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Intertemporal Decision Making After Brain Injury: Amount-Dependent Steeper Discounting after Frontal Cortex Damage

Abstract: Traumatic brain injuries to the frontal lobes are associated with many maladaptive forms of behavior. We investigated the association between brain damage and impulsivity, as measured by the rate of delay discounting (i.e., the extent to which future outcomes are devalued in time). The main aim of this study was to test the hypothesis of steeper discounting of different amounts in a group of patients with frontal lobe damage. We used a delay discounting task in the form of a structured interview. A total of 117 participants were divided into five groups: three neurological groups and two groups without brain damage. Our analyses showed that patients with focal damage to the frontal lobes demonstrated steeper delay discounting than other participants. Other clinical groups demonstrated similar discounting rates. The data pattern related to the magnitude effect on the group level suggested that the magnitude effect is absent in the group of patients with damage to the frontal lobes; however, results were less consistent on an individual level. Amount-dependent discounting was observed in only two groups, the healthy control group and the neurological group with other cortical areas damaged.

Keywords: impulsivity, brain injury, delay discounting, frontal lobes, magnitude effect

Introduction

From the molar point of view, maladaptive decision making in the intertemporal choice-behavior involves not taking into account future, delayed consequences, and can lead to socially problematic behavior. Patients with frontal lobe damage may show various forms of dysfunctional, problematic behavior, such as lack of planning and deliberation before taking action, impatience, and engaging in risky behavior and antisocial behavior (Sener, Ozcan, Sahingoz, & Ogul, 2015; Szczepanski & Knight, 2014). Impulsivity is an important characteristic of people with frontal lobe disorder. In psychology and the behavioral sciences, impulsivity may reflect different characteristics and is a multi-faceted construct (Bauman & Odum, 2012; Mitchell, 1999).

In our approach, we have defined impulsivity as a choice in which future consequences, such as rewards, fail

to influence present decisions. Specifically, it is a choice of the smaller sooner reward over the larger later one. One of the proposed mechanisms of the impulsive behavior is delay discounting, which is the process by which delayed outcomes are devalued. Steep discounting is a tendency to devalue future rewards at a higher rate. Consequently, if future consequences have only little subjective value, a person is more prone to choose immediate options. In that sense, the person is more likely to make impulsive choices (e.g., Logue, 1988). Some brain regions have been found to be related to more impulsive choices. For example, orbitofrontal cortex damage in humans has been found to lead to increased impulsivity (Bechara, Tranel, & Damasio, 2000; Berlin, Rolls, & Kischka, 2004). Here, we present a clinical investigation of delay discounting of hypothetical monetary rewards of different amounts in groups of patients with different types of focal brain damage, with special interest in patients with frontal lobe damage.

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Delay discounting is a decrease in the subjective value of the reward with the increase in the discounting factor. The longer the delay to the reward, the smaller subjective value it has. That is why people generally prefer instant, positively valued outcomes to the same rewards when delayed. This devaluation of consequences can sometimes be adaptive, but can also form a pathology. Impulsively choosing PLN 100 now over PLN 200 in 30 years from now would be a better choice, while choosing PLN 100 now over PLN 200 in one hour would be regarded in many situations as inferior. Impulsivity, as measured by delay discounting, is linked to various forms of behavior, indicating some form of reinforcement pathology (Bickel, Johnson, Koffarnus, McKillop, & Murphy, 2014). Such behavioral constellation may be present in substance abuse (Yi, Mitchell, & Bickel, 2010), compulsive gambling (Reynolds, 2006), risky sexual behaviors, drug abuse (Johnson, Johnson, Herrmann, & Sweeney, 2015), resulting in poorer outcomes of treatment for substance dependence (e.g., Sheffer et al., 2012; Stanger et al., 2012), and increased risk of relapse (e.g. Stevens, Verdejo-García, Roeyers, Goudriaan, & Vanderplasschen, 2015).

