An fMRI-based brain marker predicts individual differences in delay discounting

Leonie Koban (✉ leonie.koban@gmail.com)

Paris Brain Institute (ICM), CNRS UMR7225 <https://orcid.org/0000-0002-3121-6491>

Sangil Lee

University of Pennsylvania <https://orcid.org/0000-0002-4443-9926>

Daniela Schelski

University of Bonn

Marie-Christine Simon

University of Bonn

Caryn Lerman

University of Southern California

Bernd Weber

University of Bonn

Joseph Kable

University of Pennsylvania <https://orcid.org/0000-0001-6495-9040>

Hilke Plassmann

INSEAD

Article

Keywords: fMRI, brain marker, delay discounting

Posted Date: August 11th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-538708/v1>

License: This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

An fMRI-based brain marker predicts individual differences in delay discounting

Leonie Koban^{1,2,*}

Sangil Lee³

Daniela S. Schelski^{4,5}

Marie-Christine Simon⁶

Caryn Lerman⁷

Bernd Weber^{4,5}

Joseph W. Kable³

Hilke Plassmann^{1,2}

¹ Marketing Area, INSEAD, Fontainebleau, France

² Control-Interoception-Control Team, Paris Brain Institute (ICM), INSERM U 1127, CNRS UMR 7225, Sorbonne University, Paris, France

³ Department of Psychology, University of Pennsylvania, Philadelphia, PA, USA

⁴ Center for Economics and Neuroscience, University of Bonn, Bonn, Germany

⁵ Institute of Experimental Epileptology and Cognition Research, University of Bonn, Germany

⁶ Institute for Nutrition Science, University of Bonn, Bonn, Germany

⁷ Norris Comprehensive Cancer Center, University of Southern California Los Angeles, CA, USA

Running Head: A BRAIN SIGNATURE OF IMPATIENCE

*Please address correspondence to:

Leonie Koban, PhD
Control-Interoception-Attention Team
Institut du Cerveau (ICM)
Hôpital Pitié Salpêtrière
47, bd de l'hôpital
75013 PARIS - France

Email: leonie.koban@icm-institute.org
Telephone: +33 (0)6 36 44 56 90

Abstract

Individual differences in impatience—how much we discount future compared to immediate rewards—are associated with general life outcomes and related to substance use, psychiatric diseases, and obesity. Here, we use machine-learning on fMRI activity during an intertemporal choice task to develop a brain marker of individual differences in delay discounting. Study 1 (N=110) was used as a training and cross-validation set, resulting in significant prediction accuracy ($r = 0.49$) and suggesting an interplay between brain regions associated with affect, value, and cognitive control. The validity of the brain marker was replicated in an independent data set (Study 2, N=145, $r = 0.45$). In both studies, responses of the marker significantly differed between overweight and lean individuals. This pattern is a first step towards a generalizable neuromarker of delay discounting and a potentially transdiagnostic phenotype, which can be used as a brain-based target measure in future studies.

Author note

We received funding from the ANR (Tremplin-ERC grant “Brain Gut Decision” to HP), Campus France (Marie-Sklodowska-Curie co-fund fellowship PRESTIGE-2018-2-0023 to LK), French federal funding (program ‘*Investissements d’avenir*’, ANR-10-IAIHU-06), the Federal Ministry of Education and Research Germany (Diet-Body-Brain grant 01EA1809B to BW), the National Cancer Institute (R35CA197461[C.L.] and R01CA170297 [J.K and C.L]). We thank P. Trautner for help with the programming of the task (Study 1), A. Simonetti, M. Boerth, A. Koehlmoos, S. Winkler, L. Bernardo, A. M. Burke, M. K. Caulfield, N. Cooper, G. Donnay, M. Falcone, J. Jorgensen, R. Kazinka, J. Luery, M. McConnell, Rickie Miglin, D. Mukherjee, T. Parthasarathi, S. Price, M. Schlusssel, R. Sharp, H. J. Sohn, D. Spence, and K. Terilli for help with data acquisition and T. D. Wager for the Canlab toolbox and helpful discussion.

Author contributions

LK and HP conceptualized the study. DS acquired the data for Study 1, JK and SL contributed and re-analyzed existing data (Study 2). LK analyzed and visualized the data. LK wrote the first draft of the manuscript, with revisions from HP. HP, JK, CL, MCS, and BW obtained funding, provided resources, and supervised the project. All authors edited and approved the manuscript.

Introduction

Many decisions in daily life require choices that have consequences at different points in time. For example, most people need to decide at the end of the month whether to put part of their paycheck towards a retirement fund or directly spend it on something fun like a short vacation. Those trade-offs between choosing an option that is immediately rewarding versus one that will be more rewarding in the long run are hard and people differ substantially in their ability to delay immediate gratification. Both intuition and accumulating evidence suggests that *patience for rewards*—the ability to make choices that are aligned with long-time goals and to resist immediate gratification—is closely related to other advantageous behaviors, better health, and overall life achievement ^{1,2}.

In economics, patience for rewards is typically measured as *delay discounting* (DD), which quantifies how much people discount future compared to immediate rewards ³⁻⁵. Most people prefer sooner rather than later rewards (e.g., almost everybody would prefer 100 Euro right now over 101 Euro in 2 years). Yet, the degree to which delayed rewards are discounted varies widely between individuals. Greater delay discounting (i.e., greater impatience) is associated with smoking, obesity and overweight ⁶⁻¹⁰. Abnormally high discounting has also been found in a number of psychiatric conditions, including depression, schizophrenia, and bipolar disorder ¹¹. Delay discounting has therefore been proposed as a potential transdiagnostic marker of psychopathology ^{11,12} and as a risk factor for many health conditions that are largely driven by short-sighted behaviors such as unhealthy diet, smoking, alcohol and drug use ¹³⁻¹⁶. The goal of the present study is to identify and validate a predictive fMRI-based brain marker of individual differences in impatience as measured by delay discounting.

A growing number of neuroimaging studies has begun to explore the neurophysiological correlates of individual differences in delay discounting, based on both structural and functional brain measures ¹⁷⁻²², for a review see ²³. Several of these studies suggest a role of prefrontal areas, including those involved in reward processing and valuation (especially ventromedial prefrontal cortex/vmPFC and the ventral striatum/VS) ^{20,24}, and those central for cognitive control (especially dorsolateral prefrontal cortex/dlPFC) ²⁵. Brain areas associated with memory and prospection, especially hippocampus and entorhinal cortex, are found to contribute to individual differences in delay discounting as well ^{22,26-28}. This is in line with findings from psychology and behavioral economics that show that subjective time perception ²⁹ and the ability to imagine future events ²⁸ and one's future self ²¹ in rich detail are contributing to patience

and intertemporal decision-making. Several other studies have focused on structural and functional connectivity measures, demonstrating that fiber tract strengths between frontal and striatal areas are associated with decreased discounting, whereas strength of striatal-subcortical connections are associated with increased discounting¹⁹. The structure of midbrain dopaminergic nuclei and the ventral striatum has also been associated with self-reported trait impulsivity³⁰. Thus, previous findings regarding the brain bases of individual differences in impatience and impulsivity offer a mixed picture: different regions seem to be involved and findings are not always replicated across studies.

