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Differential effects of social versus monetary incentives on inhibitory control under acute inflammation

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ABSTRACT

While the impact of chronic, low-grade inflammation on cognitive functioning is documented in the context of neurodegenerative disease, less is known about the association between acute increases in inflammation and cognitive functioning in daily life. This study investigated how changes in interleukin-6 (IL-6) levels were associated with performance on an inhibitory control task, the go/no-go task. We further examined whether the opportunity to earn different incentive types (social or monetary) and magnitudes (high or low) was associated with differential performance on the task, depending on IL-6 levels. Using a within-participant design, individuals completed an incentivized go/no-go task before and after receiving the annual influenza vaccine. Multilevel logistic regressions were performed on the trial-level data (N_{obs} = 30,528). For no-go trials, we did not find significant associations in IL-6 reactivity and changes in trial accuracy between sessions. For go trials, we found significant differences in the associations between IL-6 reactivity and changes in accuracy as a function of the incentive condition. Notably, greater IL-6 reactivity was consistently associated with fewer omission errors (i.e., greater accuracy on go trials) on high-magnitude social incentives (i.e., viewing a picture of a close-other) when compared to both low-magnitude social and high-magnitude monetary incentives. Together, these results suggest that mild fluctuations in inflammation might alter the valuation of an incentive, and possibly a shift toward devoting greater attentional resources when a large social incentive is on the line. Overall, this study sheds light on how everyday, low-grade fluctuations in inflammation may influence cognitive abilities essential for daily life and effective inhibitory control.

Over recent decades, mounting evidence in both animal models and clinical studies has demonstrated that low-grade levels of inflammation are significantly associated with cognitive impairments (Lai et al., 2017; Su et al., 2019; Saleem et al., 2015). Indeed, chronic inflammation may expedite age-related neurodegenerative diseases (Gorelick, 2010; Bettcher & Kramer, 2014; Beydoun et al., 2019; Long et al., 2023), with one study demonstrating that increased levels of peripheral inflammation account for a 45 % increased risk for all-cause dementia (Koyama et al., 2013). While the association between chronic inflammation and cognitive impairment has received much attention in cross-sectional studies (Leonardo & Fregni, 2023), fewer studies have explored how acute increases in inflammation are related to changes in cognitive performance in humans. Of the few laboratory-based studies exploring these associations, there are mixed findings regarding whether acute increases in inflammation impair (Capuron et al., 2001; Meyers & Abbruzzese, 1992) or *improve* (Cohen et al., 2003; Grigoleit et al., 2011) performance on cognitive tasks. Thus, more mechanistic work is needed to delineate the effects of short-term, acute increases in inflammation on cognitive function.

One potential reason for the mixed findings in experimental studies exploring links between acute inflammation and cognitive function may be due to variability in the specific cognitive tasks employed. Within this literature, neuropsychological batteries are the most frequently used measures of cognitive function (for reviews see Fard et al., 2022; Bollen

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et al., 2017). These tests have the advantage of being well-validated and reliable, providing a comprehensive overview of an individual's cognitive profile across a variety of domains, including memory, attention, language, and executive functions. However, these tests are optimized to identify broad pathological impairments in cognition (e.g., following stroke, after a traumatic brain injury; Belleville et al., 2017), and they are consequently less effective at detecting more subtle or specific changes in cognitive functioning (Chaytor & Schmitter-Edgecombe, 2003) of the sort that are likely to accompany mild, acute increases in inflammation. This limitation is partly because neuropsychological batteries are designed with a focus on pathology, diagnosis, and assessment, relying on standardization and norm-based comparisons that may not be sensitive enough to capture minor within-person variations or subtle cognitive shifts associated with transient biological changes such as acute inflammation. While neuropsychological tests are important in aiding in more general clinical assessment, there is a need for studies that explore links between acute increases in inflammation and more fine-grained changes in cognitive function that may happen in everyday life.

One key facet of everyday cognitive function is executive functioning, or the ability to plan, adapt, maintain, and manipulate information to achieve goal-directed behaviors (Miyake et al., 2000; Miyake & Friedman, 2012; Gilbert & Burgess, 2008). Executive function involves several cognitive processes that help an individual navigate daily life such as planning a course of action, attending to relevant task demands while ignoring irrelevant information, adapting to unforeseen situations, or controlling impulsive behaviors that may not be appropriate in a given context. A core sub-component of executive functioning is inhibitory control (Diamond, 2013). Like the ability to brake suddenly if an animal runs in front of a car or suppress a response to electronic notifications while focusing on work, inhibitory control is important for completing everyday tasks. Specifically, it involves the suppression of prepotent but unwanted actions that may interfere with higher-order cognitive or motor goals. Deficits in inhibitory control are implicated in several neuropsychiatric disorders (Mostofsky & Simmonds, 2008), such as attention-deficit hyperactivity disorder (ADHD) and dementia (Migliaccio et al., 2020), and effective inhibitory control facilitates self-regulatory health behaviors related to diet, exercise, and stress management (Papies et al., 2008; Nederkoorn et al., 2010). As a sub-component of executive function that has important implications for daily functioning, health, and well-being, inhibitory control is thus a critical cognitive processes to examine when exploring the links between inflammation and cognitive function.

There are several prior studies that have examined the association between acute increases in inflammation, specifically the inflammatory cytokine interleukin-6 (IL-6), and inhibitory control. The first few studies in this area explored whether inducing inflammation using the typhoid vaccine (Brydon et al., 2008) or a lipopolysaccharide (LPS) injection (Grigoleit et al., 2010; van den Boogaard et al., 2010) was associated with changes in performance on the Stroop color-word naming test, a task commonly employed to probe inhibitory control. Generally, these studies reported no significant changes in accuracy on the Stroop task following inflammatory challenge. In a more recent study, Handke et al. (2020) explored whether an inflammatory challenge modulated performance on another inhibitory control task, the go/no-go task. Similar to studies using the Stroop task, this study did not find a significant difference in accuracy on a three-minute go/no-go task by injection type (i.e., LPS vs placebo), or as a function of IL-6 concentrations within the LPS condition. The lack of significant findings in these studies may be due to factors such as small sample sizes, a low number of trials probing inhibition (i.e., incongruent or no-go trials), or a low error rate on the tasks (i.e., ceiling effects). Recent work by Madison et al. (2023) overcame some of these limitations in a randomized crossover trial using the typhoid vaccine in a larger sample of women (N=171) who were postmenopausal breast cancer survivors. In this study, participants completed several inhibitory control tasks, including a 15-minute continuous performance task, similar to a go/no-go task. This study found no significant differences in task accuracy in the typhoid versus placebo condition. Thus far, the accumulated data provides no strong evidence to support a link between acute increases in inflammation and inhibitory control.

This absence of robust evidence from human studies contrasts with preclinical findings, which consistently suggest a connection between acute inflammation and cognitive impairments (Kaiser et al., 2014; Cunningham & Hennesey, 2015; Skelly et al., 2019; Savi et al., 2021). While the differences may be due to cross-species translational challenges, there may be other factors explaining the lack of evidence in human research. For example, the lack of significant findings in Madison et al. (2023) may be due to unique sample characteristics, such as the complex physiological and cognitive changes resulting from cancer treatment among the sample of breast cancer survivors (Janelsins et al., 2014). Or it could be that there are secondary factors that influence executive functioning indirectly. For example, as described below, the potential influence of motivation and incentives on task performance were not directly manipulated or measured in these studies, but could play a critical role in modulating inhibitory control (Kouneiher et al., 2009; Botvinick & Braver, 2015; Chiew & Braver, 2011; Padmala & Pessoa, 2010). Thus, while the current human literature does not find a consistent relationship between increases in inflammation and inhibitory control, sample characteristics and a failure to consider the role of motivation and incentive in contributing to task performance may be masking significant relationships.

Inhibitory control processes do not operate in isolation; motivational states can significantly influence inhibitory control performance via the modulation of effort allocation (e.g., how much effort one decides to expend on a task; Botvinick & Braver, 2015, Madan, 2017). Further, incentive type (e.g., food, money, social cues) and incentive magnitude (e.g., a larger or smaller sum of money) can significantly impact response inhibition and attentional mechanisms that may contribute to inhibitory control performance. While a recent *meta*-analysis reported that incentives generally improve inhibitory control (Burton et al., 2021), individual studies have also found that the balance and characteristics of an incentive differentially alters performance on inhibitory control tasks (e.g., Asci et al., 2019). For example, performance on inhibitory control tasks may decrease as the magnitude of the incentive increases (Freeman & Aron, 2016). At higher magnitudes, incentives may intensify difficulty in inhibiting a prepotent response, suggesting a conflict between a natural tendency to approach incentives and the task demands of withholding such approach responses (Verbruggen & McLaren, 2018). Overall, incentives can enhance or impair inhibitory control based on the context and nature of the incentives involved (Chiew et al., 2016; Geier & Luna, 2012; Wang et al., 2018).