One of the common effects of delay discounting is the magnitude effect, which means that there is an inverse relationship between the amount of the reward and the discounting rate (Green, Myerson, Oliviera, & Chang, 2013; Green, Myerson, & Ostaszewski, 1999; Thaler, 1981). In other words, a large reward will lose proportionally less with delay than a smaller one. The magnitude effect is a widely studied behavioral effect that contradicts the standard microeconomic assumptions. An understanding of this effect may provide more insight into the nature of impulsivity. In a recent study, Mellis et al. (2017) found that the differences between high-risk substance users and controls depended on the magnitude of the reward. In a smaller range of rewards, there were no differences between groups. However, the differences were more pronounced as the magnitude of the delayed reward increased.

Focal brain damage as a result of stroke, craniocerebral trauma, hyperplastic lesions, poisoning or hemodynamic changes, may affect the course of many forms of behavior. This also applies to the processes of discounting, which can provide conceptual space for impulsive behavior (Dixon et al., 2005; McHugh & Wood, 2008; Sellitto, Ciaramelli & di Pellegrino, 2010; Peters & D'Esposito, 2016). The first empirical attempts to relate brain damage to impulsivity investigated the overall effect of the brain injury itself, regardless of specific localization. A study carried out by Dixon and colleagues (2005) highlighted a few important factors, which we also acknowledged in our study. In the first experiment, they showed that discounting in patients with acquired brain damage can be very unsystematic when the payoff is too large (it was \$1000). The second experiment concluded that not every patient with brain injury discounts delayed rewards more steeply than controls. The authors further pointed out the importance of studying the specific localization of neurological deficits in the cortex. A similar study was conducted by McHugh and Wood (2008), which showed that patients with traumatic brain injury were more impulsive than controls.

Heterogeneity of treatment groups, i.e. diversity in terms of the location of brain damage, in the context of delay discounting, was included in the studies by Sellitto et al. (2010). It was already known that the medial orbitofrontal cortex is activated when choosing between delayed rewards (Bickel et al., 2007; Yarkoni, Braver, Gray, & Green, 2005). However, the role of the medial orbitofrontal cortex during intertemporal choices remained unclear. Further studies focused on focal damage of this region and demonstrated that it is associated with increased preference toward immediacy (Sellitto and al., 2010; Peters and D'Esposito, 2016).

Another line of research, based on neuroimaging, confirmed the importance of frontal lobes in intertemporal decision making. For example, regions of the lateral prefrontal cortex along with posterior parietal cortex engage in any choice when the delay is involved (McClure, Laibson, Loewensten, & Cohen, 2004). Furthermore, parts of the ventromedial prefrontal cortex, posterior cingulate cortex, and bilateral parietal cortex may be activated proportionally to the subjective value of the delayed reward (Massar, Libendinsky, Weiyan, Huettel, & Chee, 2015). In other studies, the negative correlation between the rate of discounting and gray matter volume in the insula area in the left hemisphere and the orbitofrontal cortex in the right hemisphere, and positive correlation between the gray matter of the frontal pole in the left hemisphere was observed (Mohammadi et al., 2016). This leads to the conclusion that the greater the volume in the insula area of the left hemisphere and the orbitofrontal cortex of the right hemisphere, the more steeply discounted the rewards. On the other hand, the greater the volume in the gray matter of the frontal pole of the left hemisphere, the shallower the discounting rate.

The observed impulsivity in patients with focal brain injuries, especially located in the frontal cortex, might be an effect of different discounting characteristics in this group. We hypothesize that patients with frontal lobe damage, who are described in clinical practice as impulsive, may discount delayed rewards faster than patients with other damaged cortical areas.

Thus, although there is a vast amount of well-known research on the role of frontal lobes in regulating behavior, we observed that, with few exceptions (Sellitto, Ciaramelli, & Pellegrino, 2010; Peters & D'Esposito, 2016), there is a lack of clinical studies investigating amount-dependent delay discounting in clinical and non-clinical settings among healthy and hospitalized participants with or without brain damage.

In our study, we formulated two main directional hypotheses that are theory-driven and based on robust findings in the fields of decision making, neuropsychology, and neuroscience. We predicted that patients with damage to the frontal cortex would discount the delayed rewards more steeply than all other groups. We also predicted that in all groups there would be a magnitude effect, meaning



that small rewards would be discounted steeper than large rewards. Therefore, the present study had two main aims: 1) to investigate whether patients with frontal lobe damage discounted delayed rewards at a higher rate than control groups; 2) to determine whether the magnitude effect depends on the clinical characteristics which imply specific localization of brain damage.

