this explanation.

Alternative explanations for the results of Bickel et al. (9), Petry (28), and Sweitzer et al. (40) warrant

consideration. For example, the lower level of delay discounting in ex-smokers when compared with current smokers (9) could result from reversible effects of nicotine or from a predisposition for better self-control that enabled the ex-smokers to quit smoking. Similarly, the higher levels of delay discounting in current smokers may have resulted from more impulsive predispositions that led them to initiate smoking. Longitudinal studies are needed to distinguish between these possibilities.

Just as administration of a drug may increase delay discounting, drug deprivation may also increase delay discounting in substance-dependent individuals. Mild opioid deprivation increased the degree that opioid-dependent outpatients discounted delayed heroin and money (42). The degree of discounting was higher for heroin than for money and was inversely related to the magnitude of the reward. Consistent with these results, deprivation from cigarette smoking increased impulsive choice for both cigarette and monetary rewards in a delay-discounting task (43), although discounting for cigarettes was not statistically different from discounting for monetary rewards. Nicotine deprivation may have increased delay discounting (44), but deprived smokers' impulsive choices were increased only for cigarettes and not for money. The described research may indicate that withdrawal from a substance of dependence increases delay discounting, with some research (42,44) suggesting that rates of discounting for the substance itself are especially elevated during withdrawal. However, the results of Mitchell's research, in which people were paying more "later money" to "smoke now," may also be explained as a shift in the valuation of cigarettes during nicotine withdrawal. Increased delay discounting or increased substance valuation during substance deprivation may increase vulnerability for relapse during the acute withdrawal phase and may also influence how individuals with addictions manage their money.

FINANCIALLY RELATED NON-SUBSTANCE ("BEHAVIORAL") ADDICTIONS: PG AND COMPULSIVE SHOPPING

Similar to drug addictions, non-substance or "behavioral" addictions may involve persistent or compulsive patterns of behavior that are associated with short-term reward and diminished self-control (45). Impulse control disorders (ICDs) have been described as behavioral addictions, but not all ICDs may constitute addictions (45). As in addictions to psychoactive substances, characteristics of behavioral addictions and ICDs include repetitive or compulsive engagement in a specific behavior (e.g., gambling) despite adverse consequences, diminished control over the problematic behavior, and tension or an appetitive urge state prior to engagement in the behavior (46–48). Behavioral addictions may include PG, kleptomania, compulsive buying, compulsive sexual behaviors (sexual addiction or non-paraphilic hypersexuality), excessive tanning, problematic computer/video game playing, and problematic Internet use or Internet

addiction (45). Individuals with behavioral addictions have scored high on self-report measures of impulsivity and sensation seeking (45,49–52), and people with greater baseline delay discounting may be more prone to develop ICDs (6). Individuals with behavioral addictions may excessively engage in a broad array of behaviors, and these behaviors may involve excessive spending and diminished control over finances. Money mismanagement may be particularly relevant to two behavioral addictions: PG and compulsive shopping. Elevated delay discounting may underlie PG and compulsive shopping and contribute to financial mismanagement and substance abuse in people with PG and compulsive shopping.

Several studies have indicated that people with PG have elevated rates of delay discounting (53,54). Subjects with PG discounted delayed rewards more rapidly than the control comparison subjects in a laboratory setting (54) as well as in a naturalistic setting for gambling behavior, an off-track betting facility (53). In a study of 62 individuals with PG, problem gambling severity was the single best predictor of impulsive behavior in a delay-discounting task (31). Multiple laboratory studies have revealed a relationship between PG and delay discounting. People with PG will discount delayed monetary consequences to an even greater degree when in a gambling context (e.g., an off-track betting facility) than when outside a gambling context (e.g., a coffee shop or restaurant) (55). Therefore, just as elevated delay discounting may drive financial mismanagement and substance abuse, it may also contribute importantly to gambling behaviors in PG. Consistent with a role of elevated delay discounting in these behaviors, rates of substance abuse and financial mismanagement are high in PGs.