Given the complexity of intertemporal decisions, it is unlikely that individual differences in delay discounting can be explained by one brain region or one functional network. Instead, they likely result from an interplay between different functional networks, including those associated with the presumed underlying functional processes of valuation, cognitive control, and prospection^{12,27}. However, surprisingly few studies have investigated multivariate or distributed patterns predicting individual differences in delay discounting^{17,31,32}. Further, most previous studies used relatively small sample sizes for exploring individual differences, increasing the risk of both false-positive and false-negative results³³. A further limitation of previous findings is that they are often based on correlation or regression models that are not cross-validated on independent (hold-out) data samples³⁰. Thus, such models run the risk of overfitting the model to the data, thereby again increasing the risk of false-positive findings that cannot be replicated in independent data sets.

In this paper, we address these limitations by using a machine-learning-based 'brain model' approach^{34,35} in a larger data sample (total N=255). Brain models or 'brain signatures' are patterns of activity across the whole brain that are trained to predict a given mental process (e.g., in the case here, impatience as measured by delay discounting) and that can be applied to new, independent data to test their validity and generalizability^{33,34,36}. As such, these models provide a novel approach to analyze fMRI data that goes beyond reporting peak coordinates by providing specific models that can be replicated, validated, or falsified in an easy and quantifiable way. This approach has been successfully applied to brain-based prediction of pain^{35,37}, working memory³⁸, affective states³⁹, and physiological measures like heart rate and blood pressure^{40,41}, among others.

Here, we build on this approach to predict individual differences in delay discounting. We used an established machine-learning algorithm^{42,43} on fMRI data from

two independent studies, from different scanners, labs, and countries. Study 1 (acquired at Bonn University, N=110) was used for training and cross-validation of a predictive model of individual differences in delay-discounting. Baseline data from Study 2 (University of Pennsylvania, N=145, ⁴⁴) was used as an independent test dataset to test the validity and replicability of the predictive model. If there is a consistent pattern of functional activity across the whole brain associated with individual differences in delay discounting during intertemporal discounting tasks, then this pattern should be able to predict delay discounting in new data (hold-out subjects) and even completely independent datasets. Further, comparing the resulting pattern to specific meta-analysis-based masks allows us to assess the contribution of brain areas associated with valuation and affect, cognitive control, and memory/prospection.

Results

Individual differences in delay discounting

We analyzed behavioral and fMRI data from two independent studies and two different versions of a standard intertemporal choice task (ITC). In Study 1 (collected at Bonn University, N=110 male participants), the ITC task consisted of 108 incentive-compatible choices between smaller sooner (SS) and larger later (LL) monetary rewards. Both SS and LL amounts varied. LL options had delays from 2 to 240 days (see Fig. 1A and Methods for details). Participants' choices allowed us to compute the discounting parameter k . This $\log(k)$ -parameter describes the steepness of discounting as modeled by the hyperbolic discounting function ³. Higher $\log(k)$ parameters reflect steeper discounting and thus greater impatience, and smaller (more negative) values reflect less steep discounting and thus more patient decision-making.

On average, participants chose the SS option in 43.7% of all trials (median = 48.1%) and had a fitted $\log(k)$ parameter of -5.70 (median $\log(k)$ = -5.28, corresponding to a k of 0.0051). Choice behavior was characterized by substantial individual differences, with %SS choices ranging from 5.6% to 88.8%, and $\log(k)$ ranging from -9.90 to -1.36 (see Fig. 1B).

In Study 2 (collected at the University of Pennsylvania, N=145), participants made 120 incentive-compatible choices, in which they were presented varying amounts of LL rewards and delays. Participants had to either accept or reject the LL option, in which case they would receive a fixed SS amount of US\$20 (see Fig. 1A). On average, participants in Study 2 chose the SS option in 57.4% of trials (median = 60.0%) and had an average

fitted $\log(k)$ of -4.08 (median $\log(k) = -3.95$, corresponding to a k of 0.0193). Again, individuals varied substantially in their intertemporal preferences, with %SS ranging from 0.8% to 99.2%, and $\log(k)$ ranging from -7.08 to -2.12 (see Fig. 1B).

Taken together, our data confirm the substantial variability in impatience known from previous work^{17,45}. They show that both data sets had sufficient variability in discounting behavior to investigate the neurofunctional bases of these individual differences.

Significant cross-validated prediction of delay discounting based on fMRI

We applied a LASSO-PCR (least absolute shrinkage and selection operator-principal component regression)^{42,43}—a machine learning algorithm especially suitable for the multidimensionality of brain imaging data⁴³ to the fMRI data of Study 1. More specifically, we trained a multivariate classifier—a pattern of brain activity across the whole brain (gray-matter-masked)—to reliably predict individual differences in $\log(k)$. This ‘ k -marker’ (see Fig. 2A, more details below) was trained based on a feature space that combined individual contrast images for three regressors of a standard univariate GLM first-level model: *Choice* onset, its parametric modulation by *Relative LL Amount*, and its parametric modulation by *Delay* (see Fig. 1C, Methods for details). It captures a combination of functional processes that together determine delay discounting. The predictive accuracy of the classifier (k -marker) was assessed using a 10-fold cross-validation procedure in which each participant’s contrast images were included in nine training folds and held out in a tenth fold to assess the *predicted* $\log(k)$ and thereby the accuracy of the model (see Fig. 1C). This training and cross-validation procedure resulted in a cross-validated prediction-outcome correlation of $r = 0.49$ (permutation test: $p < 0.0002$), a mean squared error of 2.84 (permutation test: $p < 0.0002$), and a mean absolute error for predicted $\log(k)$ of 1.32 (permutation test: $p < 0.0002$, see Fig. 2B and Suppl. Fig. S1).

Validation of the k -marker in an independent test data set (Study 2)

Brain markers of individual differences based on brain images become more meaningful if the results can be applied and validated (i.e., confirmed versus falsified) in different and completely independent datasets³⁴. The validity of the marker should not depend on study-specific parameters like the type of scanner used for data acquisition, preprocessing software, or other aspects of the dataset³⁴⁻³⁶.

We therefore tested whether the k -marker—developed and cross-validated entirely on Study 1—could be applied to a completely independent dataset. The validation dataset (Study 2) was acquired on a different scanner, in a different lab and country, using a different participant sample and different task characteristics, and was preprocessed and analyzed using a different MRI analysis software. Evaluating the performance of the k -marker in Study 2 is therefore an even stronger test of the robustness and replicability of the pattern than cross-validation in Study 1 alone.

For this purpose, we computed the pattern expression of the k -marker using the matrix dot product for each participant's data (contrast images for Choice-onset, parametric modulation for LL-amount and Delay) in Study 2. The resulting predicted $\log(k)$ values were significantly correlated with actual $\log(k)$ values ($r = 0.45$, $p = 1.06 \times 10^{-8}$, mean absolute error of 1.68) demonstrating the replicability of the k -marker in a completely independent dataset.