Method

The main aim of this study was to focus on the intertemporal choice of rewards of different amounts in patients with frontal lobe damage. To validate our hypotheses, we employed a design with control groups. All groups other than the frontal lobe damage group served as controls for this single experimental group. Two groups without neurological disorders or damage (one healthy and one clinical) were used to rule out the possible confounder of being hospitalized or having cortical damage. We also included two additional clinical groups with cortexes damaged in areas other than the frontal lobes. One with cortical damage in the insula or in another lobe (parietal, temporal and occipital), and the other with mixed cortical damage (at least two lobes) or damaged deep subcortical brain structures. Inclusion of these groups provided us with the evidence that possible changes in discounting rates are specifically due to damage to the frontal lobes, not hospitalization or any other cortical damage.

Participants

One hundred seventeen participants (61 males; 56 females) ranging in age from 22 to 80 years were included in the study. All participants provided signed informed consent prior to participation in the study. The experimental protocol was approved by the local ethics committee. Participants were divided into five groups (three neurological and two control). The neurological groups were comprised of patients undergoing neurological rehabilitation with ischemic or hemorrhagic etiology and patients with craniocerebral damage. For clarity, throughout the text, we use abbreviations to indicate a given group. The first group consisted of 16 patients with frontal lobe damage (PFC, participants with frontal cortex damage); the second group consisted of 35 patients with mixed focal damage or subcortical structure damage (PSS, patients with subcortical structure damage); the third group consisted of 18 patients with damage to other structures (POC, patients with other cortical areas damaged), such as the insula, temporal lobes, parietal lobes, or occipital lobes. The two control groups were the clinical control group (n = 26); CCG) and the healthy control group (n = 22; HCG). The healthy control group was comprised of participants who were not patients of any healthcare facility and agreed to participate in the study. The clinical control group consisted of patients without brain damage who were hospitalized (for at least a month) in the same healthcare facility and diagnosed with gonarthrosis, coxarthrosis, or degenerative changes of the spine. Basic demographic measures along with hemispheric lateralization of cortex damage for neurological groups are presented in Table 1.

Table 1. Characteristics of five groups included in the study

Group	Sex (n Males, n Females)	Age (Mean ± SD)	Localization of cortical damage (LH; RH)*
PFC	7; 9	62 ± 11.7	8; 8
POC	9; 9	64 ± 14.3	13; 5
PSS	19; 16	62 ± 12.9	16; 19
CCG	14; 12	63 ± 13.3	_
HCG	12; 10	62 ± 9.0	_

^{*} LH – left hemisphere; RH – right hemisphere.

The subjects with impaired speech due to aphasia had preserved speech understanding of at least 80%. This criterion was determined on the basis of standard procedures used in the healthcare facility for diagnosing speech disorders (trials from the Boston Diagnostic Aphasia Examination test (Goodglass & Kaplan, 1983). All patients were also tested on cognitive behavioral deficiencies according annex (6b) to President's of the National Health Fund ordinance number 53/2010/DSOZ. The time from the neurological incident was no more than two years. Patients were without diagnosed psychiatric disorder or addiction, which was assessed based on the medical history.

Procedure and materials

After obtaining the informed consent, the experimenter gathered basic sociodemographic information from participants and the cognitive behavioral data from the patient's medical history before the onset of the main procedure. The main procedure aimed to measure the delay discounting rate of small (PLN 200) and large (PLN 2000) rewards by estimating the values of indifference points. The amount of these hypothetical monetary rewards was a within-subject factor for which the presentation was random (randomly precoded before data collection) across all participants. The rewards in each of the amount conditions were delayed by three days, one week, one month, three months, six months, and one year, yielding twelve indifference points for each participant (six for each amount) overall. Delays were also randomly presented within each amount condition. To estimate each indifference point, the experimenter asked six questions, and the participant had to state his preferences. The first question always reffered to a choice between half of the nominal amount, of a delayed amount, and of the full, undiscounted delayed reward. For example: "What do you choose, PLN 100 now or PLN 200 in one year?" Each next question was based on the participant's previous choice. If the delayed option was chosen, the immediate value was increased. If the immediate option was preferred, then



