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Vagal signaling and the somatic marker hypothesis: The effect of transcutaneous vagal nerve stimulation on delay discounting is modulated by positive mood

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ABSTRACT

Controlling impulsivity and delaying gratifications are key features of effective self-control. Delay Discounting (DD) indexes the ability to delay rewards and previous research has shown that discounting is influenced by affective states such as mood. According to the Somatic Marker Hypothesis (SMH), afferent somatic signals, such as mood, are carried by the vagus and can influence decision making. In the current study, we employed transcutaneous vagus nerve stimulation (tVNS), a novel non-invasive brain stimulation technique that stimulates the auricular branch of the afferent vagus nerve (located in the outer ear), to assess its effects on decision impulsivity, while taking into account individuals' mood and resting-state HRV as a possible confounding factor. Employing a within-subjects cross-over design, 94 participants received active or sham tVNS while performing delay discounting in two separate sessions. As compared to sham, active tVNS increased discounting, but only for individuals reporting lower positive mood, regardless of the level of negative mood reported. We evidence that the effect of tVNS on reward discounting depends on the level of positive mood. This result suggests that positive mood state might be a proxy of task-relevant arousal, likely influencing the effectiveness of afferent vagal stimulation on self-control processes, as temporal discounting.

1. Introduction

Regulating behavioral responses to environmental demands is paramount for daily life functioning. Greater regulatory control is associated with more efficient physiological, cognitive and emotional responses that make up behavior functionally adapted to the environment. One of the key factors in regulatory control is the degree to which individuals are able to inhibit impulsive decisions (Hammond et al., 2012). A classical task aimed at exploring impulsive decisions and self-regulation is the Delay Discounting Task, which requires individuals to virtually choose between a smaller, but immediate reward (i.e., 100€ now) or a delayed, but larger reward (200€ in 1 month). This task evaluates the trade-off between immediate and delayed rewards, in which more impulsive individuals are less able to delay rewards (Cona et al., 2019; de Wit, 2009; Kirby et al., 1999; Koff and Lucas, 2011; McLeish and Oxoby, 2007; Mobini et al., 2007).

The ability to delay rewards can be indexed by delay discounting (DD; Bickel et al., 2014), and particularly, by the discount constant *k* which indexes the internal rate of delay discounting (Frost and Mcnaughton, 2017; Odum, 2011a). It reflects the main behavioral model of decision impulsivity, given that their tasks have been developed to measure the rate of devaluation of a reward over time. Higher discount rates are characteristic for psychopathologies like substance abuse, depression and attention hyperactivity disorder (Bickel et al., 2012; Cona et al., 2019). Even if DD has been demonstrated to be a relatively stable trait (Bickel et al., 2014; Odum, 2011b), it can also be influenced by several factors, such as personality traits or acute stress, revealing within-individual variability over time (Frost and Mcnaughton, 2017; Kimura et al., 2013; Lempert et al., 2012; Madden and Bickel, 2010).

The affective state of the individual is an additional factor that modulates reward delaying (Herman et al., 2018; Lerner et al., 2004). For example, mood can be defined as a transient emotional positive or negative experience that influences decision making and self-regulation

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(Herman et al., 2018). In particular, the interplay between mood and DD has already been proposed in the literature although with mixed results. On one hand, some studies showed that people reporting high negative mood tend to have higher discount rates, suggesting that negative affect is associated with increased temporal impulsivity (Guan et al., 2015; Koff and Lucas, 2011; Lerner et al., 2012). On the other hand, positive mood has been related to a preference for delayed larger rewards and, in general, to lower levels of temporal impulsivity (Liu et al., 2013; Weafer et al., 2013). Taken together, positive affect seems to be associated with increased patience for a reward, while negative affect tends to be related to "near-sighted" behaviors reflecting higher levels of temporal impulsivity. However, in spite of these results, several other studies have not found any relation between either positive or negative mood and discounting (Beck and Triplett, 2009; Daly et al., 2009; Van den Bergh et al., 2008).

According to Cyders and Smith 2007, positive and negative urgency constitute a well-established personality trait by which mood can trigger rash actions and impulsive behaviors. Negative mood urgency is a tendency to act under the influence of strong impulses, often associated with negative affect, and positive urgency is the tendency to act impulsively while experiencing strong positive emotions (Cyders and Smith, 2007, 2008; Whiteside and Lynam, 2001). Although previous studies have suggested a beneficial effect of positive mood in control processes (Weafer et al., 2013), the urge to override immediate gratification driven by either positive or negative mood states (i.e., heavy drinking or emotional eating) may also have negative consequences in the future and might serve as attempts to alleviate one's mood state (Cyders and Smith, 2007; Herman et al., 2018).

In the context of decision making, the somatic marker hypothesis (SMH; Bechara et al., 2005; Damasio et al., 1991, 1996) draws attention to the interaction between affective states and cognitive processes (Critchley et al., 2013). At the neural level, a number of functional magnetic resonance imaging studies have supported the interaction between mood and decision making consistent with the SMH (Critchley et al., 2013). Key regions for affective regulation such as the prefrontal cortex, anterior cingulate cortex, amygdala or the basal ganglia important for self-control also underlie impulsive behaviors (Hinvest et al., 2011; Murphy et al., 2003). In fact, studies have identified activation in prefrontal and limbic regions as the neural system for temporal discounting (Bickel et al., 2007; Cona et al., 2019; Frost and Mcnaughton, 2017; Kishinevsky et al., 2012; Shamosh et al., 2008), supporting that neural circuits involved in impulsive behaviors (and thus self-control) and emotions processing overlap.