The training and cross-validation dataset (Study 1) consisted of male participants only, which might limit the validity of the k -marker in females. We therefore assessed the accuracy of the k -marker in Study 2 separately in male and female participants. In male participants ($N=88$), the prediction-outcome correlation was $r = 0.40$ ($p = 1.17 \times 10^{-4}$). In females ($N=57$), the prediction-outcome correlation was $r = 0.55$ ($p = 7.63 \times 10^{-6}$) and thus very comparable, if not superior, to the predictive accuracy in males (see Suppl. Figure S2). This demonstrates that the k -marker (despite being trained on male participants' data only) predicts delay discounting well for both male and female participants.

permutation test results) in Study 1. For Study 2 (independent validation data set), accuracy is assessed as the parametric prediction-outcome correlation. Mean absolute error is provided in the text.

Response of the k -marker differs between lean and overweight participants

Given previous findings of higher delay discounting in overweight and obese people^{8,46}, we next tested whether individual expression of the k -marker was associated with individual differences in body mass and overweight (as measured by the body mass index [BMI] > 25). Interestingly, neither in Study 1 nor in Study 2 was actual $\log(k)$ related to BMI. However, in Study 1, response of the k -marker significantly correlated with both BMI ($r = 0.26$, $p = 0.0054$, Fig. 3A) and percentage of body fat ($r = 0.28$, $p = 0.0047$). The k -marker response in Study 1 also differed between lean (BMI<25) and overweight-to-obese (BMI>25) participants ($t(108) = 2.85$, $p = 0.0052$, Cohen's $d = 0.55$ see Fig. 3B). In Study 2, the correlation between predicted $\log(k)$ and BMI was positive but not significant ($r = 0.09$, $p = 0.28$). However, paralleling the findings in Study 1, overweight-to-obese participants had a significantly higher k -marker response (higher predicted discounting) compared to lean participants ($t(143) = 2.11$, $p = 0.037$, Cohen's $d = 0.35$, see Figure 3B).

Fig. 3. Association of k -marker response with BMI and overweight. **A)** In Study 1 (but not Study 2), k -marker response was positively and significantly correlated with BMI. **B)** In both studies, k -marker response was significantly greater (predicting more delay discounting) in overweight (BMI > 25) compared to lean participants.

Relationship with other person-level variables

In order to delineate the discriminant and convergent validity of the k -marker, we next tested the relationship of the k -marker response to several other trait variables that have been associated with delay discounting. Delay discounting could be related to risk preferences because future rewards may be seen as less certain than immediate rewards⁴⁷, but see conflicting evidence, e.g.,⁴⁸. In Study 1, risk preferences were not significantly associated with k -marker response ($r = -0.05$, $p = 0.58$). However, in Study 2, the response of the k -marker was negatively associated with individual risk preferences (alpha estimates, $r = -0.20$, $p = 0.016$). Delay discounting has also been associated with differences in general intelligence⁴⁹. However, the k -marker response was not associated to a screening measure for intelligence (assessed in Study 2 only, using the Shipley Institute of Living Scale – Second Edition⁵⁰, $r = 0.06$, $p = 0.44$). Delay discounting may also decline with increasing age^{51,52}, but see⁵³. While in Study 1 (which had a relatively large age range) there was no significant correlation between age and k -marker response ($r = -0.01$, $p = 0.90$), higher age was associated with somewhat lower k -marker responses (predictive of less discounting) in Study 2 ($r = -0.17$, $p = 0.043$).

Thresholded activation patterns of the k -marker

Activation patterns across the whole brain gray matter and across all three contrasts are used for prediction and cross-validation. To illustrate this pattern and to identify the areas that contributed the most strongly with positive or negative weights, we used a bootstrapping procedure (5000 bootstrap samples). Bootstrapped weights were thresholded at $q < 0.05$ FDR corrected across the whole weight map of the combined feature space and displayed in Fig. 2A (see Suppl. Tables S3-5 for full results tables).

Our results revealed a distributed network of areas that jointly contributed to individual differences in delay discounting, including the vmPFC, ventral striatum, anterior midcingulate cortex (amCC), hippocampus, frontoparietal and visual areas (see Fig. 2). Predictive activity patterns differed for the processes captured by the three different contrast images. Of note, some regions showed negative weights (i.e., predicted less discounting) for one, but positive weights (i.e., predicting more discounting) for another contrast. More specifically, for the Choice (versus implicit baseline) contrast, activity in the striatum, the anterior insula and lateral prefrontal areas contributed positively to more impatience (i.e., greater discounting), whereas activity in bilateral visual areas, premotor

and motor areas contributed weights towards less impatience (i.e., less discounting). For the parametric modulation by LL Amount, activity in vmPFC, aMCC, posterior cingulate cortex (PCC), precuneus, and frontoparietal areas were contributing positive weights for greater discounting, whereas visual areas and premotor areas contributed mainly negative weights. For the parametric modulation by Delay, positive weights were observed in the most ventral part of the vmPFC (or mOFC), premotor areas, and visual cortex, while negative weights were observed in frontoparietal areas and several regions of the brainstem.

Similarity of k -marker brain patterns to meta-analytic maps and established functional networks

To investigate the similarity of these predictive patterns of the k -marker with functional networks, we next compared them with term-based meta-analytic images⁵⁴ for terms that may contribute to intertemporal decision-making (**Fig. 4**) and canonical resting-state networks⁵⁵ (**Fig. S3**). We thus computed the spatial correlation (Pearson's r) between the k -marker and these specific maps of interest c.f.,⁵⁶. While these correlations are descriptive, they can inform us quantitatively whether and in which direction (positive or negative) previously identified functional networks contribute to individual differences in delay discounting. They can also hint at the functional processes that may underlie the k -marker patterns.

We compared the similarity of the k -marker with meta-analytic maps for 1) affective and value-related, 2) conflict and cognitive-control related, and 3) memory-related processes (see **Fig. 4**). Value- and affect-related maps (especially 'affect' and 'emotion') showed consistent positive correlations (r 's > 0.05) with the Choice-related pattern of the k -marker, in line with the idea that more affect-related activity during intertemporal choices overall leads to more *impatient* decisions (greater discounting). However, stronger engagement of affective and especially 'reward and 'value'-related activity for increasing LL-Amount (and, to a lesser extent, for increasing delays) was associated with less discounting (i.e., more patience). This suggests that greater patience is associated with greater sensitivity of valuation-related signals to the amount of LL rewards.

In contrast to our initial hypothesis, cognitive control related activity was not strongly or even negatively associated with more impatience (greater discounting). Instead, there were *positive* correlations (r 's from 0.05 to 0.19) of the Choice-pattern with meta-analytic maps for the terms 'attention', 'cognitive control', 'conflict', and 'executive'

(more positive weights predicting greater discounting). Further, stronger activation of control-related maps by greater LL-Amount was associated with greater discounting (more impatience), whereas stronger activation of control-related activity for longer delays was associated with less discounting (more patience). Parallel findings were observed for the frontoparietal network, which is tightly associated with cognitive control (see Suppl. Materials and **Fig. S3**). This suggests that control-related activity is not directly and positively related to patience, but that it is engaged in more nuanced ways such as processing amounts and delays.

Finally, we assessed the contribution of brain systems related to memory or prospection. While the term ‘memory’ (which likely also includes many working-memory studies) showed a similar pattern as control-related maps, more specific terms such as ‘episodic memory’, ‘imagery’, or ‘planning’ were not substantially positively or negatively correlated with any of the *k*-marker patterns (*r*’s around 0.05 and smaller).