PG frequently co-occurs with substance use disorders (56,57), including alcohol use disorders (58). People with both gambling and substance-abuse problems have particularly high levels of delay discounting (28,59,60). Among three groups of individuals (those who abused substances and had gambling problems, those who abused substances but did not have gambling problems, and those who neither abused substances or had gambling problems), the substance-abusing problem gamblers discounted delayed rewards the most rapidly, followed by the substance abusers who did not gamble problematically, and then participants who neither gambled nor abused substances. In research using the Iowa Gambling Task (54), the presence of both substance abuse and PG had an additive effect on preferences for decks containing greater immediate gains but resulting in large intermittent financial punishments and overall net losses. These findings appear to extend to gambling behaviors in tobacco smokers, with daily smokers gambling on more days, spending more money gambling, having higher "craving" for gambling, and having lower perceived control of their gambling when compared with non-daily smokers (60). Associations between substance use and PG are well established in the literature, and individuals with both substance abuse and PG may experience particularly severe gambling problems, given poor financial

management that may be reflected in high rates of delay discounting.

It makes intuitive sense that people with PG have financial problems. Consistently, PG is associated with financial troubles (61) including personal bankruptcies (62–65). Multiple clinical factors distinguish people with PG who filed for personal bankruptcy from those who did not, including having an earlier age of problem gambling onset, reporting daily tobacco smoking, and having other substance use disorders. While many financial problems in PG may result from gambling debts, people with PG also may have an increased likelihood of engaging in other finance-consuming behaviors, including substance abuse (66). Furthermore, the use of certain substances (e.g., alcohol) has been associated with increased gambling, which could in turn worsen financial problems, particularly among people with PG (67).

Compulsive shopping is also associated with financial mismanagement and substance abuse. Another financially relevant behavioral addiction, compulsive shopping, involves chronic, repetitive, and excessive purchasing that may include features of craving and withdrawal (68). While laboratory studies of delay discounting in compulsive shopping are needed, people with compulsive shopping may prefer small immediate rewards, such as impulse purchases, to the delayed reward of saving money. Compulsive shopping can lead to financial problems. For example, college students who engage in compulsive shopping are more likely than students who do not engage in compulsive shopping to hold credit card debt (69). Compulsive shopping is associated with substance abuse (70,71) and this finding extends to unaffected first-degree relatives (72) and high-school students (73). Even among high-school students, problem shopping included features of addictions, including urges to shop, attempts to cut back, missed opportunities due to shopping, and a calming effect of shopping, suggesting that problem shopping is part of larger constellation of addictive behaviors (73).

COMMONALITIES IN THE NEUROBIOLOGY OF DELAY DISCOUNTING, FINANCIAL MISMANAGEMENT, SUBSTANCE ABUSE, AND PG

Elevated delay discounting is an important component of financial mismanagement and substance abuse, and the extent of neurobiological overlap associated with the constructs suggests that delay discounting may be a causal mechanism of the two problematic behaviors. The same neural structures and chemicals associated with high rates of delay discounting are also associated with financial mismanagement and substance abuse. Activation of the ventral striatum (VS), medial prefrontal cortex (mPFC), and posterior cingulate cortex (PCC) is associated with the subjective value of a reward (74). The choice for a small, immediate reward, which can have more subjective value, has been reported to be associated with increased activation of dopaminergically innervated areas, such as the VS, ventral PFC (vPFC), and the medial portion of

the orbitofrontal cortex (OFC) (75). In financial mismanagement, increased VS activation is associated with monetary reward anticipation and processing, risky financial choices, and risk-seeking mistakes (76). Consumption of drugs of abuse releases extracellular dopamine (DA) in the nucleus accumbens of the VS, which is also associated with the drug's rewarding effects (77–79). Therefore, the neural system implicated in the choice for an immediate reward is also implicated in financial risk-taking, financial reward, and drug intoxication. Conversely, the insula is one neural structure in which increased activity is associated with choosing a larger, delayed reward or a non-risky financial option, or with processing a financial loss (76,80,81). The lateral PFC and the parietal cortex are also activated during the choice for a larger delayed reward (75).