The SMH states that economic decision-making is influenced by autonomic/somatic bodily states signals that arise in bioregulatory processes expressed in emotions and feelings (Critchley et al., 2013; Damasio et al., 1991, 1996). The vagus nerve is a key structure of the autonomous nervous system and has been proposed as a conductor of afferent somatic signals that contribute to decision making and self-control (Martin et al., 2004). One way to study the causal role of autonomous activity in self-control is by stimulating the afferent vagus by means of transcutaneous vagus nerve stimulation (tVNS: Dietrich et al., 2008; Frangos et al., 2015). tVNS is a novel non-invasive brain stimulation technique that stimulates the afferent branch of the vagus nerve carrying somatic signals that are essential for the regulation of complex behaviors (Porges, 2007, 2003). In this sense, previous studies have pointed out a possible causal role of the vagus nerve in enhancing control processes, and in particular inhibitory control as indexed by stop-change (Steenbergen et al., 2015) and Go/NoGo performance (Beste et al., 2016), as well as adaptation to conflict (Fischer et al., 2018). Therefore, considering that mood is an afferent somatic signal that influences delay discounting and self-control, and that the vagus is a conductor of somatic markers, one would expect that the stimulation of the vagus could impact the relationship between affective mood states and delay discounting.

Although the effects of tVNS are so far thought to be unspecific of any functional neuroanatomical region, the systemic stimulation of GABA and norepinephrine (NE) in prefrontal and striatal regions might be the potential underlying mechanism (Beste et al., 2016; Ventura-Bort et al., 2018; Warren et al., 2019). Furthermore, consistent evidence supports the relationship between impulsive behavior, delaying rewards and dopamine (DA: Friston et al., 2014; Kobayashi and Schultz, 2008). Importantly, previous studies have shown that NE and DA collaborate in facilitating many cognitive and affective functions such as memory, learning, attention or addiction (Harley, 2004; Xing et al., 2016). However, given that no studies to the date have evaluated the effect of tVNS on decision impulsivity, and DD in particular, it is still unexplored whether the projections of the vagus to the locus coeruleus NE neurons can have an impact on midbrain DA neurons involved in delay discounting.

Supported by the findings above, one would hypothesize tVNS to decrease behavioral impulsiveness as indexed by discount rates. But, first of all, considering stimulation of the afferent vagus - a conductor of somatic signals - one would expect mood state to influence delay discounting (Herman et al., 2018; Koff and Lucas, 2011). However, given that findings on the relation between mood and delay discounting are still inconsistent (Beck and Triplett, 2009; Daly et al., 2009; Van den Bergh et al., 2008), tVNS allows us to explore the causal role of the somatic afferent markers in decision making. For that purpose, in a single-blind randomized design participants received active or sham tVNS stimulation in two separate sessions and while performing, among others, the delay discounting task. Prior to the start of the stimulation, we used the Positive and Negative Affect Schedule (PANAS, Watson et al., 1988) as a measure of self-reported mood. While positive mood refers to the feeling of enthusiasm and alertness of the individual, negative mood reflects to what extent an individual feel distress and discomfort. Moreover, since a body of evidence suggested a possible relation of baseline HRV, an index of efferent cardiac vagal tone, with cognitive flexibility and self-control (Colzato et al., 2018a; Colzato and Steenbergen, 2017), we decided to measure it before the stimulation, in order to control for its contribution as a possible confounding factor (Barch et al., 2013; Bickel et al., 2014).

2. Materials and methods

2.1. Participants and design

An a-priori power analysis was performed using G*Power 3.1.7 (Faul et al., 2007) to estimate the approximate number of participants required in each cell given the traditional 0.01 criterion of statistical significance (the traditional alpha = 0.05 slightly corrected for multiple testing) and desired power of 0.90. Based on prior results (Sellaro et al., 2018) using a similar study design, medium effect sizes ($r \approx 0.20$) were anticipated. Results of the power analysis estimated that the total number of participants needed was 97. Thus, ninety-eight participants participated in a single-blind, randomized sham-controlled crossover study on the effect of online (i.e., simulation during the evaluated task) tVNS on cognition. Participants were recruited via flyers hung in the institutional building, an online recruiting system, and word-to-mouth and were offered partial course credit or a monetary reward for participation. Following (Colzato et al., 2005, 2008), participants were screened individually using a questionnaire adapted from the Mini International Neuropsychiatric Interview (M.I.N.I.; Sheehan et al., 1998). Participants were considered eligible for participation if they met the following criteria: age between 18 and 30 years; no history of neurological or psychiatric disorders; no current or history of substance abuse or excessive alcohol intake (> 25 units per week); no use of soft- or hard drugs from at least one week before participation until completion of participation; no gastrointestinal disease; no mental or physical disability that will hinder participation; no history of brain surgery, tumor or intracranial metal implantation; no chronic or acute medications except for contraceptives; no pregnancy; no susceptibility to

seizures, fainting, panic attacks, or migraine; no epilepsy or first-degree relative with epilepsy; no pacemaker or other implanted devices; no skin conditions in the left ear.

Before signing the informed consent, participants received verbal and written explanation of the procedure and possible adverse effects (i.e., itching and tingling skin sensation, skin reddening, and headache). No information was provided about the different types of stimulation (active vs. sham) or about the hypotheses concerning the outcome of the experiment. Written informed consent was obtained from all participants. The experiment conformed to the ethical standards of the Declaration of Helsinki (World Health Organisation, 2013) and the protocol was approved by the local ethics committee (Leiden University, Institute for Psychological Research). Written informed consent was obtained from all participants.