Discussion

A major goal of neuroscience and psychiatry is to identify brain markers of transdiagnostic processes that may be altered across different diseases or that predispose individuals to such diseases⁵⁷. Impatience, measured by how much people discount future compared to immediate rewards, has been proposed as such a transdiagnostic process across psychopathology, addiction, obesity and eating disorders^{11,12,58}. In this paper, we aimed to advance our understanding of the brain processes that drive variability in impatience by identifying and validating a distributed pattern of functional brain activity that predicts individual differences in delay discounting. We first used a cross-validation procedure to develop a novel brain-marker of delay discounting (*k*-marker) based on whole brain, grey-matter masked fMRI data ($N_1=110$). We then validated the *k*-marker in an independent second fMRI data set ($N_2=145$) sampled in a different cohort with different socio-demographic characteristics, a different fMRI scanner, and employing a different delay discounting task. An important advance of the present study is that it provides a novel ‘brain model’³⁴ of impatient decision-making. As such, this brain model can be further tested, validated, or refined in future studies, including those that test its generalizability and predictive power in clinical populations.

Fig. 4. Spatial similarity of the *k*-marker with meta-analytic maps. In order to quantitatively compare the classifier patterns with theoretically relevant functional networks, we computed the spatial correlation of the *k*-marker patterns with Neurosynth meta-analytic maps⁵⁴ associated with **A) value and affect, B) conflict and cognitive control, and C) memory and prospection**. Meta-analytic maps from each group of terms are overlaid on the left (outline colors matching the outlines of the terms on the right) and can be inspected in greater detail online (www.Neurosynth.org). Note that spatial correlations are purely descriptive, indicating whether activity in any of the shown functional maps is rather positively ($r > 0$) or negatively ($r < 0$) associated with discounting, for each component map of the *k*-marker (Choice, LL Amount, Delay).

Our results also inform the debate regarding the contributions of specific brain regions and functional networks to understand people's ability to delay gratification. Among the brain areas that contributed most strongly with positive and/or negative weights were the vmPFC, striatum, and other regions associated with valuation and reward^{24,59,60}. This finding is in line with previous, univariate findings^{17,20,30}. The present results add to

this emergent picture, by suggesting that overall reward sensitivity is associated with greater discounting, whereas greater sensitivity to the amount of the reward is linked to more patient decision-making.

Significant weights were also found in the frontoparietal control network, which is associated with cognitive control, and has been proposed to implement self-control and far-sighted, patient decision-making^{25,61}. Yet, the present results are surprising as they draw a more complex picture of these areas' contribution to delay discounting, with modulation of these areas by greater LL rewards being positively associated with impatience, and modulation by delay being negatively associated with impatience. Thus, cognitive control-related areas were more recruited for long delays and small LL amounts for low discounters, and shorter delays and larger LL amounts for high discounters. These are the cases in which decisions are most difficult (closer to the indifference point) and therefore require resolution of response conflict⁶²⁻⁶⁵. These findings have at least three important implications for models of delay discounting and self-control in cognitive neuroscience. First, they speak against the idea of a simple dual process account of intertemporal choice and self-control⁶¹, joining previous work that suggested more complex neural processes at play^{25,45,66,67}. Second, it also speaks against the idea that more frontoparietal activity is related to higher individual levels of self-control. Instead, it suggests that *for which choice options* control-related areas are activated is more informative than their overall level of activation. This finding is in line with value-based choice models of self-control⁶⁷ and with recent evidence that high and low discounters differ in how much attention they allocate to amount versus delay information⁶⁸. It also fits with the idea that low discounters may not need 'control' to discount less¹², and that high discounters may employ cognitive control for different types of decisions. Third, our results thereby also suggest a mostly neglected role for conflict detection in inter-temporal choice feeding into cognitive control mechanisms.

Hippocampus and adjacent midtemporal areas have been associated with individual differences in discounting^{22,27,28}, in line with the importance of prospection and self-projection in intertemporal decision-making. While our pattern shows some weights in parahippocampal areas and in occipital areas, these weights seemed to be less strong than those in other brain regions. The *k*-marker patterns were not clearly associated with meta-analysis-based activation maps of episodic memory or prospection. This suggests that, at least in the present data of neurotypical healthy adults, activation in these areas might be less related to individual differences than activation patterns in prefrontal and

parietal ones. Yet, this does not preclude that interactions between hippocampus and prefrontal areas might be important or that damage in these regions may lead to substantial changes in delay discounting. Together, our findings highlight the importance of a distributed activity pattern across the whole brain instead of single regions, confirming the notion that impatience and delay discounting depend on the interactions between different functional processes and networks in the brain.

Previous work has related individual differences in delay discounting with obesity, substance use disorders, and multiple psychiatric diseases^{11,12,27}. In the two samples presented here, $\log(k)$ values based on participants' choices themselves were not significantly associated with BMI or overweight. Important to note, the two studies were not directly designed to include a large range of BMI or a high number of obese participants, which may explain this null effect. However, responses to the k -marker did significantly differ between lean and overweight participants. This finding suggests that this brain marker is reflecting variance in functional neurophysiology that is related to long-term patterns in decision-making and health parameters. It also may imply that the brain marker is more sensitive than the behavioral measures it was trained on to speak to individual differences in delay discounting. We call for future studies to test the k -marker in samples with larger numbers of participants with obesity to further investigate this idea. Future studies could also assess whether the k -marker has prognostic value for weight-related health outcomes such as cardiovascular disease or diabetes and responsiveness to drastic weight loss interventions such as bariatric surgery.

The present study also provides some initial evidence for how specific the k -marker is to delay discounting, by dissociating it from other person-level variables that are often associated with the degree of impatience. For example, responses of the k -marker were not (Study 1) or only moderately (Study 2) associated with risk preferences or participants' age. They also did not correlate with a simple measure of general intelligence (only assessed in Study 2). These results support the interpretation that the k -marker is indeed a measure of discounting, rather than reflecting other, potentially confounded variables. Future work could more closely examine the relationship of the k -marker with other related mental processes, with personality, and with clinical measures of interest.

The purpose of the present study was to investigate the functional brain activation during intertemporal choices that is predictive of individual differences in delay discounting. As a resulting limitation, the k -marker is linked to specific task-related contrasts. It cannot be readily applied to task-independent data such as resting-state fMRI

or structural MRI that might be more readily available in existing clinical or large-scale datasets. Future studies could use a similar machine-learning approach to develop markers of delay discounting based on task-independent brain measures. They could also extend this work by investigating the role of structural and functional connectivity and the potential contribution of structure-function interactions to individual differences in delay discounting.

A strength of the *k*-marker developed in this paper is that it can be applied to and tested in any other fMRI study on monetary delay discounting, for which contrast images for Choice, LL Amount, and Delay can be computed. Future work could also test the validity of the *k*-marker in other types of delay discounting paradigms that involve non-monetary rewards such as food, and to other, similar paradigms such as social discounting^{69,70}.

The classifier (*k*-marker) predicts these individual differences in neurotypical healthy adults across different populations, scanners, and analysis pipelines. Future work could test the generalizability of the *k*-marker in children, adolescents, the elderly, or in clinical populations. Such work could assess whether the *k*-marker is able to predict clinical status and health outcomes in conditions related to abnormal discounting, such as eating disorders, substance use, and other psychiatric disorders^{11,12}.