The pattern of neural activity in the limbic and frontal areas may change when gambling (a financially relevant behavior) and substance abuse become pathological or addictive (79,82). In people with PG, VS, and ventromedial PFC (vmPFC), activity is decreased during simulated gambling and exposure to gambling cues (83,84). A similar pattern of decreased activity in the VS and PFC has been reported in drug-addicted individuals during performance of some of the same tasks (85–87). In addition to decreased VS and PFC activation, people with drug addictions may also have decreased activation in the OFC and the anterior cingulate cortex (86,87), suggesting a “hypofrontality” in people who are addicted to drugs (86,87). It has been proposed that decreased activity in the VS is one of many neural changes associated with the formation of compulsions (88). These theories and findings suggest that individuals with PG or substance use disorders may not only activate less robustly regions mediating inhibition and the evaluation of ongoing processes but also exhibit blunted neural responses during reward processing as indicated by relatively decreased VS activation during the anticipation of monetary rewards. However, other studies have found that individuals with addictions show enhanced cortico-limbic activations to drug and monetary cues (89,90). As cocaine- and alcohol-dependent subjects have shown differences in VS activation during monetary reward anticipation, with increased activation observed in cocaine dependence (89) and diminished activation in alcohol dependence (85,91), it is possible that some of the factors associated with vulnerability to specific forms of addiction or some of the effects of specific addiction processes (e.g., chronic exposure to cocaine vs. alcohol) may influence the processing of monetary rewards differently. More detailed discussion of the neurobiology is as follows.

THE NEUROBIOLOGY OF ELEVATED DELAY DISCOUNTING

Impulsive behavior may be conceptualized as the manifestation of an imbalance between neurobiological systems that subserve inhibition and activation. The

neurobiology of impulsivity involves interactions among multiple neurotransmitter systems, neural structures, and neural circuits (92). Dorsolateral and inferolateral frontal cortex gray matter volumes each show inverse correlations with preference for immediate reward in healthy individuals (93). The posterior insula is also important for delaying gratification, and activation in the VS may code for time delay (94). The insula, amygdala, and vmPFC each contribute importantly to decision making (95). The insula, implicated in interoceptive processing of somatic states, interacts with the vmPFC, implicated in affective and reward processing, to influence risk/reward decision making (95).

The neurobiology of delay discounting has been studied by observing the neural activity of individuals engaged in delay-discounting tasks, using such neuroimaging techniques as functional magnetic resonance imaging (fMRI). Additionally, white matter contributions have been examined using diffusion tensor imaging (DTI). White matter integrity contributes importantly to the functioning of brain circuitry. Using fMRI, McClure and colleagues (75) identified areas of the brain that were activated when choosing to select smaller immediate rewards or larger delayed ones. Choices involving an immediate outcome were associated with activation of dopaminergically innervated limbic regions, including the VS, and vPFC including the medial portion of the OFC. Selections of larger, delayed rewards were associated with the activation of more dorsal cortical regions in the lateral PFC and parietal cortex. While these findings are consistent with the notion of cortical contributions to selection of choices involving delayed gratification, the precise involvement of cortical regions in delay discounting is incompletely understood as lesion studies have not consistently supported the interpretation of a role for the PFC in reducing delay discounting (96). Consistent with the findings of McClure and colleagues, Hariri et al. (97) reported that increased preference for the smaller immediate reward was associated with “hyper-reactive” VS circuitry and that greater magnitude of VS activation was associated with a stronger preference for immediate over-delayed rewards. The research of Wittmann et al. (80) revealed a role for bilateral activation of the posterior insular cortex in selecting a delayed option, consistent with the notion that the insula contributes importantly to choices involving delayed gratification.