2.2. Procedure

All participants were tested individually. After having read and signed the informed consent, participants were asked to turn off all mobile and Bluetooth devices they were carrying and remain seated and try to relax for 5 min, after which their HRV was recorded for 5 min. During this measuring period, participants were not instructed about breathing, but instead were breathing spontaneously. Following Denver et al. (2007), we did not consider respiration rate as a factor in the HRV assessment. Upon completion of the HRV measurement, tVNS was applied. Following previous studies (Beste et al., 2016; Colzato et al., 2018b Jongkees et al., 2018; Sellaro et al., 2018; Steenbergen et al., 2015), a 15-minute initiation period was induced to ensure effective stimulation when initiating the critical task. During this 15-minute waiting period, participants response to tVNS was monitored while they filled in well-validated dispositional questionnaires aimed at assessing mood (i.e., evaluated in both session), depression, anxiety, and stress, as well as impulsivity and sensitivity to reward and punishment (see descriptions below). After the completion of the questionnaires and after 15 min had passed, participants were asked to perform a battery of cognitive tasks about decision making,[1] among which the delay discounting task. tVNS was applied for 60 min and was stopped after completion of the cognitive tasks. At the end, participants were asked to answer a number of after-effects questions in which participants rated, on a five-point (1–5) scale, how much they experienced: (1) headache, (2) neck pain, (3) nausea, (4) muscle contraction in face and/or neck, (5) stinging sensation under the electrodes, (6) burning sensation under the electrodes, (7) uncomfortable (generic) feelings, (8) other sensations and/or adverse effects. In addition, participants answered the question which stimulation they thought to have received (sham, active, or no idea). Upon completion of this questionnaire, an appointment for the second session was made or, in case of the second session, participants were debriefed and awarded their reward.

2.3. tVNS

To avoid possible arrhythmic effects, stimulation was always applied to the left ear, in keeping with previous studies (Beste et al., 2016; Colzato et al., 2017; Colzato et al., 2018b; Jongkees et al., 2018; Sellaro et al., 2018). After cleaning the electrodes and the ear with 70% isopropyl alcohol, Cerbomed GbmH (Erlangen, Germany), Nemos® tVNS was applied to the cymba concha of the left ear to stimulate the auricular branch of the vagus. Sham stimulation was established by placing the electrode on the earlobe, which is not innervated by vagal afferents (Fallgatter et al., 2003; Peuker and Filler, 2002). This tVNS device delivers an electrical pulse of 0.5 mA every 200–300 μm at a frequency of 25 Hz by means of two titan electrodes mounted on a gel frame, generated by the portable stimulation unit. On and off stimulation windows occurred every 30 s. The ear electrode is equipped with a size-adjustable earplug inserted in the auricle like regular headphones. To keep the electrodes at a stable position, the size between the earplug and the electrodes was adjusted to optimize the fit.

2.4. Heart rate variability recordings

Following Colzato and Steenbergen (2017) and Colzato et al., 2018a, after a 5-minute resting period, inter-beat intervals (IBI) were measured for 5 min using a Polar H7 heart rate monitoring system (Polar Electro, Kempele, Finland), which receives heart rate data from a chest belt worn by the participants. Data were recorded with the Elite HRV Smart Phone Application (https://elitehrv.com/) and processed with Kubios (premium version 3.0, 2017, Biosignal Analysis and Medical Imaging Group, University of Kuopio, Finland; Tarvainen et al., 2014), using the automatic thresholding procedure to filter out artifacts, to obtain the root mean square of the successive differences (RMSSD). Given that we were mainly interested in vagally-mediated HRV, we only considered RMSSD as a time-domain measure (DeGiorgio et al., 2010; Koenig and Thayer, 2016; Sperling et al., 2010).

2.5. Questionnaires

2.5.1. Depression, Anxiety, and Stress Scales (DASS-21, Crawford and Henry, 2003)

The DASS-21 is a 42-item self-administered questionnaire that provides measurements for the magnitude of three negative emotional states: depression, anxiety, and stress. Each of the three sub-scales has seven items that comprises a statement and four short response options to reflect severity and scored from 0 (“Did not apply to me at all”) to 3 (“Applied to me very much, or most of the time”). The DASS-Depression focuses on reports of low mood, motivation, and self-esteem, DASS-anxiety on physiological arousal, perceived panic, and fear, and DASS-stress on tension and irritability. The DASS-21 is based on a dimensional rather than a categorical conception of psychological disorder, considering that the differences between the depression, anxiety and the stress experienced by normal subjects and clinical populations are essentially differences of degree. The DASS-21 therefore has no direct implications for the allocation of participants to discrete diagnostic categories postulated in classificatory systems such as the DSM and ICD (Lovibond and Lovibond, 1995).

2.5.2. Barratt Impulsiveness Scale (BIS-II; Patton et al., 1995).

To assess trait impulsivity, including impulsive and non-impulsive (reverse scored items) preferences and behaviors, we used the Barratt Impulsiveness Scale (BIS-II; Patton et al., 1995). Total impulsiveness scores are calculated based on participant's rating (i.e., on a scale from 1 = “rarely/never” to 4 = “almost always/always”) of 30 items, and range from 30 (i.e., low self-reported impulsivity) to 120 (i.e., high self-reported impulsivity). Furthermore, the scale yields 3 subscores; Attentional Impulsiveness (i.e., an impatient tendency reflected by rapid shifts in attention), Motor Impulsiveness (i.e., a ‘reckless’ tendency reflected by immediate actions), and Non-Planning Impulsiveness (i.e., a tendency to ignore long-term consequences of actions and not to plan ahead).

[1] The additional battery of tasks included the Simon task (Fischer, Plessow, & Kiesel, 2010) measuring interference control, the foraging task (Hillis et al., 2008; 2010) measuring exploration and exploitation behavior and the IOWA gambling task (Bechera et al., 1994) measuring decision making. Given that the complete study emerged from collaboration between different research teams with various aims and theoretical frameworks, results regarding these tasks are beyond the scope of the current article. None of the cognitive tasks have been published elsewhere.