Methods

Participants

For Study 1, participants were recruited in the context of a longitudinal dietary intervention study at the University of Bonn, Germany (https://osf.io/rj8sw/?view_only=af9cba7f84064e61b29757f768a8d3bf). Due to the nature of this intervention study, we only recruited male participants who further fulfilled the following inclusion criteria: age between 20-60 years, right-handedness, non-smoker, no excessive drug or alcohol use in the past year, no psychiatric or neurological disease, body mass index (BMI) between 20-34, no other chronic illness or medication, following a typical Western diet without dietary restrictions, no MRI exclusion criteria (large tattoos, non-removable piercings, metal in the body, claustrophobia, etc.). N=116 male participants performed the ITC task in Study 1. Data reported here was collected during a baseline session before the group assignment and dietary intervention (to be reported elsewhere). The data of 6 participants had to be excluded for analysis due to following

reasons: technical problems with the scanner (1), with the synchronization between stimulation software and scanner (3), with the response box (1), and due to strong motion artefacts (>5mm) and participant quitting the task mid-scan (1). Therefore, 110 participants (mean age = 31.7y) were included in the final analysis of Study 1.

Data from Study 2 was conducted in the context of a large cognitive training study at the University of Pennsylvania, PA, USA (Kable et al., 2017). The goal was to examine if commercial cognitive training software leads to significant changes in decision-making behaviors including delay discounting. Participants completed two sessions of scans 10 weeks apart. Here we only include the baseline (pre-intervention) data. Of the 160 non-pilot participants that completed session 1, we excluded those with missing runs ($n = 6$), frequent or significant head movement (any run with >5% of mean image displacements greater than 0.5mm; $n = 3$), more than 3 missing trials per run for two or more runs ($n = 2$), or lack of participant blinding ($n = 1$, one subject expressed awareness of their experimental condition, i.e. cognitive training vs. control). Of the remaining 148, we excluded three more participants whose choice was entirely one-sided (i.e., choosing only immediate reward or delayed reward), resulting in a final sample of $N = 145$ participants for Study 2 (88 male, 57 female, mean age = 24.4y).

All participants were paid for their participation in the study. The study protocols were approved by the IRB of Bonn University's Medical School (Study 1) and the University of Pennsylvania (Study 2).

Stimuli and task

In Study 1, participants performed 108 choices (trials) between varying amounts of smaller sooner (SS) and larger later (LL) options, presented on the left or right of the screen (position randomized, see Fig 1A). Participants were instructed that one of their choices might be paid out at the end of the experiment. Thus, participants' choices were non-hypothetical and incentive-compatible. During each trial, the two options were presented for 4s, during which participants could make their choice (left or right) by pressing the corresponding response key with their left or right index finger, respectively. Once the choice has been made, a yellow frame highlighted the chosen option and remained on the screen for the remainder of the 4s. Inter trial intervals were jittered using an approximately geometric distribution (2-11s)

SS options varied between 5, 10, and 20 Euro, and always had zero delay ('today'). LL options varied between 5 and 96.80 Euro and had delays between 2 days and 8 months

(~240 days). Amounts and delays were chosen in order to allow fine-grained estimation of individual k 's between 0 and 0.256. Suppl. Table 1 specifies all combinations of SS, LL, delays, and corresponding indifference k 's (k 's at which the LL and SS options would be chose at equal proportion). Trials were presented in randomized order.

The ITC task in Study 2 consisted of 120 trials, with again the same choice sets for all participants. In contrast to Study 1, SS amount was fixed to US\$20. Thus, participants were only presented with the LL option (with amount ranging from US\$22 to US\$85, and delays from 19 days to 180 days) and were instructed to press one of two keys to either accept and receive this LL offer, or to reject the LL offer and receive the SS offer (\$20 today) instead. Participants were informed that one trial would be randomly chosen at the end of the experiment and participant's choices implemented, resulting in incentive-compatible and mutually independent choices in each trial (as in Study 1).

Data analyses

Behavioral measures. For each participant, we calculated the proportion of SS choices (with respect to total number of non-misses) and the model-based discounting parameter k . More specifically, in Study 1 we computed k by calculating the proportion of SS choices for all *target* k 's (i.e., the k -value for which SS and LL options of any given choice trial should theoretically be chosen at 50% each). We then used linear interpolation to identify the individual indifference point at which the proportion of SS and LL choices was equal (50% each).

In Study 2, we fit a logit utility model on choice data via maximum likelihood estimation. The logit of the probability of choosing the delayed reward was modeled as follows:

$$\text{logit}(p(Y_t = \text{delayed})) = \sigma \left(\frac{LL_t}{1 + kD_t} - 20 \right)$$

Where LL_t is the LL amount in trial t and D_t is the delay in trial t . σ was included as a scaling parameter that controls the relationship between utility difference scale and choice. Individual k 's were log-transformed in both studies to obtain non-skewed distributions of discounting parameters.

MRI data acquisition. Functional and structural brain imaging data for Study 1 was acquired using a Siemens Trio 3T scanner (Erlangen, Germany) at the Life & Brain Institute, Bonn University Hospital, Germany. Functional images used a T2* weighted EPI-GRAPPA sequence (TR = 2.5s, TE = 30ms, flip angle = 90°, FOV = 192mm) and covered

the whole brain in 37 slices (voxel size of 2x2x3mm). Structural images were acquired using a T1 weighted MPRAGE sequence (1mm isomorphic voxels).

For Study 2, the functional and structural imaging data was acquired with a Siemens 3T Trio scanner with a 32-channel head coil. High-resolution T1-weighted anatomical images were acquired using an MPRAGE sequence (T1 = 1100ms; 160 axial slices, 0.9375 x 0.9375 x 1.000 mm; 192 x 256 matrix). T2*-weighted functional images were acquired using an EPI sequence with 3mm isotropic voxels, 64 x 64 matrix, TR = 3,000ms, TE = 25ms, 53 axial slices, 104 volumes. B0 fieldmap images were collected for distortion correction (TR = 1270ms, TE = 5 and 7.46ms).

Preprocessing and basic statistical analyses of fMRI data. Preprocessing for Study 1 was performed in SPM12 and used a standard pipeline of motion correction, slice time correction, spatial normalization to MNI space, and spatial smoothing of images using an 8mm FWHM Gaussian kernel. Preprocessing for Study 2 was performed in FSL according to the original preprocessing pipeline for Kable et al. (2017). This involved the standard pipeline of motion correction, b0 map unwarping, interleaved slice time correction, spatial smoothing with FWHM 9mm Gaussian kernel, and high-pass filtering (cutoff = 104s).

For Study 1, we used SPM12 to fit a general linear model (GLM) for each participant's imaging data, with choice onset modeled as a stick function (0s duration) as the main regressor and mean-centered parametric modulators for delay, relative LL amount, SS amount, and reaction time. Six nuisance regressors were added to control for movement artefacts.

For Study 2, FSL was used to fit an otherwise similar statistical model with a choice onset as main regressor and mean-centered parametric modulators for delay, LL amount, and reaction time. As in Study 1, six movement regressors were added to control for head movement.