However, the findings of Kable and Glimcher (74) differed from those of the McClure group and may provide an alternative explanation for the McClure group’s findings. Kable and Glimcher (74) reported that neural activity in the VS, medial PFC, and PCC correlated with the subjective value of a delayed reward. These three regions had increased activity when the objective amount of a delayed reward increased, decreased activity when the delay to this reward increased, and increased activity when the delayed reward was chosen because of its greater value. The authors also reported that delay had a stronger effect on subjective value for more impulsive discounters. From their results, Kable and Glimcher

concluded that the neural activity in the VS, mPFC, and PCC tracks the subjective value of rewards as determined from behavior, rather than tracking a theoretically defined component of value. Although the findings of McClure and colleagues and Kable and Glimcher may seem disparate, a conceptual framework has been proposed to accommodate both findings (74). Kable and Glimcher, who found that activity in the VS, PFC, and PCC tracked the subjective value of rewards, suggested that the McClure group may have found increased activity in those regions during the selection of an immediate reward because the subjective value of immediate rewards is greater than that of delayed rewards.

Using DTI, Olson and colleagues (98) examined white matter integrity in children, adolescents, and young adults (age range: 9–23 years) to investigate the relationship with delay discounting. They reported better white matter integrity in the pathways that connect the lateral prefrontal and temporal parietal cortices with other brain regions associated with lower temporal discounting rates. They also reported that lower discounting was associated with better white matter organization in regions of the frontal cortex bilaterally, including areas near the insula and the OFC, the dorsolateral regions, and the left temporal lobe. Interestingly, there were no regions in which better white matter integrity was associated with higher rates of discounting. Some but not all findings persisted when controlling for age and intelligence. The authors offer the interpretation that connections involving the dorsolateral PFC may be particularly important in delay discounting. In individuals with low rates of delay discounting, prefrontal areas, which are associated with cognitive control, may be more effective in exerting cortical control over subcortical regions like the VS involved in reward valuation and processing. Self-regulation in response to reward cues may depend on top-down control from the PFC over subcortical regions involved in reward and emotion (99). Such mechanisms may be particularly important in addictions. When instructed to inhibit craving, cocaine users and smokers had increased activity in the PFC and reduced activity in regions associated with reward processing, such as the OFC and VS (100,101). It is possible that choosing the larger delayed reward involves the same type of top-down inhibition (101).

Multiple neurotransmitter systems influence impulsive tendencies and these include serotonin, norepinephrine, glutamate, and DA (92). Altered serotonin (5-HT) neurotransmission is implicated in impulsivity, but the exact mechanisms by which 5-HT neurotransmission impacts impulsivity are unclear (92). Dietary tryptophan depletion reduces the levels of serotonin, but this manipulation had no effect on a measure of delay discounting in people (102). Individuals characterized by increased impulsivity (those with pyromania, PG, impulsive aggression, and alcoholism) show differences from control comparison subjects in biochemical, behavioral, and neural responses to serotonergic drugs (82,103). For example, *meta*-chlorophenylpiperazine (*m*-CPP), a partial agonist at serotonin 5HT1 and 5HT2 receptors, leads to

differential prolactin release and subjective responses, with impulsive individuals tending to report a high or a buzz and control comparison subjects an aversive response. *m*-CPP administration also leads to differential neural activations in impulsive and control comparison subjects, with the former group showing relatively blunted responses to the drug in the vmPFC (104). Given *m*-CPP's affinity for 5HT1B receptors, it is possible that some of the effects of the drug are mediated through this receptor, one that influences VS DA function and has been implicated in alcohol dependence (105) and PG (106). However, the complexity of the 5-HT system and its interactions with other neurotransmitters that also influence impulsivity, such as norepinephrine, glutamate, and DA, complicate ascertaining serotonin's precise role in impulsivity as aspects thereof like delay discounting.