2.5.3. Sensitivity to reward and punishment questionnaire (SRPQ, Torrubia et al., 2001)

The SPSRQ is a 48-item scale in a yes/no format that provides a score for individual sensitivity to punishment, related to behavioral inhibition, and a score for sensitivity to reward, related to behavioral activation system (Gray, 1981). Both the sensitivity to punishment and reward measures have shown satisfactory test re-test reliability and convergent and discriminant validity (Torrubia et al., 2001).

2.5.4. Positive and Negative Affect Scale (PANAS; Watson et al., 1988)

Furthermore, we used the positive and negative affect scale (PANAS; $\alpha = 0.90$, Watson et al., 1988) to assess mood state. The PANAS is a validated questionnaire consisting of two 10-item mood scales providing measures of positive and negative affect. Participants are asked to rate the extent to which they experience each of the 10 positive and 10 negative emotions at this moment; 1 'very slightly or not at all', 2 'a little', 3 'moderately', 4 'quite a bit' and 5 'very much'. Summing the respective items results in a positive affect mood score a negative affect mood score, both for which the minimum score is 10 and the maximum 50.

2.6. Delay discounting task

The adjusting-immediate-amount task (AIA, Holt et al., 2012) was used to assess delay discounting. Participants were asked to imagine that the following choice occurred in real life, and had to choose between a smaller, immediate reward and a larger, delayed reward displayed on the computer screen. There were 2 delayed 'reference' amounts: 200€ and 40,000€, and six delays (1 month, 6 months, 1 year, 3 years, 5 years, and 10 years), resulting in a total of 12 conditions. The order of presentation of the 12 conditions was randomized for each participant. In order to determine the amount of immediate reward that a participant judged equal in value to the delayed (200€ or 40,000€) reward for each delay and delayed amount (i.e. the indifference point), the amount of the immediate reward was adjusted on each trial, adding or subtracting (i.e. based on the choice) half the amount of the previous change (Du et al., 2002). Specifically, on the first trial of each delay condition, the immediate amount was always half of the delayed amount (i.e. with a delayed reward of 200€, the presented immediate reward was 100€). In case a participant chose the immediate reward on the first trial (i.e. 100€), the subsequent presented immediate reward was half the value of the previous trial (i.e. to 50€). In case the participant would again choose the immediate reward, it was again decreased with half the difference of the previous change (i.e. to 25€). Would the participant then favor the delayed reward, the immediate reward would be increased with half the difference of the previous change (i.e. to 37,50€). Participants made a total of 5 choices in each condition; the indifference point was set at the value of the immediate reward that the participant would have been confronted with on the sixth trial, which has been demonstrated a reliable method to quickly estimate indifference points (Koffarnus and Bickel, 2014). Performance on this task results in an indifference point (V) for each delay, that follows as a function of the presented amount of the reward (A), the delay in time units (D) and parameter k; $V = A / (1 + kD)$ (Mazur, 1987). In other words, to model the value of the indifference point, the delay (D) is multiplied by k (equal to $((A/V) - 1) / D$), which describes how much devaluation takes place. The larger k is, the stronger the effect of delay (D) on devaluation of the reward (A) and therefore, the more discounting takes place (Frost and Mcnaughton, 2017).

2.7. Statistical analyses

Analyses were performed using IBM SPSS 23.0 (Chicago, IL) for Windows. In case of violation of the sphericity assumption, Greenhouse-Geisser correction was applied and corrected values are reported. A significance level of $p < .05$ was adopted for all statistical tests. To obtain a more reliable and stable index of mood (i.e. as assessed using the PANAS) as well as for HRV, we took the average of the baseline measures of the two sessions and used this for further analyses. To assess the strength of the effects, significant results are further analyzed within the Bayesian framework, to quantify evidence for the alternative (i.e. the presence of an effect of tVNS and/or mood) vs. the null hypothesis on delay discounting (BFinclusion) using the default settings; r scale fixed effects = 0.5, r scale random effects = 1, r scale covariates = 0.35, samples = auto (10000).

3. Results

3.1. Participant characteristics

Two participants were excluded from all analyses due to not meeting the study criteria. Two other participants dropped out, resulting in 94 participants considered for analyses.

For one participant that completed the two sessions, HRV recording failed in the session in which they received active tVNS, hence the average HRV measures were replaced with the values observed in the single session in which HRV succeeded. We applied the same method to deal with missing PANAS scores of a participant in his/her sham session (i.e. replacing them by his/her score in the active session). In addition, seven participants demonstrated an RMSSD value in one of the two sessions that was out of the range normally observed across the lifespan (i.e. 7–103 ms; Umetani et al., 1998), leading us to suspect that the HRV measurement was not reliable. Hence, for these cases, the average HRV measures were replaced by the value of the other session. Further, one participant did not perform the delay discounting task, and nine participants were excluded from further analyses, as they demonstrated extreme values (> 3SDs from the mean) on the critical k variable. Taken all the previous conditions together, the final sample size resulted in 84 participants (52 females, 32 males) considered for further analyses. See Table 1 for participant characteristics.