Individual contrast images were calculated for the following three regressors of interest that were available for both studies: 1) Choice onset versus implicit baseline, 2) parametric modulation by (relative) LL amount, and 3) parametric modulation by Delay. Contrast images were gray-matter-masked, in order to remove voxels that are unlikely to contain meaningful BOLD signal, and individually z-scored to remove differences in scale across images and thereby make results transferable across studies and datasets.

Training and cross-validation. Training and cross-validation was performed on data from Study 1 only. In order to combine information across all three contrasts images of interest (Choice onset, relative LL amount, and Delay), we concatenated the contrast images for each participant, resulting in a feature space that was triple the size of a single brain image. LASSO-PCR (least absolute shrinkage and selection operator-principal component regression)⁴²—a machine-learning based regression algorithm—was then used to train a classifier to predict $\log(k)$ across all voxel weights of the concatenated contrast images. LASSO-PCR first performs a data reduction using principal component regression, thus identifying brain regions and networks that are highly correlated with each other. It then performs the LASSO algorithm, which shrinks regression weights towards zero, thus reducing the contribution of less important and more unstable components. LASSO-PCR has been shown to be advantageous for brain images for several reasons see^{37,43}: it is adequate for predictions based on hundred-thousand voxels, it takes into account multicollinearity between voxels and brain regions, and it yields interpretable results by allowing reconstruction of voxel weight maps based on PCR results.

To assess the predictive accuracy of the classifier in new subjects, we used 10-fold cross-validation. Thus, the training data was split up in 10 stratified combinations of training (90%) and test sets (10%), such that every subject's data was used for the training of the classifier in 9 folds and held out in the remaining fold to independently assess the prediction-outcome correlation. Tenfold cross-validation was chosen *a priori* as a good compromise between maximizing the sample size in each training set, while at the same time avoiding leave-one-out-cross-validation, which can underestimate accuracy by inducing a systematic bias towards negative prediction-outcome correlations^{36,71}. A priori set default regularization parameters were used for all machine-learning analysis to avoid biasing the model parameters to the data and thereby generating over-optimistic accuracy scores. Permutation tests (5000 iterations of randomly permuting the $\log(k)$ -values) were used to generate null-distributions and to assess the statistical significance of the prediction-outcome correlation and the mean absolute error.

Bootstrapping and thresholding. To identify the brain areas contributing with the most reliable positive or negative weights, we performed a bootstrap analysis. 5000 samples with replacements were taken from the paired brain and outcome data and the LASSO-PCR was repeated for each bootstrap sample. Two-tailed, uncorrected p-values were calculated for each voxel based on the proportion of weights above or below zero^{37,43}.

False Discovery Rate (FDR) correction was applied to p-values to correct for multiple comparisons across the whole feature space (three combined brain maps).

Independent test set. Study 2 was used as an independent test set to assess validity and generalizability of the brain pattern classifier developed based on Study 1 (i.e., the 'k-marker'). To assess the response of the predictive marker in Study 2 we calculated the matrix dot product between the k -marker and the concatenated contrast images (Choice onset, LL amount, and Delay) from each participant. The dot product reflects the pattern similarity between the classifier and each participant's set of contrast images and, in sum with the classifier's intercepts provides a predicted value of k . Predictive accuracy of the marker was quantified by correlating the predicted value of k with the actual k 's of each participant and by calculating the mean absolute error for each prediction.

References

- 1 Moffitt, T. E. *et al.* A gradient of childhood self-control predicts health, wealth, and public safety. *Proc. Natl. Acad. Sci. U. S. A.* **108**, 2693-2698, doi:10.1073/pnas.1010076108 (2011).
- 2 Mischel, W., Shoda, Y. & Peake, P. K. The nature of adolescent competencies predicted by preschool delay of gratification. *J. Pers. Soc. Psychol.* **54**, 687-696, doi:10.1037//0022-3514.54.4.687 (1988).
- 3 Kirby, K. N. & Herrnstein, R. J. Preference Reversals Due to Myopic Discounting of Delayed Reward. *Psychol. Sci.* **6**, 83-89, doi:10.1111/j.1467-9280.1995.tb00311.x (1995).
- 4 Frederick, S., Loewenstein, G. & O'Donoghue, T. Time Discounting and Time Preference: A Critical Review. *J. Econ. Lit.* **40**, 351-401, doi:10.1257/002205102320161311 (2002).
- 5 Laibson, D. Golden eggs and hyperbolic discounting. *Q. J. Econ.* **112**, 443-478 (1997).
- 6 Mole, T. B. *et al.* Impulsivity in disorders of food and drug misuse. *Psychol. Med.* **45**, 771-782, doi:10.1017/S0033291714001834 (2015).
- 7 MacKillop, J. *et al.* Delayed reward discounting and addictive behavior: a meta-analysis. *Psychopharmacology* **216**, 305-321, doi:10.1007/s00213-011-2229-0 (2011).
- 8 Amlung, M., Petker, T., Jackson, J., Balodis, I. & MacKillop, J. Steep discounting of delayed monetary and food rewards in obesity: a meta-analysis. *Psychol. Med.* **46**, 2423-2434, doi:10.1017/S0033291716000866 (2016).
- 9 Bickel, W. K., Odum, A. L. & Madden, G. J. Impulsivity and cigarette smoking: delay discounting in current, never, and ex-smokers. *Psychopharmacology* **146**, 447-454, doi:10.1007/pl00005490 (1999).
- 10 Price, M., Lee, M. & Higgs, S. Impulsivity, eating behaviour and performance on a delay discounting task. *Appetite* **71**, 483, doi:10.1016/j.appet.2013.06.053 (2013).
- 11 Amlung, M. *et al.* Delay Discounting as a Transdiagnostic Process in Psychiatric Disorders: A Meta-analysis. *JAMA Psychiatry* **76**, 1176-1186, doi:10.1001/jamapsychiatry.2019.2102 (2019).
- 12 Lempert, K. M., Steinglass, J. E., Pinto, A., Kable, J. W. & Simpson, H. B. Can delay discounting deliver on the promise of RDoC? *Psychol. Med.* **49**, 190-199, doi:10.1017/S0033291718001770 (2019).