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its value in the subsequent step was decreased to make it less attractive. For the PLN 200 condition, the immediate value changed in the subsequent choices by 50, 25, 13, 6, 2, and 2 (all values are in PLN). This means that when the first choice was made, the immediate value increased or decreased by PLN 50, and after the 2nd choice the value changed by PLN 25. In the large reward condition, the immediate reward changed by the same values, but multiplied by 10 (because the larger reward was 10 times larger than the smaller reward). This algorithm of changes was based on standard procedures (for example, see Du, Green, & Myerson, 2002). The difference between these similar procedures was that our approach relied on a structured interview with predefined questions rather than a paper questionnaire or computer program. Such approach was employed because neurological patients had deficits as hemiplegias, hemiparesis, or hemispatial neglect, which may impair marking options in the questionnaire or their perception.

Data analysis and measures

We used the area under indifference points as a measure of the discounting rate, primarily in order to avoid making an a priori decision to choose a certain theoretical approach, and to preserve model-free testing of our main hypotheses. Instead of using a traditional approach to area under the curve (AUC) calculations, we followed the guidelines provided by Borges et al. (2016) and computed AUC_{logD}. We used this new alternative approach to override possible weaknesses of the original approach. Unlike standard AUC, the AUC_{logD} incorporates the subjective experience of time using the logarithmic transformation, and furthermore corrects for unequal contributions of each indifference point to the computed discounting rate (for more in depth explanation, see Borges et al., 2016). It is important to note that the interpretation of this new measure remains the same as the standard AUC (i.e. the smaller the value, the steeper the discounting), indicating that the smaller the area under indifference points, the greater the delay discounts value from the nominal delayed amount. For main analyses, we used mixed analysis of variance (ANOVA). Although, we are aware of the unequal sample sizes, this assumption is of a real concern only when there are large inequalities between factors, which is not the case in

Because of the strong support of this theory and empirical evidence, in the main analyses of this study we tested directional hypotheses, so reported statistical values of multiple comparisons are one-tailed. This was a valid approach in this case because all hypotheses were directional and theory-driven (Jones, 1952, 1954; Kimmel, 1957). Furthermore, it is a clinical study with a rather small sample size per group, and therefore, we used a liberal approach to testing multiple comparisons, but these were always protected (i.e., the main omnibus test (ANOVA) was significant). This liberal approach, however, did not affect conclusions based on simple effects relating the magnitude effect of different groups because the factor had two

levels. Similarly to some studies on discounting (see: Yi & Landes, 2012), we report unadjusted p values for multiple comparisons, leaving such possibility to a reader or applying preferred adjustment (Bailar & Mosteller, 1988). In preliminary analyses, we used a two-tailed hypothesis testing paradigm with $\alpha = .05$.

Results

We divided all analyses into two main parts. Firstly, we focused on preliminary analyses which served to address possible confounding variables in further analyses. Secondly, the main analysis compared the delay discounting rates among five groups.