Studies implicate DA neurotransmission in impulsivity (48,92). Psychostimulants, such as amphetamines, are pro-dopaminergic and can influence other neurotransmitter systems (e.g., noradrenergic). Amphetamines and other stimulants, like cocaine, may enhance impulsive behaviors, and individuals dependent on stimulants often display elevated impulsivity (10). Effects of psychostimulants on individuals with attention deficit hyperactivity disorder (ADHD), a disorder that includes heightened impulsivity as a major component, also suggest a role for DA neurotransmission in impulsivity. However, stimulants in individuals with ADHD typically reduce impulsivity, suggesting that there exist individual differences in stimulant responsiveness relating to impulsivity. Additionally, the effectiveness of stimulants to reduce impulsivity in individuals with ADHD suggests that an optimal dopaminergic tone is needed to reduce impulsivity, and aberrations in either direction from this optimal dopaminergic tone may lead to increased impulsivity. Genetic polymorphisms associated with relatively increased striatal DA release and synaptic availability and decreased postsynaptic inhibition have been linked to reward-related VS reactivity, which in turn has been associated with impulsivity (107). Impulsivity has been correlated with D2/D3 receptor availability in the substantia nigra/ventral tegmental area and with the magnitude of amphetamine-induced DA release in the striatum (108). These results suggest that differences in function within ascending dopaminergic projection pathways subserving reward and motivation may underlie deficits in impulse control and increase vulnerability to substance abuse (108).

NEUROBIOLOGY OF FINANCIAL MISMANAGEMENT: INSIGHTS FROM REWARD PROCESSING EXPERIMENTS

Significant effort has been devoted to understanding the neurobiological underpinnings of reward processing, and this information can be interpreted in the frameworks of financial mismanagement and addictions.

In a series of fMRI experiments, Knutson and colleagues used a monetary incentive delay task to investigate the neural correlates associated with phases of reward and loss processing. Important neural structures involved in reward and loss processing include the VS and insula, respectively, although multiple other brain regions have also been implicated (81). Activation of the VS is observed in anticipation of monetary rewards (109), and errors in gain prediction, as well as monetary reward outcome notifications, are reflected by activation of the mesial PFC (MPFC), a region of the vmPFC (110).

Brain structures associated with risky financial decision making have also been investigated. Kuhn and Knutson (76) reported that VS activation preceded both risky choices and risk-seeking mistakes while anterior insula activation preceded both riskless choices and risk-aversion mistakes in a financial decision-making task. These findings resonate with others implicating the anterior insula in harm avoidance and loss prediction (111). In a subsequent investigation of neural antecedents of risky financial decision making, Knutson et al. (112) found that VS activation was positively correlated with preference during the product and price periods, MPFC activation was positively correlated with the price differential (the difference between what a participant was willing to pay and the displayed price of the product), and insula activation was negatively correlated with purchasing during the choice period. These findings are consistent with findings of increased activation of the VS in association with expected reward, and increased insula activation in association with expected risk (113). In addition, increased VS activity predicted making risky investments, making purchases, and investing in a cooperating partner. Increased insula activity, on the other hand, predicted making "safe," or non-risky, investments, refusal to purchase a product, and defecting against an unfair partner.

Research in which VS activation was associated with gain prediction and expected reward, preceded risky choices and risk-seeking mistakes, and predicted making risky investments and product purchases suggests an important role for the VS in financial risk-taking behavior and money mismanagement (76,109,112). However, other factors may link VS activation and risky financial decision making. VS activation is increased during anticipation of not only financial rewards (109,114) but also other rewarding stimuli, such as erotic pictures (115,116), and these may interact. Anticipation of viewing erotic stimuli increased financial risk taking, an effect that was partially mediated by increases in VS activation (117). These findings suggest not only that VS activation may underlie anticipatory responses to a wide range of pleasurable and rewarding experiences but also that specific environmental stimuli (sexual or possibly substance-related cues) may influence VS activation and behaviors that might in a complex fashion relate to poor financial management.