- 691 13 Mozaffarian, D., Wilson, P. W. F. & Kannel, W. B. Beyond established and novel
692 risk factors: lifestyle risk factors for cardiovascular disease. *Circulation* **117**, 3031-
693 3038, doi:10.1161/CIRCULATIONAHA.107.738732 (2008).
- 694 14 Audrain-McGovern, J. *et al.* Does delay discounting play an etiological role in
695 smoking or is it a consequence of smoking? *Drug Alcohol Depend.* **103**, 99-106,
696 doi:10.1016/j.drugalcdep.2008.12.019 (2009).
- 697 15 Fernie, G. *et al.* Multiple behavioural impulsivity tasks predict prospective alcohol
698 involvement in adolescents. *Addiction* **108**, 1916-1923, doi:10.1111/add.12283
699 (2013).
- 700 16 Scarborough, P. *et al.* The economic burden of ill health due to diet, physical
701 inactivity, smoking, alcohol and obesity in the UK: an update to 2006–07 NHS
702 costs. *J. Public Health* **33**, 527-535, doi:10.1093/pubmed/fdr033 (2011).
- 703 17 Pehlivanova, M. *et al.* Diminished Cortical Thickness Is Associated with Impulsive
704 Choice in Adolescence. *J. Neurosci.* **38**, 2471-2481,
705 doi:10.1523/JNEUROSCI.2200-17.2018 (2018).
- 706 18 Bernhardt, B. C. *et al.* Medial prefrontal and anterior cingulate cortical thickness
707 predicts shared individual differences in self-generated thought and temporal
708 discounting. *Neuroimage* **90**, 290-297, doi:10.1016/j.neuroimage.2013.12.040
709 (2014).
- 710 19 van den Bos, W., Rodriguez, C. A., Schweitzer, J. B. & McClure, S. M. Connectivity
711 strength of dissociable striatal tracts predict individual differences in temporal
712 discounting. *J. Neurosci.* **34**, 10298-10310, doi:10.1523/JNEUROSCI.4105-
713 13.2014 (2014).
- 714 20 Cooper, N., Kable, J. W., Kim, B. K. & Zauberman, G. Brain activity in valuation
715 regions while thinking about the future predicts individual discount rates. *J.*
716 *Neurosci.* **33**, 13150-13156, doi:10.1523/JNEUROSCI.0400-13.2013 (2013).
- 717 21 Ersner-Hershfield, H., Wimmer, G. E. & Knutson, B. Saving for the future self:
718 neural measures of future self-continuity predict temporal discounting. *Soc. Cogn.*
719 *Affect. Neurosci.* **4**, 85-92, doi:10.1093/scan/nsn042 (2009).
- 720 22 Lebreton, M. *et al.* A critical role for the hippocampus in the valuation of imagined
721 outcomes. *PLoS Biol.* **11**, e1001684, doi:10.1371/journal.pbio.1001684 (2013).
- 722 23 Kable, J. W. & Levy, I. Neural markers of individual differences in decision-making.
723 *Curr Opin Behav Sci* **5**, 100-107, doi:10.1016/j.cobeha.2015.08.004 (2015).

- 724 24 Bartra, O., McGuire, J. T. & Kable, J. W. The valuation system: a coordinate-based
725 meta-analysis of BOLD fMRI experiments examining neural correlates of
726 subjective value. *Neuroimage* **76**, 412-427,
727 doi:10.1016/j.neuroimage.2013.02.063 (2013).
- 728 25 Hare, T. A., Hakimi, S. & Rangel, A. Activity in dlPFC and its effective connectivity
729 to vmPFC are associated with temporal discounting. *Front. Neurosci.* **8**, 50,
730 doi:10.3389/fnins.2014.00050 (2014).
- 731 26 Lempert, K. M. *et al.* Neural and behavioral correlates of episodic memory are
732 associated with temporal discounting in older adults. *Neuropsychologia* **146**,
733 107549, doi:10.1016/j.neuropsychologia.2020.107549 (2020).
- 734 27 Peters, J. & Büchel, C. The neural mechanisms of inter-temporal decision-making:
735 understanding variability. *Trends Cogn. Sci.* **15**, 227-239,
736 doi:10.1016/j.tics.2011.03.002 (2011).
- 737 28 Benoit, R. G., Gilbert, S. J. & Burgess, P. W. A neural mechanism mediating the
738 impact of episodic prospection on farsighted decisions. *J. Neurosci.* **31**, 6771-
739 6779, doi:10.1523/JNEUROSCI.6559-10.2011 (2011).
- 740 29 Zauberman, G., Kim, B. K., Malkoc, S. A. & Bettman, J. R. Discounting Time and
741 Time Discounting: Subjective Time Perception and Intertemporal Preferences. *J.*
742 *Mark. Res.* **46**, 543-556, doi:10.1509/jmkr.46.4.543 (2009).
- 743 30 MacNiven, K. H., Leong, J. K. & Knutson, B. Medial forebrain bundle structure is
744 linked to human impulsivity. *Sci Adv* **6**, eaba4788, doi:10.1126/sciadv.aba4788
745 (2020).
- 746 31 Berman, M. G. *et al.* Dimensionality of brain networks linked to life-long individual
747 differences in self-control. *Nat. Commun.* **4**, 1373, doi:10.1038/ncomms2374
748 (2013).
- 749 32 Li, N. *et al.* Resting-state functional connectivity predicts impulsivity in economic
750 decision-making. *J. Neurosci.* **33**, 4886-4895, doi:10.1523/JNEUROSCI.1342-
751 12.2013 (2013).
- 752 33 Poldrack, R. A. *et al.* Scanning the horizon: towards transparent and reproducible
753 neuroimaging research. *Nat. Rev. Neurosci.* **18**, 115-126,
754 doi:10.1038/nrn.2016.167 (2017).
- 755 34 Kragel, P. A., Koban, L., Barrett, L. F. & Wager, T. D. Representation, Pattern
756 Information, and Brain Signatures: From Neurons to Neuroimaging. *Neuron* **99**,
757 257-273, doi:10.1016/j.neuron.2018.06.009 (2018).

- 758 35 Woo, C.-W., Chang, L. J., Lindquist, M. A. & Wager, T. D. Building better
759 biomarkers: brain models in translational neuroimaging. *Nat. Neurosci.* **20**, 365-
760 377, doi:10.1038/nn.4478 (2017).
- 761 36 Scheinost, D. *et al.* Ten simple rules for predictive modeling of individual
762 differences in neuroimaging. *Neuroimage* **193**, 35-45,
763 doi:10.1016/j.neuroimage.2019.02.057 (2019).
- 764 37 Wager, T. D. *et al.* An fMRI-based neurologic signature of physical pain. *N. Engl.*
765 *J. Med.* **368**, 1388-1397, doi:10.1056/NEJMoA1204471 (2013).
- 766 38 Rosenberg, M. D. *et al.* Behavioral and neural signatures of working memory in
767 childhood. *J. Neurosci.* **40**, 5090-5104, doi:10.1523/JNEUROSCI.2841-19.2020
768 (2020).
- 769 39 Yu, H. *et al.* A Generalizable Multivariate Brain Pattern for Interpersonal Guilt.
770 *Cereb. Cortex* **30**, 3558-3572, doi:10.1093/cercor/bhz326 (2020).
- 771 40 Gianaros, P. J. *et al.* A Brain Phenotype for Stressor-Evoked Blood Pressure
772 Reactivity. *J. Am. Heart Assoc.* **6**, doi:10.1161/JAHA.117.006053 (2017).
- 773 41 Eisenbarth, H., Chang, L. J. & Wager, T. D. Multivariate Brain Prediction of Heart
774 Rate and Skin Conductance Responses to Social Threat. *J. Neurosci.* **36**, 11987-
775 11998, doi:10.1523/JNEUROSCI.3672-15.2016 (2016).
- 776 42 Tibshirani, R. Regression Shrinkage and Selection Via the Lasso. *J. R. Stat. Soc.*
777 *Series B Stat. Methodol.* **58**, 267-288, doi:10.1111/j.2517-6161.1996.tb02080.x
778 (1996).
- 779 43 Wager, T. D., Atlas, L. Y., Leotti, L. A. & Rilling, J. K. Predicting individual
780 differences in placebo analgesia: contributions of brain activity during anticipation
781 and pain experience. *J. Neurosci.* **31**, 439-452, doi:10.1523/JNEUROSCI.3420-
782 10.2011 (2011).
- 783 44 Kable, J. W. *et al.* No Effect of Commercial Cognitive Training on Brain Activity,
784 Choice Behavior, or Cognitive Performance. *J. Neurosci.* **37**, 7390-7402,
785 doi:10.1523/JNEUROSCI.2832-16.2017 (2017).
- 786 45 Kable, J. W. & Glimcher, P. W. The neural correlates of subjective value during
787 intertemporal choice. *Nat. Neurosci.* **10**, 1625-1633, doi:10.1038/nn2007 (2007).
- 788 46 Jarmolowicz, D. P. *et al.* Robust relation between temporal discounting rates and
789 body mass. *Appetite* **78**, 63-67, doi:10.1016/j.appet.2014.02.013 (2014).
- 790 47 Rachlin, H., Raineri, A. & Cross, D. Subjective probability and delay. *J. Exp. Anal.*
791 *Behav.* **55**, 233-244, doi:10.1901/jeab.1991.55-233 (1991).