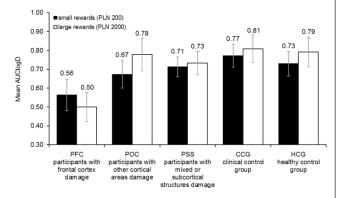
Preliminary analyses

To ensure that there were no confounding variables related to group composition, we ensured that there were no differences across groups in sociodemographic variables. We report these results in brief because of their secondary nature. We found that there were no significant differences among the five compared groups in gender ($\chi^2(4; n=117) = 0.630; p=.960; \chi^2 \text{ test}$), age $(F(4;112) = 0.125; p = .973; \eta_p^2 = .004; between-subject one$ way ANOVA), and years of education (F(4;112) = 1.558; p = .191; $\eta_p^2 = .053$; between-subject one way ANOVA). Especially the lack of differences in age composition of the groups is important, because some studies show that discounting rate changes with age (Green, Myerson, Rosen & Fry, 1996). Furthermore, there were no significant differences in degree of neuropsychological (both cognitive and behavioral) deficits (all p > .005, as compared with H Kruskal-Wallis test because the scale was ordinal), as measured in accordance with annex to President's of the National Health Fund ordinance number 53/2010/DSOZ (that is in: motor speech, language skills, non-verbal aspects of communication, memory functions, praxis, gnosis visual and auditory gnosis, executive functions, emotion and personality, and phagia). The only difference was in attention measure ($\chi^2(2, N=69) = 7,772$; p = .021; Kruskal-Wallis test). Due to this fact, we performed a set of multiple pairwise comparisons among the neurological groups in regard to the attention variable. We found that the only significant difference was between the PFC and POC (other cortical; U=65.5; p=0.006; r=.50) with the former having a mean rank of 22.41 and the latter having a mean rank of 13.14). This means that the PFC group scored significantly worse on attention measures than the POC group. Closer inspection of the data distribution shows that out of 16 participants in this group, 4 demonstrated no deficiencies, 7 demonstrated light deficiencies, 2 demonstrated moderate deficiencies, and 3 demonstrated heavy deficiencies. Because this was a possible confounder, we checked whether there was a relationship between severity of attention deficiencies and the main dependent measure in our study, AUC_{logD}. We found no evidence of such a relationship in the neurological patients (for small reward, $\rho = -.229$, p = .059; for large reward $\rho = -.123$, p = .315).



In our main analyses, we relied on a model-neutral approach by examining areas under the empirical data points, not the discounting parameters derived from mathematical discounting models. Informal comparison of molar discounting rates, as evidenced by AUC_{logD} presented in Figure 1, among the five groups suggests that patients from the frontal group most steeply discounted rewards, especially in the large amount condition (PLN 2000). Furthermore, the reward amount effect in the frontal group suggests the reverse magnitude effect, and in all other groups it suggests the standard magnitude effect. All means, along with 90% confidence intervals are presented in Figure 1 (we based inferences on directional hypotheses).

Figure 1. Discounting rate (AUClogD) of small and large delayed rewards among the five groups included in the study. The lower the score, the higher the discounting rate, indicating higher impulsivity. Error bars represent 90% confidence intervals.



To address these informal observations, we used a 2x5 mixed factorial ANOVA (Pillai's Trace reported, multivariate approach). The first factor is within-subject and refers to the amount of the reward, and the second factor is between-subject and represents the two control groups and three neurological groups. We found a significant main effect of magnitude of the reward (F(1;112) = 5.02; p = .027; $\eta_p^2 = .043$) and of group membership (F(4;112) = 5.68; p < .001; $\eta_p^2 = .169$). There was also a significant interaction between two factors (F(4;112) = 2.95; p = .023; η_p^2 = .095), which indicates that the magnitude of a reward has different effects depending on the group membership. To further investigate the nature of the interaction, we calculated simple effects. In this case, interaction can be examined from two perspectives. Firstly, it can be examined by looking at the presence and direction of the magnitude effect in different groups. The effect of the amount on the delay discounting rate was present in two groups, POC (p<.001) and HCG (p=.03). This effect was unidirectional and was in line with our hypotheses. The impact of the amount on the discounting rate requires further clarification in relation to the PFC group. As Figure 1 shows, in the PFC group, the large rewards were discounted at a steeper rate, pointing to a possible reverse magnitude effect. Although this difference was significant (p = .05 i.e., 0.047), due to unidirectional hypotheses, it should be treated as meaningless from the analytical point of view because the directional hypothesis referred to the other direction of the effect. In the POC and HCG groups, we observed the magnitude effect, indicating that larger rewards were discounted less steeply than smaller rewards. In the remaining two groups, there was no effect of the amount of the discounting rate (PSS, p=.21; CCG, p=.10).

Secondly, we examined the interaction by looking at the differences among compared groups separately in the conditions of small and large rewards from the perspective of PFC. In the small reward discounting condition, we noted highly significant differences between the PFC group and the other three groups, PSS, CCG, and HCG (all at p < .001). The difference between PFC and POC was insignificant, but showed a trend toward significance (p = .06). The pattern of differences was more evident in the large reward condition. The frontal group discounted large reward more steeply than all other groups ($p \le .001$).