Expanding upon the complexities of incentivized inhibitory control, physiological factors including inflammation may also shift the valuation of incentives (Vichaya & Dantzer, 2018) and thus influence incentivized inhibitory control. Inflammation is generally associated with reductions in motivation (Frenois et al., 2007; Shen et al., 1999; Eisenberger et al., 2010; for review see Boyle et al., 2023). These reductions in motivation are well-supported by preclinical literature demonstrating that inflammation in the periphery can access the brain to influence neural processes associated with motivated behavior (see Haroon et al., 2012 & Dooley et al., 2018 for reviews), particularly via changes in the dopaminergic system (see Felger & Treadway, 2017). However, mounting evidence provides more nuance regarding the relationship between inflammation and motivation (Draper et al., 2018), suggesting that both incentive magnitude and incentive type may modulate the link between acute inflammation and motivated behavior. For example, one pre-clinical study found that following an endotoxin challenge, rodents only chose to expend effort to obtain food incentives only when it was highly advantageous (i.e., when the magnitude of the potential gain was greatest; Vichaya et al., 2014). This was demonstrated by an overall reduction in energy expenditure among rodents

exposed to an inflammatory challenge (compared to those exposed to placebo), including less effort made to obtain "typical" food, but a relative increase in effort to obtain more calorie-rich food. These results have been recapitulated in humans (Lasselin et al., 2017), such that individuals exposed to an inflammatory challenge were more motivated to expend effort to obtain an incentive when the probability to win was greater. These experimental findings suggest that inflammation may alter the perceived value of an incentive, which could in-turn shift behavior.

Inflammation has also been demonstrated to alter sensitivity to incentives depending on whether the incentive is non-social (e.g., winning money) or social (e.g., receiving positive feedback from a stranger). For example, inflammation may decrease neural sensitivity to monetary incentives (Eisenberger et al., 2010; Burrows et al., 2021) but enhance incentive-related neural sensitivity to positive *social* cues (Inagaki et al., 2015; Muscatell et al., 2016; for review see Eisenberger et al., 2017). Furthermore, inflammation may motivate different behavior depending on the social relationship one has with the target (Hennessy et al., 2014; Eisenberger et al., 2017; Muscatell & Inagaki, 2021). Specifically, recent work suggests that higher levels of inflammation may incentivize affiliating with a close other (i.e., a high magnitude social incentive), more so than with a stranger (i.e., a low magnitude social incentive, Inagaki et al., 2015; Jolink et al., 2022). Altogether, this work prompts the question: does the magnitude and type of incentive interact with levels of inflammation to modulate inhibitory control?

Given these nuances in links between inflammation, cognitive function, and motivation, the current study examined whether increases in the inflammatory cytokine interleukin-6 (IL-6) in response to a mild inflammatory challenge were associated with change in performance on an incentivized inhibitory control task. To test this, participants were exposed to the influenza vaccine and completed a novel go/no-go task immediately before and one day after the vaccine. The influenza vaccine has been shown to reliably elicit a mild inflammatory response (Christian et al., 2013; Radin et al., 2021) and to drive subtle within-participant changes in affective processes, including mood and social behaviors (Kuhlman et al., 2018; Jolink et al., 2022; Feldman et al., 2023). In the current study, participants provided blood samples to measure circulating levels of IL-6 before and 24 h after the influenza vaccine. In this adaptation of the go/no-go task, we manipulated incentive magnitude and type by specifying at the start of each block of the task whether participants could earn one of four incentives: high social (i.e., time viewing a picture of their close other), low social (i.e., time viewing a picture of a stranger), high monetary (i.e., large magnitude monetary incentive), and low monetary (i.e., lower magnitude monetary incentive). In our analysis, we examined whether greater IL-6 reactivity to the influenza vaccine was associated with accuracy on the go/no-go task and whether the association was modulated by incentive type (social versus non-social) and incentive magnitude (low versus high).

1. Methods

1.1. Participants

Fifty-five undergraduate students (37 assigned female at birth; M_{age} = 20.06 years, SD_{age} = 1.34) at a southeastern university in the United States participated in the Inflammation and Cognitive, Affective, and Social Processes Project-2 (ICASP-2), a larger study examining the relationship between inflammatory reactivity to the influenza vaccine and cognitive, affective, and social processes. Participants primarily identified as White American (43 %), 30 % identified as Asian American, 9 % as Latinx or Latin American, 2 % as Black American, and 16 % identified as multiracial. Participants were recruited via postings to class listservs and on social media, in which they were first directed to an online eligibility questionnaire. Inclusion criteria were similar to prior studies using the influenza vaccine paradigm (Jolink et al., 2024; Jolink et al., 2022; Boyle et al., 2019; Kuhlman et al., 2018).

Participants were eligible for the study if they were between 18 and 25 years of age and had a non-familial close other in their lives whom they saw every day. To participate in the study, participants were asked to choose and provide four smiling photos of a close other who "is not a family member and who you see almost every day (close friend, roommate or romantic partner); this is someone in your life you can go to for help or for comfort." They were told that they would be asked questions about this person throughout the study. Individuals who reported that they did not have a non-familial close other that they saw daily in the pre-screening questionnaire were not eligible to participate in the study (N=34). Interested individuals were also excluded if they (a) had already received the annual influenza vaccine or had had influenza that season, (b) used tobacco products, (c) used mood or immune-altering medications (e.g., anti-depressants, antihistamines), (d) had a current psychiatric diagnosis or reported history of depression or anxiety, (e) had any major medical condition (e.g., diabetes, asthma), (f) had had Guillain-Barre Syndrome (GBS), (g) were allergic to the influenza vaccine or ingredients present in the vaccine (e.g., eggs), or (h) had a current illness. Because the study was conducted during the COVID-19 pandemic, participants were also screened for self-reported exposure to COVID-19 or any current respiratory symptoms. Among 268 individuals who expressed interest in our study, 127 were deemed ineligible, while 131 met the eligibility criteria. Of the eligible participants, 57were successfully enrolled and participated before the depletion of the flu vaccine supply. Of these participants, 55 advanced to the post-vaccine session, though two only completed the pre-vaccine session.

1.2. Experimental design

ICASP-2 data were collected in 2022 (see also Jolink et al., 2024). Eligible participants provided written informed consent and were scheduled for two consecutive morning in-lab study sessions, one the day of receiving the annual influenza vaccine and one the day after. Given evidence that IL-6 levels peak approximately 24 h after influenza vaccination (Radin et a., 2021), the post-vaccine session was scheduled to take place approximately 24 h after the pre-vaccine session. Before the pre-vaccine session, participants provided photos of one close other who they identified as a consistent support figure for them (see photo details below in the section titled "Measures"). Both sessions were similar in that participants completed several computer tasks and surveys and provided a blood sample to be assayed for levels of IL-6. The distinguishing feature between sessions was that at the end of the pre-vaccine session, participants received the 2022 influenza vaccine. The vaccine was a 0.5 mL single-dose of GSK's Flulaval Quadrivalent and included the following virus strains: A/California/07/2009 (H1N1), A/Texas/50/2012 (H3N2), B/Massachusetts/02/2012 (Yamagata lineage), B/Brisbane/60/2008 (Victoria lineage).

To compute the sample size for the ICASP-2 study, we conducted a power analysis using the 'mixedpower' package in R (Kumle et al., 2021), as outlined in Feldman et al. (2022). This analysis, based on effect sizes from similar influenza vaccine studies, allowed us to estimate several fixed effects and their interactions simultaneously. We based simulations on linear mixed model from Inflammation and Cognitive, Affective, and Social Processes Project-1 (ICASP-1) data, focusing on simulating power for 2- and 3-way interactions across various sample sizes. In addition, simple power analyses were conducted using G*power (Faul et al., 2007) to evaluate power for a regression model with the same predictors. These analyses suggested that a total sample size of 126 was needed to detect an effect size of $f^2 = 0.05$, and a size of 79 to detect an effect size of $f^2 = 0.08$, both with an alpha of 0.05 at 80 % power. Given that data for the ICASP-2 study are nested within participant, these sample sizes were considered conservative estimates. Despite aiming to recruit 100 participants, data collection was limited when university's student pharmacy ran out of influenza vaccines in April 2022. Within this timeframe, we collected complete data from 55

subjects. We believe this sample size is sufficient to detect small to medium effects, as supported by post-hoc power analyses on similar data (Feldman et al., 2023).