NEUROBIOLOGY OF SUBSTANCE ABUSE

Commonalities between the neural systems underlying drug addiction and impulsivity have been described (48,118). Dopaminergic systems are implicated in addiction, with low D2/D3 DA receptor availability observed in individuals with addictions (119). Abstinent individuals with alcoholism and individuals dependent on cocaine, heroin, and methamphetamine have reduced levels of D2/D3 DA receptors in the dorsal striatum (120), perhaps reflecting a genetic vulnerability, resulting from downregulation of D2/D3 DA receptors following continued DA release with repeated drug administration or occurring through other non-mutually exclusive mechanisms. Individuals with alcoholism show relatively diminished VS activity during monetary reward anticipation, with VS activation correlating inversely with impulsivity in the alcohol-dependent group but not in control comparison subjects (85). Such findings are consistent with findings of relatively reduced VS activation in PG subjects during simulated gambling and an inverse relationship between VS activation and problem gambling severity in PG (83). Neural changes in addicted individuals, including reduced VS activity during monetary reward processing, suggest that circuits involving VS underactivation may represent neurobiological substrates of elevated delay discounting, financial mismanagement, and substance abuse. However, other studies of reward processing in alcohol dependence have not observed relatively diminished VS activation (121), perhaps because of relevant individual differences. For example, the study in which diminished VS activation did not associate with alcohol dependence included a subject group with frequent co-occurring cocaine abuse/dependence. As a recent study observed relatively increased VS activation during reward processing in cocaine dependence (89), the findings suggest that factors like drug exposure may alter cortico-limbic function as related to the neural correlates of reward processing. "Hypofrontality," or relatively diminished activation of frontal cortical regions including PFC, may also contribute and reflect diminished top-down control over reward-seeking behaviors (86,87). When performing decision-making tasks, drug-addicted individuals show functional abnormalities in the insula, a structure involved in delaying gratifications and risky decision making, particularly with respect to loss aversion (94,122). From a broader perspective, overlapping neurobiological mechanisms in drug addiction, delay discounting, and financial mismanagement may mediate increased propensity to develop drug addiction or its severity, as well as how addicted individuals respond to or manage their money.

NEUROBIOLOGY OF PG

Abnormalities in levels of the neurotransmitters, serotonin (5-HT), DA, and glutamate, may contribute to PG (123). Serotonin is hypothesized to contribute to the control or cessation of certain behaviors. Low levels

of the serotonin metabolite, 5-hydroxyindolacetic acid, have been found in the cerebrospinal fluid of individuals with PG, as well as in individuals with substance use disorders (124). DA is involved in the rewarding and reinforcing properties of drug addiction. A cross-priming role of amphetamine, a drug that influences neurotransmission of DA and norepinephrine, in PG suggests that the promotion of DA or norepinephrine neurotransmission may increase PG behaviors (125). The DA receptor antagonist haloperidol also appears to enhance rewarding and priming effects in PG (126), suggesting that a role for DA in PG is complex. Other neurotransmitters, such as glutamate, may influence reward-seeking behavior by modulating DA release in the VS, and glutamatergic compounds like *N*-acetyl cysteine have shown preliminary success in the treatment of PG and substance use disorders (127,128).

Like people with drug addictions, those with PG show reduced activity in the PFC, particularly in its ventromedial component. Relatively diminished activation of the vmPFC has been observed in PG subjects during simulated gambling (83), cognitive control (84), and when viewing gambling stimuli (129). When compared with control subjects, individuals with substance use disorders with or without gambling problems showed relatively diminished vmPFC activation during the performance of the Iowa Gambling Task (130). Studies have also shown relatively diminished VS activation in PG during simulated gambling and in response to gambling cues, with the latter findings demonstrating similarities to people with cocaine dependence (82,83). Reduced activation of the vmPFC and VS in PG may involve reduced white matter tract integrity, particularly in individuals with PG and alcohol use problems (131). Taken together, delay discounting, financial mismanagement, substance abuse, and PG involve overlapping neural substrates.