By evaluating these comparisons in conjunction with the interaction effect, the analyses revealed differences in the discounting rate between the frontal group and other groups in both the small and large amount conditions. By performing multiple comparisons, we showed that this effect was not a by-product of the brain injury itself (as compared to other clinical groups with brain damage) nor of hospitalization (comparison with both control groups). Specifically, participants with frontal lobe injuries differed from all other groups in the perception of a large reward, being less sensitive to amount.

Discussion

The clinical characteristics of patient's behavior as linked to cerebral cortex damage are of great importance because of diagnostic value and the rehabilitation process. In our study, we demonstrated that there is an interaction between the magnitude of reward and group membership on the rate of delay discounting. The patients with frontal cortex damage showed steeper discounting, especially when comparing the large reward rate of discounting to that of other groups. Furthermore, we observed no magnitude effect in three of the groups: patients with frontal lobe damage, those with deep subcortical structure or mixed focal damage, and clinical controls, which may indicate different processing of reward's amount.

We did not note the magnitude effect in all groups. However, we may find some support of the possibility in different processing of reward magnitudes in the group of patients with frontal lobe damage. As shown by research on animal models (Cardinal, 2006; Cardinal, Winstanley, Robbins, & Everitt, 2004) and neuroimaging studies on healthy participants (Ballard & Knutson, 2009), damage to the anterior cingulate cortex in patients with frontal lobe disorder might result in the lack of differences in the rate of discounting of small and large rewards.

Damage to the frontal lobes is not the only cause of frontal lobe syndrome. The lack of a link between the frontal lobe and other cortical structures may also result in similar symptoms. The neuropsychological literature



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shows that multiple problems arise during rehabilitation and in everyday situations (Kennerley & Walton, 2011; Szczepanski & Knight, 2014). The patient's cooperation with other people may be problematic. This applies not only to family and relatives but, more importantly, to doctors and the therapeutic team, leading to difficulties with rehabilitation. Substantial progress in rehabilitation may not be achieved because the services are hard to deliver, and therefore the rehabilitation is ineffective. Our research shows that the level of impulsivity might be a problem in patients with frontal lobe damage. Also, the lack of strong evidence for different rates of discounting between the POC and PFC groups (only statistical trend) might be due to involvement of insula damage in the POC group. The anterior insular cortex (AI) has been reported to be causally related to impulsive-compulsive decision making in rats (Belin-Rauscent et al., 2016). This is supported by results from human studies and the involvement of insula areas in decision making (Mohammadi et al., 2016). Specifically, damage to the AI, an important part of the cortical salience network (Menon & Uddin, 2010), may result in a pattern of behavior lacking discrimination between rewards of smaller and larger importance and consequently, not taking action aimed at obtaining a more valuable reward.

Although, we formulated a directional hypothesis regarding the effect of amount on discounting, we actually observed a reverse pattern. While such differences and results are quite meaningless in the light of supporting data and hypotheses, we would like to speculate about the reverse magnitude effect in delay discounting. We stress once again, that the assumed a priori logic of our analyses showed that there was no difference between discounting rates of different amounts in the PFC group. Nevertheless, such effect requires further investigation. One possibility is that in extremely impulsive populations, what really is important is having the given amount immediately. Such view should be combined with a demand for reinforcement. We can imagine that when the marginal utility curve for certain reinforcement flattens out, there is little difference in utility with one more unit of a given good. The same can happen in the situation of high impulsivity, and we may obtain a ceiling effect. In other words, assuming that all the person needs is PLN 100 in a given situation, changing the amount to PLN 110 would not, or would only marginally, change the real utility. In such cases, PLN 100 is 50% of the larger reward of PLN 200, but is only 5% of the award of PLN 2000. This pattern of results points to the reverse magnitude effect in delay discounting. As suggested by Green et al. (2004) in relation to animal studies, the lack of the magnitude effect can result in the discounting rate being affected by amounts up to a certain point, and this specific point can lie lower for more impulsive populations (see also, Green, Myerson, Oliviera, & Chang, 2013). This result is also supported by research showing a ceiling effect in discounting when the rewards are of a small amount (Mellis, Woodford, Stein & Bickel, 2017).