1.3. Materials and measures

1.3.1. Close other and stranger photos

We focused on non-familial relationships such as friends, roommates, and romantic partners for our study due to their relevance in the college-bound social circles of participants, all of whom were students, and the practical consideration that these peer relationships are volitionally formed and more likely to provide in-person support during the study. To ensure that participants selected individuals who were a supportive close other, two questions about the person selected were assessed in the lab: (1) "can you rely on this person for help if you have a serious problem?" and 2) "can you really count on this person to help you feel better when you are feeling generally down-in-the dumps?" On a 7-point response scale, responses to these two items ranged from 4.5 to 7 with a mean of 6.4. These data suggest participants selected highly-supportive close others. Because participants could select their choice of a support figure, the selections spanned various relationship types, with the majority choosing a close friend (N=41) or romantic partner (N=14). Prior to participant arrival, a photo of a smiling stranger was selected by the researcher that matched the close other's gender and ethnic presentation. This photo was selected to be used as the low-magnitude social incentive.

1.3.2. Inflammation

As described in further detail in Jolink et al., 2024, interleukin-6 (IL-6) was chosen as the primary inflammatory cytokine for our study because it has been consistently linked to behavioral and neural changes following immune challenge (DellaGioia & Hannestad, 2010; Inagaki et al., 2012; Kuhlman et al., 2018; Jolink et al., 2022; Lacourt et al., 2015; Madison et al., 2023; Wright et al., 2005) and is known to significantly increase around 24 h after the influenza vaccine (Christian et al., 2013; Tsai et al., 2005; Radin et al., 2021). Given the established focus on IL-6 in similar studies involving mild inflammatory challenges (Carty et al., 2006; Christian et al., 2011; Edwards et al., 2006; Segerstrom et al., 2012; Tsai et al., 2005), we aligned our research with existing literature to maintain continuity.

We examined inflammatory reactivity by comparing levels of IL-6 in dried blood spots before and after the vaccine. In previous studies examining within-subject variations in inflammation in response to the influenza vaccine, IL-6 exhibits consistent increases after vaccination (Jolink et al., 2022; Christian et al., 2013; Segerstrom et al., 2012; Tsai et al., 2005; Boyle et al., 2019; Kuhlman et al., 2018; 2020; Radin et al., 2021). Additionally, dried blood spot assays for IL-6 have been validated against venipuncture blood serum samples (McDade et al., 2021; Miller & McDade, 2012). At both dried blood spot collections, approximately 20uL of blood was collected by finger prick using Neoteryx's Mitra Clamshell devices (https://www.neoteryx.com/mitra-clamshell-blood-collection-device). Samples were dried overnight and stored in a −80 freezer until study completion. Assays were conducted in triplicate using a high-sensitivity ELLA immunoassay platform to assess IL-6 levels. All samples contained detectable concentrations of IL-6 ranging from 0.56–2.37 pg/mL pre-vaccine and 0.59–2.4 pg/mL post-vaccine. Across all assay plates, the intra-assay coefficient of variation (CV) was 5.8 % and the inter-assay coefficient of variation was < 12 %. Three IL-6 values were more than 3 SDs above the mean; those values were winsorized and retained in the data for analyses. All IL-6 values were log-transformed to adjust for a positive skew in the data.

1.3.3. Go/no-go task

We used a novel version of the well-validated go/no-go task to measure executive function, specifically response inhibition and attention (Iaboni et al., 1995; Nosek & Banaji, 2001, Laurenson et al., 2015). In this task, participants are instructed to respond to a more frequently presented stimulus (go trials) while withholding a response to a less frequently presented stimulus (no-go trials). The task thus evaluates an individual's ability to inhibit the automatic tendency to press a button (i.e., the prepotent response) when encountering a no-go cue, as well as their ability to quickly and accurately respond when encountering a go cue. In the present study, participants completed an incentivized go/no-go task wherein they were instructed to press the 'J' key using their dominant hand as fast as possible when a blue circle appeared on the screen (go trials). However, they had to inhibit the pre-potent "go" response and NOT press the key when an orange circle appeared on the screen (no-go trials). Each participant completed 288 trials per session, of which 83.33 % (N=240) were go trials and 16.67 % (N=48) were no-go trials. To promote sustained attention and effort throughout the task, trial length varied between 250–500 ms by 50 ms intervals (Benikos et al., 2013) such that the trials advanced on their own if participants did not respond in time.

There were 36 trials per block, and each of the four block types (i.e., high monetary, low monetary, high social, low social) was administered twice (see Fig. 1 for task diagram). Blocks were presented in randomized order across participants and sessions. At the start of each block of the task, participants were informed whether accurate performance on that block would earn them money (i.e., monetary condition) or time viewing an image of a person (i.e., social condition). The magnitude of the incentive to be earned also varied across blocks. For the monetary incentive blocks, participants could either win $0.08 (high-monetary block) or $0.01 (low-monetary block) for correctly pressing for go cues or $0.25 (high-monetary block) or $0.03 (low-monetary block) for correctly withholding a response for no-go cues. For the social incentive blocks, participants could win time viewing a photo of their close other (high-social block) or a stranger (low-social block). For correctly pressing during go cues, participants earned 150 ms of photo viewing, and for correctly withholding a response for no-go cues, participants earned 500 ms of photo viewing. Participants received feedback on their performance at the end of each incentive block; they were shown the total money they earned for that block or the total time they could view the picture of the close other or stranger. Participants could earn a maximum of $0.96 (M=0.72, SD=0.08) for the low money condition, a maximum of $7.80 (M=6.05, SD=0.63) for the high money condition, a maximum of 13.75 s (M=11.25, SD=1.24) viewing an image of a stranger, or a maximum of 13.75 s (M=11.25, SD=1.04) viewing an image of their close other. At the end of the study, all participants received the same monetary bonus for participation on the incentivized go/no-go task. Following prior data cleaning practices (e.g., Freeman & Aron, 2016), trials for which responses were made in less than 100 ms were removed from analyses ($N_{no\text{-}go}$ = 105; N_{go} = 793; from N=55 participants). To assess task compliance, we also examined whether participants performed worse than chance on the task. We found that no participant performed lower than 50 % on the task during either session, with mean accuracies significantly above chance ($p < 0.001$, mean = 78 %, SD=5.80 %).

To assess the reliability of our incentivized go/no-go task, we conducted a split-half reliability analysis. Based on recommendations for robust estimates of reliabilities (Pronk et al., 2022), the task was divided into two halves based on odd and even numbered trials within each session and incentive condition, for go and no-go trials separately. We aggregated the scores to create a consolidated dataset where each row represented the mean accuracy scores for the odd and even halves for each participant, trial type, and session. This was then used to compute the Spearman correlation between the two halves for each task and session combination utilizing the Spearman-Brown prophecy formula (for discussion see de Vet et al., 2017). We found that reliabilities ranged from 0.76 to 0.90 for the task (see Supplemental Table S1), suggesting that there was moderate to high reliability.

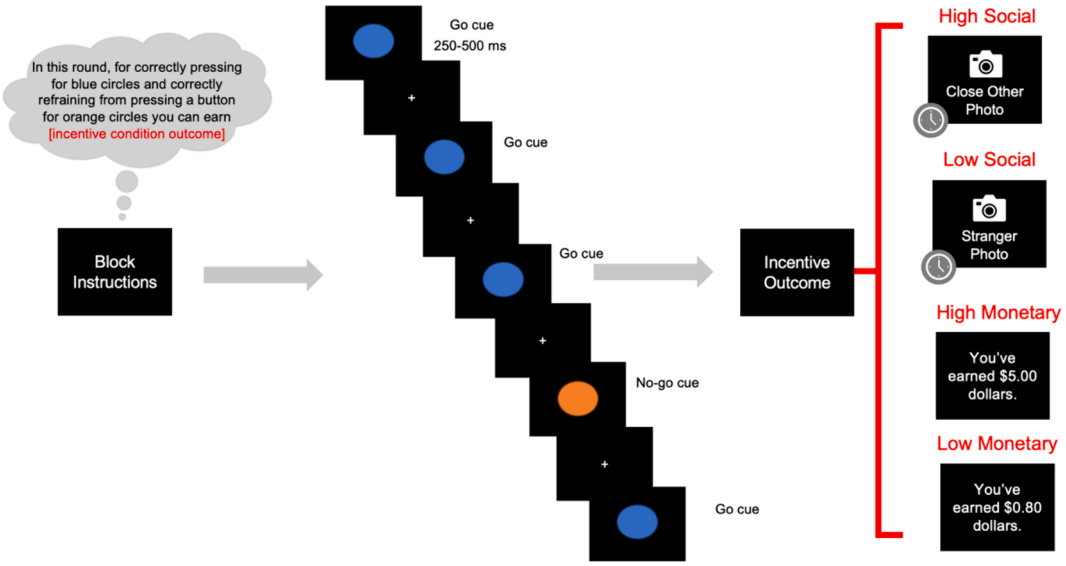

Fig. 1. Incentivized go/no-go task diagram. This diagram depicts an example sequence for one block of the incentivized go/no-go task. In each session (i.e., pre- and post-vaccine), there were 36 trials per block, and each of the four block types (i.e., high monetary, low monetary, high social, low social) were administered twice. Trials were timed and the duration ranged from 250 to 500 ms. Blocks were presented in randomized order for participants. Each participant completed 288 trials per session, of which 83.33 % (N=240) were go trials and 16.67 % (N=48) were no-go trials.