The Wilcoxon-signed rank test for dependent samples revealed no differences between active and sham session in RMSSD ($Z = 0.82$, $p = .41$, $M_{active} = 50.43 \pm 4.30$, $M_{sham} = 55.92 \pm 5.85$) or negative mood ($Z = 0.82$, $p = .41$, $M_{active} = 19.08 \pm 0.68$, $M_{sham} = 19.45 \pm 0.72$). However, participants' positive mood was higher in the sham ($M = 35.46 \pm 0.48$) than in the active ($M = 34.62 \pm 0.47$) tVNS session, $Z = 2.72$, $p = .02$. Given that positive and negative mood, as well as HRV, were measured prior to the start of tVNS and the stimulation sessions were counterbalanced, hereafter we considered the average of both sessions as measures of mood. Based on a median split, participants were assigned to low and high negative (Median = 18.0) or positive mood (Median = 35.0) group, resulting in the distribution displayed in Table 2. For example, participants scoring high in items such as "distress, guilty, irritable or hostile" revealed high *negative* mood, while those scoring high in items like "excited, proud, inspired or active" showed high *positive* mood. Overall, participants reported higher scores in the indicators of positive rather than negative mood. No significant differences in the distribution of participants over mood groups were observed, $X^2 = 0.07$, $p = .78$. Further, bivariate Pearson correlation between positive and negative

Table 1
Participant characteristics: Mean scores on the assessed variables are given with standard deviations in parentheses. Values represent the average of the two testing sessions.

N[M,F]	84[52,32]
Age	22.32 (2.71)
BMI	24.13 (3.81)
RMSSD	45.37 (21.85)
PANAS – positive	35.25 (3.92)
PANAS – negative	19.25 (6.06)

Table 2
Participant group distribution and PANAS scores as a function of positive or negative mood. Mean scores on the low and high positive (Median = 35.00) and negative (Median = 18.00) mood are given with standard deviations in parentheses. Please note that the terms 'low' and 'high' are relative to the median and might indicate actual low or high scores.

	Low positive	High positive	Total
Low negative	N: 24	N: 23	N: 47
	Low Pos: 32.83 (1.85)	High Pos: 38.13 (2.18)	Pos: 35.42 (3.34)
	Low Neg: 15.12 (2.28)	Low Neg: 15.02 (2.31)	Neg: 15.07 (2.72)
High negative	N: 20	N: 17	N: 37
	Low Pos: 31.67 (2.73)	High Pos: 38.97 (2.83)	Pos: 35.03 (4.60)
	High Neg: 25.10 (4.77)	High Neg: 23.94 (5.60)	Neg: 24.57 (5.13)
Total	N: 44	N: 40	N: 84
	Pos: 32.31 (2.34)	Pos: 38.49 (2.48)	Pos: 35.25 (3.92)
	Neg: 19.66 (6.17)	Neg: 18.81 (5.98)	Neg: 19.25 (6.06)

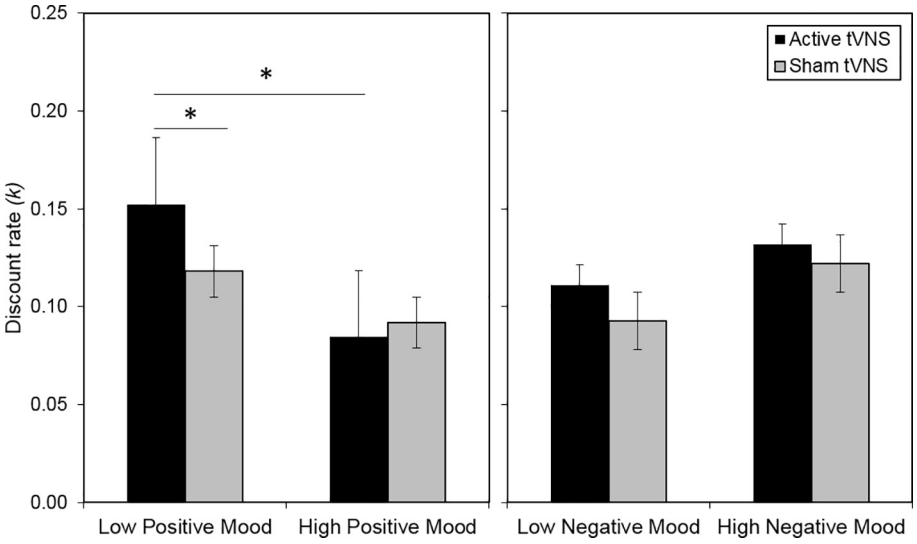

Fig. 1. Discount rate (k) for the active and sham tVNS sessions as a function of positive and negative mood groups (low vs. high). k represents the discount rate averaged for the 200 and 4000€ reward conditions. Asterisks indicate significant differences between active and sham sessions ($p < .05$). Vertical capped lines top bars indicate standard error of the mean.

mood revealed that positive and negative mood scores did not significantly correlate in our sample ($r = -0.18$, $p = .09$). Based on equation modelling analysis, Schmukle et al. (2002) suggested that while situation-specific state positive and negative affect are negatively correlated (the more positive mood, the less negative), dispositional and trait components are unrelated. That is, in the study by Schmukle et al. (2002), situation-specific positive and negative affect showed correlations within a range from $r = -0.32$ to -0.51 whereas dispositional positive and negative affect were uncorrelated. Given the absence of a significant correlation between positive and negative affect in our study, and given that we averaged scores across sessions, we may consider our affect measures predominantly dispositional; enabling individuals to report both high negative and high positive affect (see also Diener et al., 1985).

3.2. Personality questionnaires

In the first session, participants completed a battery of personality questionnaires and, overall, their self-reported scores assessing trait impulsivity, sensitivity to reward and punishment, states of depression, anxiety and stress, fell in the normal range: $BIS_{total} = 61.48 \pm 0.95$ (range 43.00–90.00), $BIS_{Attentional} = 16.35 \pm 0.30$ (range 10.00–25.00), $BIS_{Motor} = 21.60 \pm 0.47$ (range 13.00–37.00), $BIS_{NonPlanning} = 23.54 \pm 0.45$ (range 14.00–34.00), Sensitivity to reward = 11.31 ± 0.45 (range 3.00–20.00), Sensivity to punishment = 9.80 ± 0.56 (range 1.00–23.00), $DAS_{Anxiety} = 12.43 \pm 1.05$ (range 0.00–50.00), $DAS_{Depression} = 11.52 \pm 0.89$ (range 0.00–40.00), $DAS_{Stress} = 7.79 \pm 0.81$ (range 0.00–34.00).