We failed to demonstrate the usual magnitude effect in all groups, and we discussed a rationale for this result in the PFC group. It maybe also possible that some other

cortical or subcortical structures are involved in the reward magnitude processing because we did not observe a reliable magnitude effect in the PSS group, in which damage was extensive in multiple areas. We find a limitation in present results because of the lack of the magnitude effect in the POC group. However, as discussed earlier, this group included patients with anterior insula damage. According to Menon and Uddin (2010), this structure forms a salience network, which is supposed to "segregate the most relevant among internal and extrapersonal stimuli in order to guide behavior" (Menon & Uddin, 2010, p. 655). Therefore, it is possible that the injury makes it difficult to distinguish between rewards of larger and smaller values. On the other hand, the clinical control group should have maintained the usual encoding of reward magnitude. The only explanation found for the lack of the magnitude effect in the clinical control group, is the possibility that the relatively small difference between discounted amounts could contribute to two things: a relatively small magnitude effect in groups, and a different trend in patients with frontal lobe damage. We used such amounts to make them as meaningful and easy to imagine to participants as possible, and based our approach on the study by Dixon et al. (2005) that showed that when the reward is too large, patients with brain damage, may discount it in a more unsystematic way. Also, according to Sellitto et al., 2010 it is worthy to test delay discounting rate in patients with ventromedial orbitofrontal cortex (vmOFC) damage to obtain more insight to the neural mechanism of discounting. Including such localization could possibly account for even stronger discounting and presence of lack of magnitude-related effects.

Although, we showed that the PFC group discounted delayed rewards at the highest rate, not every case of focal brain damage in this area has to led to impulsive behavior. Mar et al. (2011) showed that lesions of different subregions of the orbitofrontal cortex manifest in dissociable effects in impulsive choice in rats. Lesions to the medial subregions of the orbitofrontal cortex resulted in decreasing impulsive choices, whereas lateral subregions of orbitofrontal cortex resulted in increased impulsive choice as compared to sham controls. From this context, we may predict that not every type of brain damage leads to impulsive choice, and not every case of frontal cortex damage results in an increased preference for smaller, sooner rewards. Further research in clinical settings may extend the presented study to take into account not only heterogeneity of brain damage referring to different lobes, but also the within-lobe heterogeneity. Another aspect that might be interesting to include in future studies is other types of reward devaluation, for example effort discounting (Klein-Flügge, Kennerley, Saraiva, Penny, & Bestmann, 2015; Mitchell, 2004; Nishiyama, 2016; Ostaszewski, Babel & Swebodziński, 2013). It has been demonstrated that delay and effort discounting share areas that are involved in reward value encoding, but they simultaneously utilize other cortical areas to derive subjective value (Massar et al., 2015). Similarly, Peters and Büchel (2009) showed parallel results when participants made choices during delay and probability discounting. Again, some

neural systems overlapped in coding subjective value, while at the same time having separate, unique areas involved in the decision making. Because of the overlapping nature of these constructs (see: Białaszek, Gaik, McGoun, & Zielonka, 2015; Mitchell, 2004), treatment involving one kind of discounting could also spread also to other forms of impulsive decision making. However, this hypothesis remains untested.

The main novelty of our study is the inclusion of four groups other than the group with frontal cortex damage when assessing the level of delay discounting. The design of our study also allowed us to compare the discounting rate and the presence of magnitude effect not only among patients with frontal lobe damage and healthy control group, but also in relation to groups with other cortical and subcortical damage and controlling for the hospitalization. We demonstrated that patients with frontal lobe damage behave impulsively, choosing smaller and sooner rewards, regardless of their magnitude. The lack of the magnitude effect in this group provides also some practical information for ongoing rehabilitation. It suggests that choice is insensitive to reward magnitude, and therefore, in rehabilitation, using larger, delayed rewards in this group of patients to direct behavior to larger, delayed outcomes cannot be justified. Moreover, from the perspective of impulsive behaviors, it should be acknowledged, that not all brain injury is linked to higher impulsivity and is localization specific.

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