1.4. Analytic strategy

Data were analyzed to explore the association between IL-6 and within-person changes in go/no-go task accuracy before and after the influenza vaccine. Multilevel models were employed to assess these relationships using the 'lme4' package (Bates et al., 2015), and hypothesis testing for the fixed effects was performed using the 'lmerTest' package (Kuznetsova et al., 2017) in R (Version 4.3.3; R Core Team, 2024) with level for significance set at $p < 0.05$. To harness the full potential of our data, multilevel logistic regression models were performed to compute the likelihood of a correct response on each trial (N_{obs} = 30,528). This approach avoids aggregation and allows us to model trial performance nested within the same participant to maximize power. Given that our outcomes of interest were binary (i.e., accuracy on no-go trials, accuracy on go trials), logistic regressions were run using the 'glmer' function, while the 'performance' and 'DHARMa' packages were utilized to assess model fit and diagnostics. Our models were fit using a binomial distribution to accommodate the binary outcome (i.e., trial accuracy), with the bobyqa optimizer for parameter estimation. As recommended for logistic regression (Bauer & Sterba, 2011), numerical integration was approximated using an adaptive Gauss-Hermite quadrature (i.e., nAGQ=1) to derive robust estimates for complex models with multiple random effects and interactions. Our main analyses focused on examining whether IL-6 reactivity was associated with differences in trial accuracy as a function of incentive type (i.e., monetary or social) or incentive magnitude (i.e., high or low) between the two time points. Additionally, we also assessed whether IL-6 reactivity was associated with differences in trial accuracy by magnitude within incentive type (i.e., high social versus low social; high money versus low money) between the two sessions.

For our two primary outcomes, no-go trial accuracy and go trial accuracy, we completed the following series of steps (see Supplemental Table S2 for full model details). We began analyses by computing an unconditional mean model[1] to assess the average accuracy across participants, excluding the influence of time. The intraclass correlation coefficient (ICC) was calculated on the unconditional model to partition the variance into between-person (level-2) and within-person (level-1) components and to assess the degree of clustering ($ICC_{no\text{-}go}$ = 0.12; ICC_{go} = 0.05). An unconditional linear model followed[2] where we tested a model that added a random slope for session. The first session (pre-vaccine) served as the referent condition and was coded as 0. This model adjustment provided a better fit to the data than a model with only a random intercept, as indicated by a significant chi-square test (χ^2 = 48.527, df = 2, $p < 0.001$).

Next, we incorporated incentive-related factors as level-1 predictors, given that each participant completed all incentive conditions.[3] Factor variables indicating incentive type (i.e., social or monetary) and incentive magnitude (i.e., low or high) were added as fixed effects and interacted with the session variable time to examine their potential effects on individual baseline performance and change between sessions. Including the incentive condition variables and their interaction with session provided a better fit to the data than the unconditional model with random intercept and random slope for session (χ^2 = 22.906, df = 6, $p < 0.001$). Given recommendations to include random effects for task conditions to mitigate the risks of type 1 error (Barr et al., 2013), we also added random slopes of incentive condition to our model.[4] This addition provided a better fit to the data (χ^2 = 94.945, df = 7, $p < 0.001$) than the previous model, which only included random slope for session. Thus, our final models included random intercepts for participant ID and random slopes for session and the relevant incentive condition.

Finally, we incorporated the time-varying covariate—namely, IL-6 values at each session (Curran & Bauer, 2011). IL-6 values were session mean-centered to more precisely assess how deviations before and after the vaccine were associated with changes in task accuracy. This approach (Biesanz et al., 2004) accounts for and isolates the effects of IL-6 in light of its expected overall increase following vaccination. The key aspect of our model is the interaction between session and session mean-centered IL-6, which helps us discern whether variations in IL-6 levels, above or below the session norm, are associated with change in task accuracy from Session 1 to Session 2. This interaction term can be interpreted as representing a significant association between change in

[1] Model 1: Trial Accuracy ~ (1|ID).

[2] Model 2: Trial Accuracy ~ Session + (1+Session|ID).

[3] Model 3: Trial Accuracy ~ Sociality*Magnitude*Session + (1+Session|ID).

[4] Model 4: Trial Accuracy ~ Sociality*Magnitude*Session + (1+Session+Sociality+Magnitude|ID).

task performance and change in IL-6 insofar as a significant and positive effect would represent an increase in IL-6 from session 1 to session 2 greater than 0. Thus, while we report all effects, the discussion of our findings will center on probing interactions between session and session mean-centered IL-6.

Given the complexity of the interactive effects (i.e., cross-level, four-way interactions) and to facilitate interpretation, we computed separate models for each incentive dimension. For example, we first report associations for low magnitude trials to explore differences between low social incentives (picture of stranger) and low monetary (less than $1). The same was conducted among high magnitude trials. In follow-up analyses, we investigated the influence of incentive magnitude *within* social and monetary incentive types. For example, we explored the effects of magnitude within social trials (i.e., high social vs low social) and within monetary trials (i.e., high money vs low money). Significant 2- and 3-way interactions were probed using the 'interactions' package using simple slopes analyses. Additionally, relationship type, body mass index (BMI),[5] and sex assigned at birth[6] were included as covariates, as recommended by O'Connor et al. (2009). Continuous predictors were grand mean-centered to facilitate the interpretation of fixed effects.

2. Results

2.1. Assessing IL-6 reactivity to the influenza vaccine

First, we assessed whether there was a significant increase in IL-6 from before to after the vaccine. As reported in Jolink et al., (2024), we found that there was a significant difference in IL-6 between the pre- and post-vaccine sessions. Concentrations of log-transformed IL-6 (pg/mL) were significantly higher after compared to before the influenza vaccine; $t(54) = 2.435$, $p = 0.018$, 95 % CI [0.008, 0.088].

2.2. Effects of IL-6 reactivity on No-Go trial accuracy

2.2.1. Time-varying IL-6 effect on low incentive magnitude no-go trials

To compare whether no-go accuracy changed between sessions as a function of IL-6 among low magnitude trials, we examined the interactions between session, IL-6, and incentive type (i.e., low social vs low monetary). There was no main effect or interaction effects with IL-6 on low incentive magnitude no-go trials ($p > 0.05$; see Table 1 for full model details).

2.2.2. Time-varying IL-6 effect on high incentive magnitude no-go trials

Next, to compare whether no-go accuracy changed between sessions and as a function of IL-6 among high magnitude trials, we examined the interactions between session, IL-6, and incentive type (i.e., high social vs high monetary). There was no main effect or interaction effects with IL-6 on high incentive magnitude no-go trials ($p > 0.05$; see Table 1 for full model details).

2.2.3. Time-varying IL-6 effect on social incentive no-go trials

To compare whether no-go accuracy changed between sessions and as a function of IL-6 among social trials, we examined the interactions between session, IL-6, and social incentive magnitude (i.e., high social vs low social). This model showed no significant main or interaction effects with IL-6 ($p > 0.05$; see Table 1 for full model details).

2.2.4. Time-varying IL-6 effect on monetary incentive no-go trials

To compare whether no-go accuracy changed between sessions and as a function of IL-6 among monetary trials, we examined the interactions between session, IL-6, and incentive magnitude (i.e., high monetary vs. low monetary). This model showed no significant main or interaction effects with IL-6 ($p > 0.05$; see Table 1 for full model details).

2.3. Effects of IL-6 reactivity on go trial accuracy

2.3.1. Time-varying IL-6 effect on low incentive magnitude go trials

Paralleling the approach for no-go trials, we conducted a series of analyses subset by incentive types for go trial accuracy. First, we focused on the low magnitude trials and examined the interactions between session, IL-6, and incentive type (i.e., low social vs low monetary). There was no main effect of IL-6 on go trial accuracy ($p > 0.05$; see Table 2 for full model details). However, there was a significant interaction between session and IL-6 and incentive type (log odds = -2.06, SE=0.832, $p = 0.013$); see Fig. 2.

For low monetary trials, simple slopes analyses revealed that the slope of session was significantly positive for those with average (log odds = 0.31, SE=0.08, $p < 0.001$) and higher than average levels of IL-6 at session 2 (log odds = 0.40, SE=0.12, $p < 0.001$), but not among those with lower than average session 2 IL-6 levels (log odds = 0.21, SE=0.12, $p = 0.12$). These results suggest that participants with greater IL-6 reactivity were more likely to correctly respond on low monetary incentive go trials (see Fig. 2).

In contrast, on low social trials, simple slopes analyses did not find significant differences between sessions for lower than average (log odds = 0.16, SE=0.12, $p = 0.20$), average (log odds < -0.001, SE=0.08, $p = 0.96$), or higher than average levels of IL-6 at session 2 (log odds = -0.17, SE=0.12, $p = 0.17$). These findings indicate that, unlike the low monetary condition, accuracy during the low social incentive condition remained stable across sessions regardless of IL-6 reactivity.