3.3. Discounting rate (k)

The constant k reflects the extent to which participants discount the delayed gains at the empirical level (Frost and Mcnaughton, 2017), with higher k values indicating steeper discounting rates. We performed rmANOVA on k derived as a function of amount (200€ vs. 40,000€) and tVNS session (active vs. sham), with positive and negative mood group as between-subject factors. The analysis revealed three significant sources of variance. First, a main effect of amount, showing significantly higher discounting in the 200€ reward condition ($M_k = 0.17 \pm 0.01$) than in the 40,000€ reward condition ($M_k = 0.06 \pm 0.01$), ($F(1,80) = 69.15$, $p < .01$, $\eta_p^2 = 0.46$, MSE = 0.01). Second, a main effect of positive mood group ($F(1,80) = 5.75$, $p = .02$, $\eta_p^2 = 0.07$, MSE = 0.03), and third, an interaction between treatment and positive mood group (low vs. high) ($F(1,79) = 6.36$, $p = .01$, $\eta_p^2 = 0.07$, MSE = 0.01). Most importantly, within-group post-hoc tests indicated that the interaction between positive mood group and treatment was driven by an increase in discounting as a result of tVNS when experiencing low positive mood ($k_{sham} = 0.12 \pm 0.01$, $k_{active} = 0.15 \pm 0.02$, $F(1,43) = 7.16$, $p = .01$, $\eta_p^2 = 0.14$, MSE = 0.01) versus the fact that no difference in discounting was observed when experiencing high positive mood ($k_{sham} = 0.09 \pm 0.01$, $k_{active} = 0.08 \pm 0.01$, $F(1,39) = 0.34$, $p = .56$, $\eta_p^2 = 0.01$, MSE = 0.01), see Fig. 1. The other way around, there were no differences between the two mood groups in the sham session, ($k_{low} = 0.12 \pm 0.01$, $k_{high} = 0.09 \pm 0.01$, $F(1,82) = 1.81$, $p = .18$, $\eta_p^2 = 0.02$, MSE = 0.02). Importantly, compared to the high positive mood group, the low positive mood group demonstrated steeper discounting in the active session ($k_{low} = 0.15 \pm 0.02$, $k_{high} = 0.08 \pm 0.02$, $F(1,82) = 8.71$, $p < .01$, $\eta_p^2 = 0.09$,

MSE = 0.02). We neither observed a main effect of negative mood group ($F(1,79) = 0.91$, $p = .34$, $\eta_p^2 = 0.01$, MSE = 0.03), nor an interaction between stimulation treatment and negative mood ($F(1,80) = 1.48$, $p = .23$, $\eta_p^2 = 0.02$, MSE = 0.03), nor an interaction between treatment, positive and negative mood groups ($F(1,80) = 2.84$, $p = .09$, $\eta_p^2 = 0.03$, MSE = 0.01). Altogether, this suggests that differences in k were independent of the level of reported negative mood (see Fig. 1). No further sources of variance were observed, all $p_s \leq .95$ and $\geq .0.9$.

In addition, we repeated the similar analysis as before but introducing RMSSD - a measure of baseline HRV - as a covariate to control for its possible confounding role. Analysis revealed a similar pattern of results and neither the main effect of the covariate ($F(1,79) = 0.94$, $p = .33$, $\eta_p^2 = 0.01$, MSE = 0.03) or the interaction between session and RMSSD reached significance ($F(1,79) < 0.01$, $p = .39$, $\eta_p^2 = 0.53$, MSE = 0.01). Therefore, we can conclude that the effect of tVNS on delay discounting depends on positive mood, but not on baseline HRV.

3.4. Additional analysis

The present results support the role of the vagus nerve in modulating the discount of future rewards under the influence of positive mood state. We also analyzed our data within the Bayesian framework, in order to quantify and compare the relative likelihood of the data under two competing hypotheses, namely, the alternative (H1) and the null (H0) hypothesis, as indexed by the Bayes factor (BF_{10}) (Etz, 2015; Morey and Rouder, 2011). A BF_{10} of 3 or above indicates substantial evidence for the alternative over the null hypotheses (Jeffreys, 1961), whereas (by symmetry) $BF_{10} < 1/3$ indicate substantial evidence for the null hypothesis. Values of BF_{10} between roughly 1/3 and 3 indicate that the evidence is insensitive at distinguishing between the null and alternative hypotheses (Dienes, 2014). Analyses were performed using JASP 0.8.1.1 software (available on https://jasp-stats.org/). A Bayesian repeated-measures ANOVA was carried out to quantify evidence for the presence of a tVNS effect on delay discounting when considering positive and negative mood as between-groups factors. Results showed that, compared to the null model, models of positive mood group and its interaction with the treatment received strong support from the data ($BF_{treatment} = 0.48$, $BF_{positive\ mood} = 3.21$, $BF_{treatment} + BF_{positive\ mood} = 1.65$, $BF_{treatment} + BF_{positive\ mood} + BF_{treatment*positive\ mood} = 3.64$). In contrast, the models including negative mood reveal the lack of a significant effect of negative mood and tVNS treatment in delay discounting ($BF_{negative\ mood} = 0.58$, $BF_{treatment} + BF_{negative\ mood} = 0.29$, $BF_{treatment} + BF_{negative\ mood} + BF_{treatment*negative\ mood} = 0.07$). In sum, the model that received the strongest support against the null model was the interaction model between treatment and positive mood by a Bayes factor of 3.64, which was preferred to the model with only positive mood ($BF_{positive\ mood} = 3.21$) and to any of the remaining models (all BFs < 3). Taken together, the results of the Bayesian analyses are consistent with the conclusion that tVNS enhances the effect of low positive mood on delay discounting.