PARKINSON'S DISEASE AND ICDs

The frequent occurrence of ICDs in medicated Parkinson's disease (PD) patients may reflect a manifestation of the neurobiological overlap of elevated delay discounting, financial mismanagement, and addiction. ICDs have been observed in patients with PD (6,132). In a cross-sectional study of over 3000 PD patients, DA agonist treatment was associated with 2- to 3.5-fold increased odds of having an ICD, such as problem gambling or PG, compulsive buying, compulsive sexual behavior, and binge or compulsive eating (132). In addition, multiple factors related to PD (age at PD onset), PD treatment (both DA agonists and levodopa), mental health problems (ICD prior to treatment onset), and factors seemingly unrelated to PD or psychiatric disorders *per se* (marital status and geographic location) have been associated with ICDs, suggesting that multiple factors may influence the development of ICDs in PD. In a comparison between PD patients with ICDs (PDI) and PD controls, DA agonist use status was associated with greater impulsive choice in PDI patients (6). Individuals with PDI

when compared with those with PD have scored highly on measures of self-reported impulsivity (6) and demonstrated differences in VS DA function (133), diminished VS perfusion, and diminished VS activation during risk taking (134,135). Among ICDs in PD, similarities in clinical characteristics (elevated anxiety and obsessive-compulsive and sensation-seeking tendencies) seen in people with PG and compulsive shopping may be greater than those for other ICDs (136). Together, these findings suggest that in specific individuals, DA function contributes to impulsive behaviors, several of which (gambling and shopping) involve direct managing of finances. Steeper temporal discounting is one factor that differentiates DA agonist-medicated PDIs from PD patients who do not develop ICDs (136,137). As multiple factors related to physical and mental health and sociodemographic characteristics have also been implicated in ICDs in PD (132), additional research is needed to understand how these factors might influence money management in individuals with impaired impulse control or addictions.

Associations among PG, substance abuse, delay discounting, and financial mismanagement may result from the effects of the variables on one another (e.g., the disinhibitory effects of substance abuse on delay discounting, gambling, and spending behavior). Alternatively, or in addition, associations among these variables may result from shared neurobiological vulnerabilities and effects of one type of behavior (e.g., substance abuse) on the brain, which then lead to increased levels of the other behaviors (e.g., delay discounting, gambling, and spending behavior). Delay discounting, financial mismanagement, substance abuse, and PG share important characteristics. Each construct involves an orientation toward immediate rewards and a rapid and frequently detrimental discounting of future rewards and consequences. In addition to this behavioral similarity, overlapping neurobiological characteristics contribute to each of these types of behaviors. In particular, the VS, vmPFC, insula, and dorsal PFC contribute importantly to risky decision making, and individuals with substance or behavioral addictions demonstrate functional differences in these regions in a manner that suggests that the differences may underlie behavioral aspects of financial mismanagement. Therapies that alter function of these regions, potential intermediary phenotypes like choice impulsivity, or financial mismanagement warrant consideration in the treatment of addictions (138,139).

CONCLUSION

In conclusion, delay discounting, financial mismanagement, and substance and behavioral addictions are linked conceptually. The associations between delay discounting and addictions, both behavioral and substance related, are substantial. Considering financial mismanagement as it influences addictions is important, and targeting intermediary endophenotypes like choice impulsivity may concurrently target addictive behaviors and

money mismanagement. The overlapping neurobiological mechanisms underlying delay discounting, financial mismanagement, and substance abuse suggest a shared vulnerability. Understanding the nature in which delay discounting, financial mismanagement, and addictive behaviors contribute to one another may lead to the development of more effective interventions that target all three behaviors.

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