2.3.2. Time-varying IL-6 effect on high incentive magnitude go trials

Next, to compare whether go accuracy changed between sessions and with IL-6 among high magnitude trials, we examined the interactions between session, IL-6, and incentive type (i.e., high social vs high monetary). There was no main effect of IL-6 on go trial accuracy ($p > 0.05$; see Table 2 for full model details). However, there was a significant interaction between session and IL-6 and incentive type (log odds = 1.64, SE=0.819, $p = 0.046$).

For high monetary trials, simple slopes analyses revealed that the slope of session was significantly positive for those with average (log odds = 0.17, SE=0.08, $p = 0.04$) and lower than average levels of IL-6 at session 2 (log odds = 0.25, SE=0.12, $p = 0.04$), but there was no significant effect of session at high levels of IL-6 (log odds = 0.08, SE=0.12, $p = 0.48$). These results suggest that participants with average and lower than average IL-6 reactivity were more likely to respond correctly on high monetary incentive go trials (see fig. 3), whereas those with higher-than-average IL-6 reactivity were not.

For high social trials, simple slopes analyses revealed that the slope of session was significantly positive for those with average (log odds = 0.23, SE=0.08, $p = 0.01$) and higher than average levels of IL-6 at session 2 (log odds = 0.36, SE=0.12, $p < 0.001$), but not among those with lower than average levels of IL-6 (log odds = 0.11, SE=0.13, $p = 0.39$). These results suggest that participants with average and higher than average IL-6 reactivity were more likely to respond correctly on high social incentive go trials.

[5] The accuracy of BMI measurement can be influenced by many factors including age, sex, race/ethnicity, muscle mass, and readers should interpret this measure with caution. For more information on considerations of BMI metrics, see Stanford et al. (2019) or Heymsfield et al. (2016).

[6] The assessment of sex and gender in this study were not consistent with current guidelines and recommendations on bias-free language on gender from the American Psychological Association (2020). Participants were asked "What is your biological sex? Please respond based on anatomy as your answer prompts questions relevant to biological processes (e.g., menstrual cycle)." Response options included "Male", "Female", and "Let me specify." Given the phrasing of this question, we interpret participants' response as best reflecting their sex assigned at birth (predicated on their genitalia, chromosomes, anatomical structures, or phenotype) which is different from their gender. Thus, we cannot identify whether individuals in this sample identified as cisgender, transgender, or non-binary. None of our participants self-specified "intersex.".

Table 1
Predicted Accuracy for No-Go Trials by Incentive Condition.

	Low Incentive Trials				**High Incentive Trials**				**Social Incentive Trials**				**Monetary Incentive Trials**			
Predictors	*Odds Ratios*	*Statistic*	*CI*	*p*	*Odds Ratios*	*Statistic*	*CI*	*p*	*Odds Ratios*	*Statistic*	*CI*	*p*	*Odds Ratios*	*Statistic*	*CI*	*p*
(Intercept)	**3.73**	**3.61**	**1.826 – 7.632**	**0.00**	**2.80**	**3.50**	**1.575 – 4.984**	**0.00**	**2.46**	**2.98**	**1.360 – 4.455**	**0.00**	**2.89**	**3.04**	**1.458 – 5.743**	**0.00**
Session	0.92	−0.56	0.679 – 1.240	0.58	1.02	0.15	0.767 – 1.362	0.88	0.81	−1.46	0.616 – 1.073	0.14	0.97	−0.20	0.730 – 1.292	0.84
IL-6	1.41	0.34	0.194 – 10.282	0.73	1.05	0.06	0.211 – 5.195	0.95	1.27	0.29	0.256 – 6.316	0.77	1.29	0.28	0.213 – 7.791	0.78
Session:IL-6	3.41	0.92	0.249 – 46.881	0.36	1.28	0.20	0.108 – 15.161	0.84	1.45	0.29	0.118 – 17.777	0.77	3.53	0.97	0.275 – 45.227	0.33
Sociality	0.81	−1.55	0.616 – 1.058	0.12	0.80	−1.73	0.614 – 1.030	0.08								
Sociality:Session	0.84	−0.93	0.584 – 1.211	0.35	1.02	0.11	0.710 – 1.467	0.91								
Sociality:IL-6	1.10	0.10	0.160 – 7.619	0.92	1.10	0.10	0.165 – 7.375	0.92								
Sociality:Session:IL-6	0.54	−0.41	0.028 – 10.442	0.68	3.20	0.77	0.164 – 62.693	0.44								
Magnitude									1.03	0.25	0.799 – 1.335	0.80	1.07	0.48	0.811 – 1.412	0.63
Magnitude:Session									1.24	1.19	0.870 – 1.771	0.23	1.00	−0.01	0.689 – 1.447	0.99
Magnitude:IL-6									1.01	0.01	0.156 – 6.495	1.00	0.94	−0.06	0.131 – 6.815	0.96
Magnitude:Session:IL-6									2.81	0.69	0.149 – 52.809	0.49	0.53	−0.41	0.025 – 11.226	0.68
Relationship Type	0.75	−1.11	0.458 – 1.243	0.27	0.89	−0.55	0.586 – 1.349	0.58	0.90	−0.47	0.573 – 1.405	0.64	0.85	−0.71	0.530 – 1.347	0.48
BMI	1.00	−0.07	0.938 – 1.061	0.95	0.99	−0.31	0.942 – 1.044	0.75	0.99	−0.56	0.934 – 1.039	0.58	1.00	−0.02	0.943 – 1.059	0.98
Age	1.05	0.56	0.891 – 1.229	0.58	1.04	0.56	0.904 – 1.198	0.58	1.00	−0.05	0.865 – 1.147	0.96	1.13	1.59	0.972 – 1.319	0.11
Sex	0.87	−0.60	0.546 – 1.378	0.55	1.06	0.29	0.722 – 1.546	0.78	0.96	−0.19	0.652 – 1.422	0.85	1.01	0.03	0.640 – 1.582	0.98
Random Effects																
σ^2	3.29				3.29				3.29				3.29			
τ_{00}	0.81 $_{ID}$				0.24 $_{ID}$				0.35 $_{ID}$				0.66 $_{ID}$			
τ_{11}	0.21 ID.session				0.13 ID.session				0.16 ID.session				0.09 ID.session			
	0.03 ID.Sociality				0.01 ID.Sociality				0.02 ID.Magnitude				0.06 ID.Magnitude			
ρ_{01}	−0.62				0.25				0.13				−0.26			
	−0.93				−0.29				−0.64				−0.88			
Observations	2431.00				2457.00				2432.00				2456.00			
Marginal R^2	0.02				0.01				0.01				0.01			
AIC	2903.96				2899.68				3004.21				2809.89			
log-Likelihood	−1433.98				−1431.84				−1484.11				−1386.95			

Table 2
Predicted Accuracy for Go Trials by Incentive Condition.