3.5. After effects

Paired samples t-tests revealed participants reported a higher level of burning sensation under the electrodes in the active ($M = 2.08$, $SD = 1.27$) as compared to the sham session ($M = 1.62$, $SD = 0.98$), ($t(84) = 3.29$, $p < .01$).

Given that tVNS produced stronger burning sensations than sham stimulation, we ran additional statistical analysis to rule out the possibility that our results could be affected by acute stress experienced by the participants (Kimura et al., 2013). Therefore, we repeated the similar rmANOVA on k introducing amount (200 vs. 40,000€) and session (active vs. sham) as within-subjects factors, positive and negative mood group as between subject factors and the self-reported burning sensation in the active and sham sessions as covariates. We observed a similar pattern of results with the same three significant sources of variance: main effects of amount ($F(1,78) = 8.17$, $p < .01$, $\eta_p^2 = 0.09$, MSE = 0.01), positive mood group ($F(1,78) = 5.49$, $p = .02$, $\eta_p^2 = 0.06$, MSE = 0.03) as well as the significant interaction between session and positive mood group ($F(1,78) = 6.99$, $p = .01$, $\eta_p^2 = 0.07$, MSE = 0.01). The main effect of burning sensations during active ($F(1,78) = 0.01$, $p = .92$, $\eta_p^2 < 0.01$, MSE = 0.03) or sham sessions ($F(1,78) = 0.04$, $p = .84$, $\eta_p^2 < 0.01$, MSE = 0.03) were not significant, so we can conclude that the higher burning sensation reported in the active session did not explain our results.

No differences were observed with regard to other side-effects assessed (i.e. headache, neck pain, nausea, muscle contractions, stingy sensation, burning sensation or generic uncomfortable feelings) were found (all $p_s \geq .06$). As for the question which stimulation participants thought they had received, participants were more accurate, as reflected in accuracy percentages, in the active ($M = 49.41 \pm 5.45\%$) as compared to the sham session ($M = 29{,}41 \pm 4.97\%$), ($t(84) = -3.22$, $p < .01$).

4. Discussion

The main aim of the current study was to assess the effect of tVNS on delay discounting, taking into account the role of mood in self-control. In line with the somatic marker hypothesis (SMH; Bechara et al., 2005; Damasio et al., 1991, 1996) and on the basis of previous evidence of a role of mood in influencing self-control (Herman et al., 2018; Lerner et al., 2015; Mayer et al., 1992), we reasoned that the effect of mood on delay discounting may depend on afferent vagal signals carrying somatic marker signals. We assumed that, if so, the effect of tVNS delay discounting might be dependent on the mood state, with mood being commonly measured using the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988).

In line with the somatic marker hypothesis and the proposed role of the vagus nerve in carrying the somatic markers influencing economic decision-making (Bechara et al., 2005; Damasio et al., 1991, 1996), we observed that tVNS affects DD, making the discounting rate steeper, but only for people with lower positive mood. That is, in the low positive mood group only, active stimulation indeed increased k compared to both the sham condition and to those participants that reported high positive mood. This effect was observed regardless of the level of negative mood reported by the participants.

The interaction between mood and delay discounting has been already demonstrated, although evidence is still inconclusive. For example, positive mood has been associated with impulsive actions and increased risk taking, and negative mood seems to affects the tendency to act on impulses by inducing a more short-term focus (Cyders and Smith, 2007; Tice et al., 2001; Youn and Faber, 2000; Yuen and Lee, 2003). While positive affect increases our ability to wait for the gratification, making us more patient, negative affect is associated with increases in temporal impulsivity. The fact that we found a modulatory effect of tVNS on discounting rate only when in a positive mood, and selectively lower positive mood, might be due to two possible reasons. First, participants reported to be generally in a positive mood; the average level of reported positive mood was higher (almost double) than the average level of reported negative mood (see Table 2). Therefore, in other words, we speculate that the effect of negative mood was not evidenced because our sample of participants was not, in general, in a negative mood. Second, in other economic decision-making tasks such as the Iowa Gambling task, it was often reported that negative somatic states specifically relate to negative outcomes (Bechara et al., 2005). In our study, individuals had to process and evaluate only possible positive outcomes as no trials about avoiding punishments and/or losses occurred in the task, hence providing a possible explanation for the relevance of positive affective state.

In addition, if one considers the V-shaped relation between valence and arousal (Kuppens et al., 2013), our findings allow the possibility

that low positive mood is a signal of lower task-relevant arousal. Arousal is concomitant to activity of the locus coeruleus (LC) and the release of norepinephrine (NE), both known to be stimulated by tVNS (Ventura-Bort et al., 2018; Warren et al., 2019). In other words, tVNS might have increased arousal, although not necessarily task-relevant (i.e., still in the presence of low positive mood), possibly explaining the increase in DD. However, an alternative explanation for the increase in tVNS-induced discounting rate when participants were in a lower pleasant mood state may be that a tVNS-mediated increase in arousal helps to execute appropriate goal-relevant behavior (i.e., in this case, to choose immediate reward in order to maintain or improve the current positive mood). Previous theories of personality have addressed the relationship between impulsivity and arousal, suggesting that impulsivity is related to low level of arousal at rest and that impulsive individuals seek stimulation to obtain an optimal level of arousal (Barratt, 1985; Schmidt et al., 2013). Because increased arousal may affect impulse control (decreased in impulsivity), it seems plausible that the tVNS effect was only evident when the level of arousal (induced by positive mood) was low, consistent with the optimal level of arousal hypothesis (Schmidt et al., 2013). Altogether, this provides additional support for the fact that affective and physiological internal states impact self-control and decision making (Herman et al., 2018), and we therefore recommend future studies to evaluate the possibility that tVNS causes an increase in NE, physiological arousal, and signaling of somatic markers.