- 792 48 Green, L. & Myerson, J. How many impulsivities? A discounting perspective. *J.*
793 *Exp. Anal. Behav.* **99**, 3-13, doi:10.1002/jeab.1 (2013).
- 794 49 Shamosh, N. A. *et al.* Individual differences in delay discounting: relation to
795 intelligence, working memory, and anterior prefrontal cortex. *Psychol. Sci.* **19**, 904-
796 911, doi:10.1111/j.1467-9280.2008.02175.x (2008).
- 797 50 Zachary, R. A. & Shipley, W. C. *Shipley institute of living scale: Revised manual.*
798 (WPS, Western Psychological Services, 1986).
- 799 51 Green, L., Fry, A. F. & Myerson, J. Discounting of Delayed Rewards: A Life-Span
800 Comparison. *Psychol. Sci.* **5**, 33-36, doi:10.1111/j.1467-9280.1994.tb00610.x
801 (1994).
- 802 52 Reimers, S., Maylor, E. A., Stewart, N. & Chater, N. Associations between a one-
803 shot delay discounting measure and age, income, education and real-world
804 impulsive behavior. *Pers. Individ. Dif.* **47**, 973-978, doi:10.1016/j.paid.2009.07.026
805 (2009).
- 806 53 Seaman, K. L. *et al.* Subjective value representations during effort, probability and
807 time discounting across adulthood. *Soc. Cogn. Affect. Neurosci.* **13**, 449-459,
808 doi:10.1093/scan/nsy021 (2018).
- 809 54 Yarkoni, T., Poldrack, R. A., Nichols, T. E., Van Essen, D. C. & Wager, T. D. Large-
810 scale automated synthesis of human functional neuroimaging data. *Nat. Methods*
811 **8**, 665-670 (2011).
- 812 55 Yeo, B. T. T. *et al.* The organization of the human cerebral cortex estimated by
813 intrinsic functional connectivity. *J. Neurophysiol.* **106**, 1125-1165,
814 doi:10.1152/jn.00338.2011 (2011).
- 815 56 Koban, L., Jepma, M., López-Solà, M. & Wager, T. D. Different brain networks
816 mediate the effects of social and conditioned expectations on pain. *Nat. Commun.*
817 **10**, 4096, doi:10.1038/s41467-019-11934-y (2019).
- 818 57 Insel, T. R. & Cuthbert, B. N. Medicine. Brain disorders? Precisely. *Science* **348**,
819 499-500, doi:10.1126/science.aab2358 (2015).
- 820 58 Bickel, W. K. *et al.* Using crowdsourcing to compare temporal, social temporal, and
821 probability discounting among obese and non-obese individuals. *Appetite* **75**, 82-
822 89, doi:10.1016/j.appet.2013.12.018 (2014).
- 823 59 Levy, D. J. & Glimcher, P. W. Comparing apples and oranges: using reward-
824 specific and reward-general subjective value representation in the brain. *J.*
825 *Neurosci.* **31**, 14693-14707, doi:10.1523/JNEUROSCI.2218-11.2011 (2011).

- 826 60 Clithero, J. A. & Rangel, A. Informatic parcellation of the network involved in the
827 computation of subjective value. *Soc. Cogn. Affect. Neurosci.* **9**, 1289-1302,
828 doi:10.1093/scan/nst106 (2014).
- 829 61 McClure, S. M., Laibson, D. I., Loewenstein, G. & Cohen, J. D. Separate neural
830 systems value immediate and delayed monetary rewards. *Science* **306**, 503-507,
831 doi:10.1126/science.1100907 (2004).
- 832 62 Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S. & Cohen, J. D. Conflict
833 monitoring and cognitive control. *Psychol. Rev.* **108**, 624-652 (2001).
- 834 63 Hutcherson, C. A., Rangel, A. & Tuschke, A. Evidence accumulation, not "self-
835 control," explains why the dlPFC activates during normative choice. *bioRxiv*
836 (2020).
- 837 64 Shenhav, A., Botvinick, M. M. & Cohen, J. D. The expected value of control: an
838 integrative theory of anterior cingulate cortex function. *Neuron* **79**, 217-240,
839 doi:10.1016/j.neuron.2013.07.007 (2013).
- 840 65 Kool, W., McGuire, J. T., Wang, G. J. & Botvinick, M. M. Neural and behavioral
841 evidence for an intrinsic cost of self-control. *PLoS One* **8**, e72626,
842 doi:10.1371/journal.pone.0072626 (2013).
- 843 66 Ballard, K. & Knutson, B. Dissociable neural representations of future reward
844 magnitude and delay during temporal discounting. *Neuroimage* **45**, 143-150,
845 doi:10.1016/j.neuroimage.2008.11.004 (2009).
- 846 67 Berkman, E. T., Hutcherson, C. A., Livingston, J. L., Kahn, L. E. & Inzlicht, M. Self-
847 Control as Value-Based Choice. *Curr. Dir. Psychol. Sci.* **26**, 422-428,
848 doi:10.1177/0963721417704394 (2017).
- 849 68 Amasino, D. R., Sullivan, N. J., Kranton, R. E. & Huettel, S. A. Amount and time
850 exert independent influences on intertemporal choice. *Nat Hum Behav* **3**, 383-392,
851 doi:10.1038/s41562-019-0537-2 (2019).
- 852 69 Jones, B. & Rachlin, H. Social discounting. *Psychol. Sci.* **17**, 283-286,
853 doi:10.1111/j.1467-9280.2006.01699.x (2006).
- 854 70 Strombach, T. *et al.* Social discounting involves modulation of neural value signals
855 by temporoparietal junction. *Proc. Natl. Acad. Sci. U. S. A.* **112**, 1619-1624,
856 doi:10.1073/pnas.1414715112 (2015).
- 857 71 Poldrack, R. A., Huckins, G. & Varoquaux, G. Establishment of Best Practices for
858 Evidence for Prediction: A Review. *JAMA Psychiatry* **77**, 534-540,
859 doi:10.1001/jamapsychiatry.2019.3671 (2020).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [02KobanetalSuppl.pdf](#)