	Low Incentive Trials				**High Incentive Trials**				**Social Incentive Trials**				**Monetary Incentive Trials**			
Predictors	*Odds Ratios*	*Statistic*	*CI*	*p*	*Odds Ratios*	*Statistic*	*CI*	*p*	*Odds Ratios*	*Statistic*	*CI*	*p*	*Odds Ratios*	*Statistic*	*CI*	*p*
(Intercept)	**2.78**	**4.50**	**1.782 – 4.343**	**<0.001**	**3.22**	**6.00**	**2.199 – 4.722**	**<0.001**	**3.17**	**4.85**	**1.988 – 5.043**	**<0.001**	**2.66**	**5.02**	**1.817 – 3.902**	**<0.001**
Session	**1.36**	**3.76**	**1.158 – 1.593**	**<0.001**	**1.18**	**2.03**	**1.005 – 1.387**	**0.04**	1.03	0.33	0.881 – 1.194	0.74	**1.40**	**4.15**	**1.196 – 1.648**	**<0.001**
IL-6	0.40	−1.66	0.136 – 1.180	0.10	1.16	0.28	0.402 – 3.367	0.78	0.96	−0.09	0.352 – 2.594	0.93	0.49	−1.38	0.178 – 1.346	0.17
Session:IL-6	2.13	1.08	0.539 – 8.437	0.28	0.52	−0.92	0.132 – 2.070	0.36	0.26	−1.94	0.065 – 1.015	0.05	2.09	1.05	0.524 – 8.338	0.30
Sociality	1.17	1.57	0.962 – 1.422	0.12	0.99	−0.09	0.839 – 1.174	0.93								
Sociality:Session	**0.73**	**−3.25**	**0.607 – 0.884**	**0.00**	1.07	0.71	0.886 – 1.292	0.48								
Sociality:IL-6	2.45	1.46	0.735 – 8.189	0.14	0.38	−1.71	0.123 – 1.154	0.09								
Sociality:Session: IL-6	**0.13**	**−2.47**	**0.025 – 0.652**	**0.01**	**5.13**	**1.97**	**1.008 – 26.135**	**0.05**								
Magnitude									0.96	−0.40	0.803 – 1.156	0.69	1.13	1.58	0.971 – 1.322	0.11
Magnitude:Session									**1.24**	**2.26**	**1.029 – 1.500**	**0.02**	0.85	−1.67	0.709 – 1.028	0.10
Magnitude:IL-6									0.36	−1.73	0.112 – 1.148	0.08	2.28	1.51	0.781 – 6.646	0.13
Magnitude: Session:IL-6									**8.98**	**2.64**	**1.764 – 45.664**	**0.01**	0.25	−1.72	0.053 – 1.208	0.09
Relationship Type	1.15	0.83	0.830 – 1.587	0.41	1.01	0.04	0.754 – 1.340	0.97	1.21	1.08	0.859 – 1.689	0.28	1.03	0.19	0.770 – 1.373	0.85
BMI	0.98	−0.77	0.946 – 1.025	0.44	0.99	−0.40	0.956 – 1.030	0.69	**0.96**	**−2.12**	**0.917 – 0.997**	**0.03**	1.00	0.23	0.970 – 1.039	0.82
Age	1.05	0.86	0.944 – 1.158	0.39	1.04	0.76	0.945 – 1.135	0.45	1.02	0.37	0.917 – 1.136	0.71	1.05	1.05	0.959 – 1.149	0.29
Sex	1.20	1.22	0.894 – 1.615	0.22	1.21	1.43	0.933 – 1.557	0.15	1.20	1.15	0.879 – 1.639	0.25	1.26	1.80	0.979 – 1.622	0.07
Random Effects																
σ^2	3.29				3.29				3.29				3.29			
τ_{00}	0.27 $_{ID}$				0.22 $_{ID}$				0.34 $_{ID}$				0.24 $_{ID}$			
τ_{11}	0.10 ID.Session				0.11 ID.Session				0.10 ID.Session				0.10 ID.Session			
	0.30 ID.Sociality				0.16 ID.Sociality				0.22 ID.Magnitude				0.11 ID.Magnitude			
ρ_{01}	−0.56				−0.60				0.07				−0.33			
	−0.27				−0.24				−0.61				−0.45			
Observations	12,057				12,120				12,057				12,120			
Marginal R^2	0.01				0.01				0.02				0.01			
AIC	11752.48				11588.47				11570.51				11762.51			
log-Likelihood	−5858.24				−5776.24				−5767.25				−5863.25			

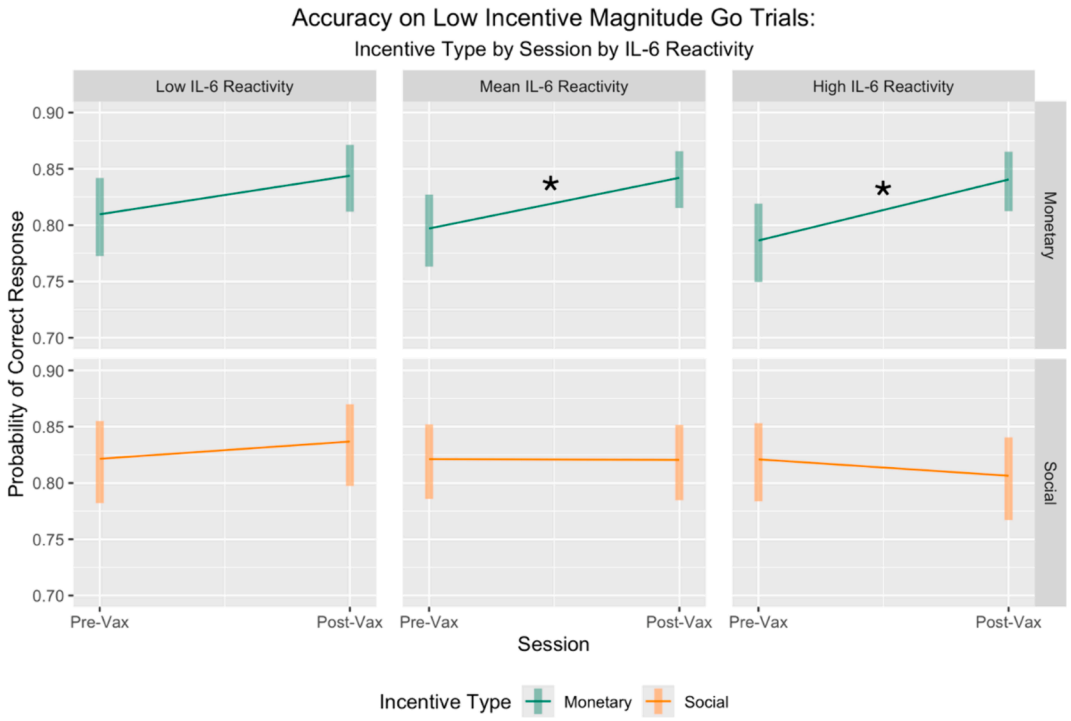

Fig. 2. Accuracy on Low Incentive Magnitude Go Trials. The graph displays the probability of correct responses among all low incentive magnitude go trials. Results are plotted for the two incentive types, monetary (green) and social (orange), and are stratified by levels of IL-6 reactivity (lower than average, average, and higher than average). Data are shown for both pre-vaccination and post-vaccination sessions. The asterisks represent a significant change in accuracy from the pre-vaccine session to the post-vaccine session.

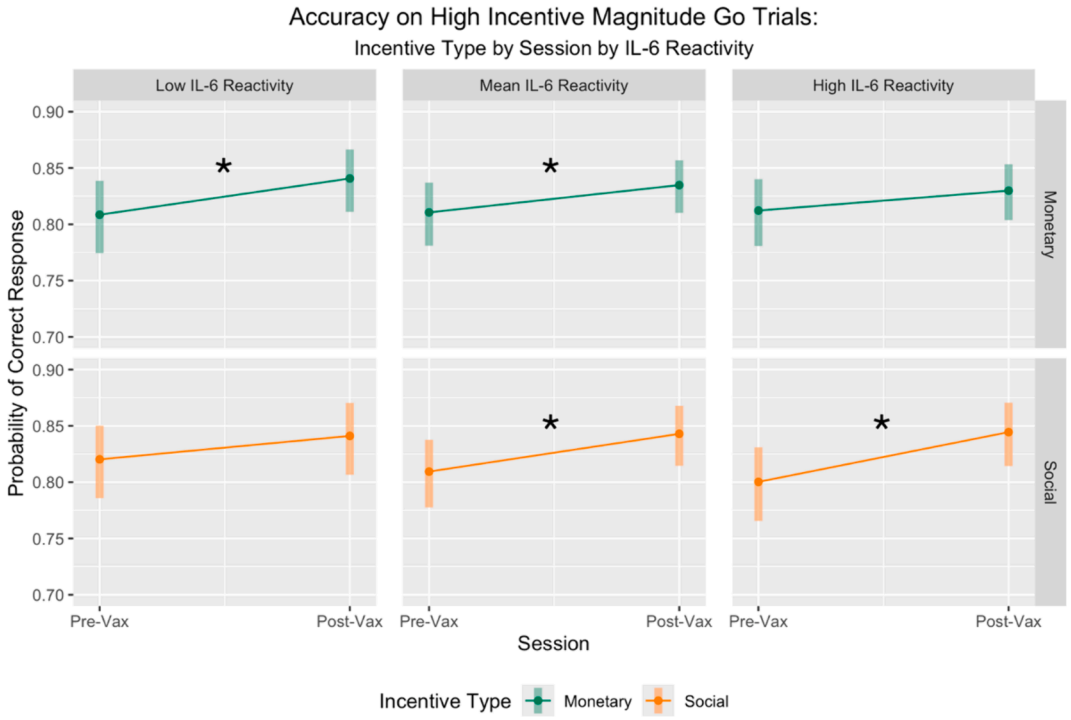

Fig. 3. Accuracy on High Incentive Magnitude Go Trials. The graph displays the probability of correct responses among all high incentive magnitude go trials. Results are plotted for the two incentive types, monetary (green) and social (orange), and are stratified by levels of IL-6 reactivity (lower than average, average, and higher than average). Data are shown for both pre-vaccination and post-vaccination sessions. The asterisks represent a significant change in accuracy from the pre-vaccine session to the post-vaccine session.

2.3.3. Time-varying IL-6 effect on social incentive go trials

To compare whether go accuracy changed between sessions and with IL-6 reactivity among social incentives, we examined the interactions between session, IL-6, and incentive magnitude (i.e., high vs low). There was no main effect of IL-6 reactivity on overall task accuracy ($p > 0.05$; see Table 2 for full model details). However, there was a significant session x IL-6 x magnitude interaction (log odds = 2.176, SE=0.840, $p = 0.010$).