One might wonder whether positive mood modulates the effects of tVNS on discounting, or tVNS the effects of positive mood on discounting. In other words, which factor is causally involved and which may be mediating. First, our study does not allow inferring any suggestions as to the direction of these processes (i.e. to indicate cause and effect). In fact, the idea that there is one cause and one effect fuels discussion on the conceptual distinction between physiological and affective processes in “explaining” a cognitive outcome. As pointed out by Hommel (2019) in this special issue, when one process (e.g., mood) feeds into another process (e.g., tVNS-induced arousal) affecting a phenomenon (e.g., self-control), this neither provides evidence that the first process was a prerequisite for the other process, nor excludes the possibility that both processes contribute to each other or to other phenomena. In other words: “There is no reason to reserve a particular explanans for just one explanandum” (Hommel, 2019).

Besides the relationship between mood and arousal in delay discounting, additional personality state- and trait-dependent factors might be also coming into play (Cona et al., 2019; Herman et al., 2018), and interact with the modulating effect of tVNS.[2] For example, Cyders and Smith (2007, 2008), suggest the existence of two personality traits called positive and negative urgency, that refer to the tendency of engaging risky behaviors when experiencing strong positive and negative affect. Given that this positive urgency to act riskily when experiencing high positive mood can have negative consequences in the long-term (Cyders and Smith, 2008), the fact that in our study the effect of tVNS was only present when participants' mood was low, suggest tVNS as a useful tool to maximize the influence of affective states in adaptive behavior and self-control. Furthermore, the relationship between mood and DD is also modulated by gender, suggesting that situational variables affect discounting differently for males and females (Koff and Lucas, 2011). In our study, the distribution of males and females was more unbalanced in the low (31 females and 13 males) than in the high positive mood (21 females and 19 males), so we cannot rule out an explanation of the effects of tVNS also on these terms. Altogether, this leaves as an open question why the effect of tVNS on DD was specific of the low positive mood, and we encourage future studies to consider potential mediators in the relationship between DD and tVNS.

Furthermore, the current study is subjective to a number of limitations worth mentioning. First, we employed a task in which participants' evaluated monetary rewards to assess discounting behavior and index behavioral impulsiveness. However, it is well-known that other types of rewards (i.e., food, drugs of abuse, etc.) are discounted more steeply than monetary rewards (Estle et al., 2007; Odum, 2011a; Odum and Rainaud, 2003; Tsukayama and Duckworth, 2010), as such possibly leaving less variance to account for in the current study. In addition, participants were aware that the rewards of 200€ and 40,000€ reflected hypothetical rewards, what means that they were not actually receiving such rewards, which might have caused participants to perform differently from how they would act when confronted with such a choice in real life. Second, our measure of mood was self-reported and, therefore, subjective. Future research should employ objective measures of mood or for example, mood induction techniques to achieve more precise control of affect. Third, future studies should also include personality questionnaires to measure extraversion, a trait that seems to modulate the effects of mood on delay discounting (Hirsh et al., 2010). Finally, tVNS is assumed to enhance NE and GABA release in the brain (Colzato and Vonck, 2017; Ventura-Bort et al., 2018; Warren et al., 2019) but, however, we can only infer our arousal-increase interpretation based on our behavioral results. Recent findings suggest that tVNS can increase hormonal physiological markers of arousal (Warren et al., 2019) and given the relationship between hormonal markers and mood (Herrero et al., 2010; Ocampo Rebollar et al., 2017), it would be interesting to replicate the current findings but measuring salivary concentrations of alpha-amylase to correlate with discounting behavior in the active condition (Ventura-Bort et al., 2018; Warren et al., 2019).

To conclude, we observed that the vagus nerve enhances the discount rate, but only for individuals reporting a lower positive mood state. These findings may stimulate new research to further extend our understanding of the specific role of the vagus nerve in economic decision-making and self-control, and supports the idea that tVNS is a promising non-invasive brain stimulation technique for modulating mental processes in healthy humans (Van Leusden et al., 2015).

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Author contribution

LSC developed the idea for the study. The design of the study was developed by LS and LSC. LS designed the protocol and supervised data collection. LS and MJM performed the analysis, LS wrote the first draft of the article and MJM revised the article. All authors approved the last version of the manuscript.

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[2] As additional exploratory analysis, we ran the similar rmANOVA on k including amountand tVNS session as within subjects-factors, positive and negative mood as between-subject factors and the personality questionnaires (DAS-21, BIS-II and SRPQ) as covariates. Only the non-planning impulsiveness subscale of the BIS-II lead to a significant interaction with the session ($F(1,72) = 4.89$, $p = .03$, $\eta_p^2 = 0.06$, MSE = 0.01). Partial correlations controlling for positive mood between k (averaged for 200 and 40,000) for both active and sham sessions and the non-planning subscale of BIS (a tendency to ignore long-term consequences of actions and not to plan ahead), revealed, although non-significant, a trend towards a positive correlation between k during active tVNS and non-planning impulsiveness ($r = 0.20$, $p = .07$) while this relationship was not existing during sham stimulation ($r = 0.06$, $p = .61$).

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