For high social incentive trials, simple slopes analyses revealed that the slope of session was significant at average (log odds = 0.24, SE=0.08, $p < 0.001$) and higher than average levels of IL-6 (log odds = 0.35, SE=0.11, $p < 0.001$), but not at lower than average levels of IL-6 (log odds = 0.13, SE=0.12, $p = 0.26$). These results suggest that participants with average and higher than average IL-6 reactivity were more likely to correctly respond on high social incentive go trials (see fig. 4).

Conversely, simple slopes analyses for the low social incentive trials did not find significant differences between sessions for lower than average (log odds = 0.20, SE=0.12, $p = 0.09$), average (log odds = 0.03, SE=0.08, $p = 0.74$), or higher than average levels of IL-6 (log odds = -0.15, SE=0.12, $p = 0.21$). These findings indicate that, unlike the high social incentive condition, performance in the low social condition remained stable across sessions regardless of IL-6 reactivity.

2.3.4. Time-varying IL-6 effect on monetary incentive go trials

Finally, we examined the interactions between session, IL-6, and incentive magnitude (i.e., high vs low) among monetary trials. There was no main or interaction effects with IL-6 ($p > 0.12$; see Table 2 for full model details).

3. Discussion

While the association between chronic low-grade inflammation and cognition has received attention, comparatively fewer studies have explored the effect of acute increases in inflammation on cognitive function in humans. In the current study, we examined how acute changes in the inflammatory cytokine interleukin-6 (IL-6), following a mild inflammatory challenge (i.e., the influenza vaccine), were associated with inhibitory control. Utilizing a within-subjects design, we assessed whether accuracy on an incentivized go/no-go task differed after receiving the influenza vaccine, and whether this effect varied by IL-6 reactivity to the vaccine, incentive magnitude (low versus high) and incentive type (social versus monetary). We did not find evidence that IL-6 reactivity to the vaccine was significantly associated with change in errors of commission (i.e., failure to omit response on no-go trials). However, we did find that individuals with higher IL-6 reactivity demonstrated fewer errors of omission (i.e., failures to respond on go trials) after the vaccine, specifically in the high-magnitude, social incentive condition (i.e., viewing an image of a smiling close other) as compared to both low-magnitude, social (i.e., viewing an image of a smiling stranger) and high-magnitude, monetary conditions. Together, these results suggest that greater IL-6 reactivity to the vaccine is associated with improved performance in specific incentive contexts on an inhibitory control task. To our knowledge, this is the first laboratory-based demonstration of inflammation-related changes in performance on an incentivized inhibitory control task. This is important given that inhibitory control is a critical skill for navigating daily life with implications for self-regulatory health behaviors (Hall et al., 2008; Daly et al., 2014; Reimann et al., 2020).

Theoretical assertations suggest that inflammation is associated with self-regulation difficulties (Shields et al., 2017, cf. Madison & Kiecolt-Glaser, 2022), in part because of empirical research demonstrating that higher levels of inflammation are associated with more present-focused and impulsive decisions (Gassen et al., 2019a, Gassen et al., 2019b). However, we did not find evidence to support that hypothesis. Specifically, we did not observe that acute increases in IL-6 following the vaccine were associated with a change in errors of commission, or failures to inhibit a prepotent response to no-go trials. This finding is aligned with previous studies that also report a null relationship between acute increases in IL-6 and response inhibition (Brydon et al.,

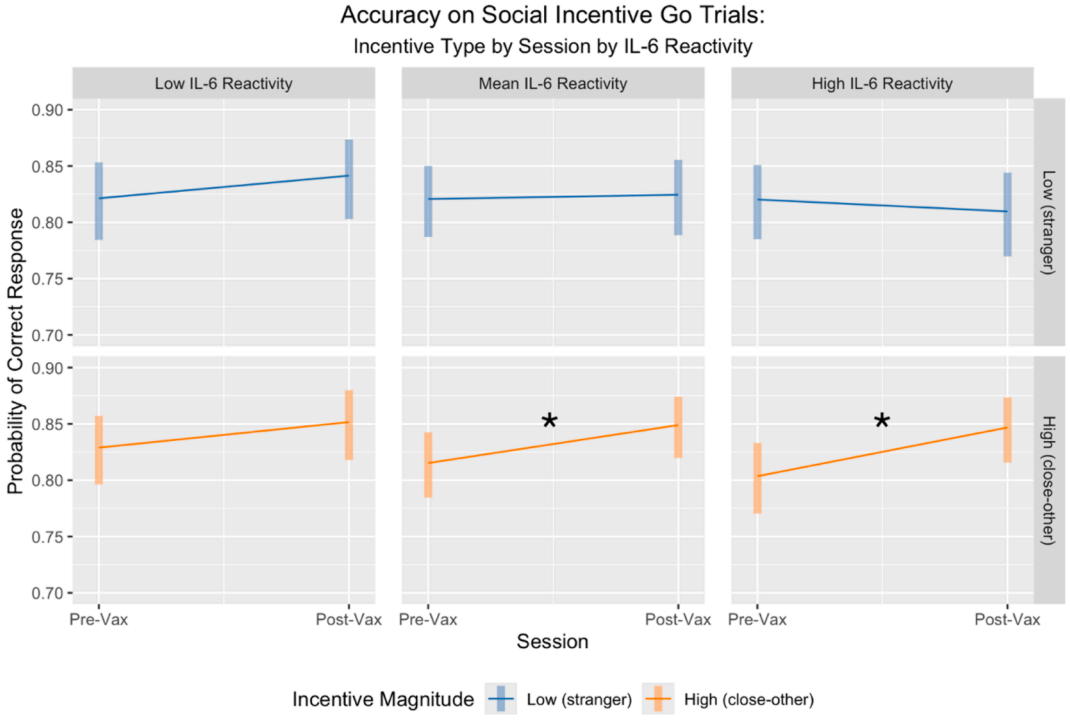

Fig. 4. Accuracy on Social Incentive Go Trials. The graph displays the probability of correct responses in go trials featuring social incentives. Low magnitude incentives (time viewing a picture of a stranger) is denoted in blue, while the high magnitude incentive (time viewing a picture of a close-others) is in orange. Results are also stratified by IL-6 reactivity (lower than average, average, higher than average). Data are shown for both pre-vaccination and post-vaccination sessions. The asterisks represent a significant change in accuracy from the pre-vaccine session to the post-vaccine session.

2008; Grigoleit et al., 2010; van den Boogaard et al., 2010; Handke et al., 2020; Madison et al., 2023). This discrepancy suggests that the association between inflammation and cognitive function may differ by domain, perhaps with decision-making processes being more susceptible than those involved in response inhibition. Alternatively, the null association observed here and in other studies might be due to methodological choices. Indeed, a recent paper notes that there are several aspects of task design that may influence participants' performance on inhibitory control paradigms (Chiew, 2021). Specifically, task difficulty and task length are both found to moderate the association between incentives and response inhibition. A follow-up study should explore whether lengthening the task, decreasing the ratio between go- and no-go trials, enhancing the incentives, and varying the reaction time window are associated with a different pattern of associations with acute inflammation.

Our next set of findings demonstrated that acute increases in IL-6 were associated with fewer errors of omission (i.e., failures to respond on go trials) during particular incentive conditions. Specifically, we found that higher IL-6 after the vaccine was associated with fewer errors of omission on trials where a high-magnitude social incentive could be earned (i.e., viewing an image of a smiling close other), as compared to both low-magnitude social (i.e., viewing an image of a smiling stranger) and high-magnitude monetary incentives. These findings align with prior work suggesting that inflammation may differentially alter sensitivity to social versus non-social incentives (Inagaki et al., 2015; Muscatell et al., 2016a). Furthermore, prior work has found that increases in inflammation may be associated with faster automatic approach behavior toward a close other; these approach effects were not found towards strangers (Jolink et al., 2022). Such findings are supported by non-human animal research indicating that inflammatory challenges that increase IL-6 levels (e.g., LPS) encourage approach behavior towards familiar social partners (Willette et al., 2007; Yee & Prendergast, 2010).

Omission errors are particularly informative as they could indicate lapses in sustained attention in contexts where people must manage the dual demands of rapid and precise responses to a primary task coupled with intermittent response inhibition requirements (Wright et al., 2014). They have further been linked to difficulties in managing attention (Bezdijan et al., 2009). These findings suggest that participants with greater IL-6 reactivity experienced enhanced sustained attention to high social incentives, resulting in fewer omission errors. This implies that inflammation may increase the saliency of close others, thereby facilitating better performance on trials when such stimuli are used as an incentive. Relatedly, prior work suggests that inflammation might be associated with subtle shifts in motivational and attentional dynamics, as evidenced by significant decreases in subjective effort (Handke et al., 2020) and increases in reaction time and reaction time variability (Handke et al., 2020; Brydon et al., 2008) as a function of an inflammatory challenge. Our study explored how IL-6 fluctuations vary with incentivized inhibitory control, thereby broadening our understanding of how inflammation could influence the dynamics between motivation and cognitive function.

Building on the interplay between inflammation-induced attentional shifts and inhibitory control, there is substantial neurobiological evidence suggesting that pro-inflammatory cytokines such as IL-6 contribute significantly to cognitive dynamics. Pro-inflammatory cytokines, such as IL-6, can alter molecular and cellular aspects of cognition (McAfoose & Baune, 2009) via neuron-to-glia communication (Jurgens & Johnson, 2012; Wohleb et al., 2013), neurogenesis (Hueston et al., 2017; McKim et al., 2016), neuroplasticity (Delpech et al., 2015; Calabrese et al., 2014), long-term potentiation (Wohleb & Delpech, 2017), and neurotransmitter systems (Dantzer et al., 2008; Zhu et al., 2006). Mounting evidence suggests that alterations in the dopaminergic neurotransmitter system due to pro-inflammatory cytokines might be one critical pathway linking motivated behavior and cognitive processes (see Haroon et al., 2012 & Dooley et al., 2018 for reviews). Interestingly, dopaminergic signaling has also been implicated in inhibitory control through well-established effects in the striatum and cortex (Westbrook et al., 2021). The basal ganglia, a region within the striatum supplied with high levels of dopamine, has been found to regulate the cognitive proactive and reactive processes needed during the go/no-go task (Beste et al., 2010; Criaud et al., 2021). While the bulk of the evidence, including that from acute models of inflammation using agents like LPS, supports immediate and transient changes in dopamine levels, chronic inflammation is also known to cause sustained alterations in dopaminergic signaling that can affect these same neural circuits over time (Hunter et al., 2007). Together, these findings suggest a possible neurobiological pathway for the observed link between inflammation, incentives, and inhibitory control. Although the dopaminergic system was not explicitly measured in the present study, future neuroimaging research should examine these possibilities to establish the neurobiological pathways through which peripheral fluctuations in inflammation may influence attention and inhibitory control as a function of incentive in humans.

These results expand upon the cross-sectional studies linking inflammation to cognitive impairment among those with Alzheimer's and dementia-related diseases. While the majority of research exploring links between inflammation and cognition has been limited to aging and dementia-related cognitive changes (Schmidt et al., 2002; Kuo et al., 2005; Koyama et al., 2013), the present results suggest that even mild, acute increases in inflammation may be relevant to subtle changes in cognitive functioning during daily life in young adulthood. One strength of the current study is that we used the well-validated go/no-go task to measure inhibitory control. Unlike previous research that has examined broad cognitive impairments using neuropsychological batteries (Fard et al., 2022), utilizing the go/no-go task allowed us to uncover associations between subtle changes in IL-6 reactivity and both inhibition and attentional aspects of inhibitory control. Furthermore, our study used an innovative approach in adapting the go/no-go paradigm to explore how the sociality and magnitude of incentives modulated the association between inflammation and inhibitory control. This unique approach allowed us to assess the impact of different types of incentives on cognitive functioning, providing a more detailed understanding of this interaction.

The present findings should be interpreted in light of the study's limitations. First, the lack of a placebo/sham vaccine control group limits the ability to make any causal inferences from our data. While we leveraged multilevel logistic regression at a trial-by-trial level to assess within person changes before and after the vaccine, future experimental work including a placebo control and/or a non-incentivized task condition is needed to delineate the causal relationships between IL-6 reactivity and incentivized inhibitory control. Second, it is possible that the sample size was too small to be generalizable, particularly considering that our sample was comprised of undergraduate students under the age of 25. It is also likely that this sample size limited our power to detect smaller effects of interest, including the three-way interactions of interest (i.e., incentive condition x session x IL-6) on the no-go trials. Despite each participant contributing up to 576 observations, only 96 of these were no-go trials. Additionally, the overall sample size of 55 may not have provided sufficient power to detect all relevant between-person interactive effects. Although model diagnostics were conducted to ensure that the effects were not driven by the undue influence of outliers, there is the potential for high values to disproportionately influence results in smaller samples and caution is warranted when interpreting the current effects. Third, it is also important to consider that this study only focused on changes in one inflammatory cytokine, IL-6, and there may be other cytokines that relate to inhibitory control. As such, the results of this study should be interpreted only for the inflammatory cytokine interleukin-6. Fourth, while the go/no-go task primarily assesses inhibitory control, the influence of cytokines on memory processes (Bollen et al., 2017), which may indirectly affect task performance (Criaud & Boulinguez, 2013), was not directly assessed.

Future studies ought to disentangle the specific effects of cytokines on various cognitive functions to clarify their effects on cognition more broadly. In conclusion, while these limitations suggest a need for careful interpretation of our findings, they also highlight opportunities for future studies to explore these complexities more thoroughly.

Although large-scale epidemiological studies suggest an association between inflammation and cognitive impairments, there was a need for laboratory-based studies to clarify the link between acute increases in inflammation and cognitive functioning. The current study specifically examined whether inhibitory control changes following a mild inflammatory challenge and whether that change varied as a function of different incentive conditions. We did not observe any significant effects between IL-6 reactivity and change in errors of commission (i.e., failure to omit response on no-go trials), but we did find differential effects related to social versus monetary incentives on errors of omission (i.e., failures to respond on go trials). Notably, IL-6 reactivity was positively associated with fewer errors of omission when a large social incentive was on the line (i.e., time viewing a smiling close other). These results suggest that IL-6 reactivity may enhance cognitive performance under specific conditions, indicating that even mild fluctuations in inflammation may selectively influence the valuation of incentives, depending on their social significance and magnitude. To our knowledge, this study is the first to demonstrate a link between IL-6 reactivity and performance on an incentivized inhibitory control task, calling for further research to elucidate the mechanistic pathways linking inflammation-related changes in motivation and cognition across the lifespan.

4. Statement on generative AI

During the preparation of this work the authors used OpenAI's ChatGPT to support the editing process and improve the clarity of the manuscript. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

CRediT authorship contribution statement

Gabriella M. Alvarez: Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Tatum A. Jolink:** Writing – review & editing, Validation, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Taylor N. West:** Writing – review & editing, Project administration, Methodology, Data curation, Conceptualization. **Megan N. Cardenas:** Writing – review & editing, Project administration, Methodology, Data curation, Conceptualization. **Mallory J. Feldman:** Writing – review & editing, Validation, Project administration, Formal analysis, Data curation, Conceptualization. **Jessica R. Cohen:** Writing – review & editing, Validation, Supervision, Conceptualization. **Keely A. Muscatell:** Writing – review & editing, Writing – original draft, Validation, Supervision, Resources, Project administration, Funding acquisition, Data curation, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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Appendix A. Supplementary data

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Reference

American Psychological Association. (2020). Bias-free language: Gender. In Publication Manual of the American Psychological Association (7th ed.). Retrieved from https://apastyle.apa.org/style-grammar-guidelines/bias-free-language/gender.

Asci, O., Braem, S., Park, H.R.P., Boehler, C.N., Krebs, R.M., 2019. Neural correlates of reward-related response tendencies in an equiprobable Go/NoGo task. Cogn. Affect. Behav. Neurosci. 19 (3), 555–567. https://doi.org/10.3758/s13415-019-00692-5.

Bates, D., Mächler, M., Bolker, B., Walker, S., 2015. Fitting linear mixed-effects models using lme4. J. Stat. Softw. 67 (1), 1–48. https://doi.org/10.18637/jss.v067.i01.

Bauer, D.J., Sterba, S.K., 2011. Fitting multilevel models with ordinal outcomes: performance of alternative specifications and methods of estimation. Psychol. Methods 16 (4), 373–390. https://doi.org/10.1037/a0025813.

Belleville, S., Fouquet, C., Hudon, C., Zomahoun, H. T. V., Croteau, J., & Consortium for the Early Identification of Alzheimer's disease-Quebec. (2017). Neuropsychological Measures that Predict Progression from Mild Cognitive Impairment to Alzheimer's type dementia in Older Adults: a Systematic Review and Meta-Analysis. Neuropsychology Review, 27(4), 328–353. https://doi.org/10.1007/s11065-017-9361-5.

Benikos, N., Johnstone, S.J., Roodenrys, S.J., 2013. Varying task difficulty in the Go/Nogo task: the effects of inhibitory control, arousal, and perceived effort on ERP components. Int. J. Psychophysiol. 87 (3), 262–272. https://doi.org/10.1016/j.ijpsycho.2012.08.005.

Beste, C., Willemssen, R., Saft, C., Falkenstein, M., 2010. Response inhibition subprocesses and dopaminergic pathways: basal ganglia disease effects. Neuropsychologia 48 (2), 366–373. https://doi.org/10.1016/j.neuropsychologia.2009.09.023.

Bettcher, B.M., Kramer, J.H., 2014. Longitudinal inflammation, cognitive decline, and Alzheimer's disease: a mini-review. Clin. Pharmacol. Ther. 96 (4), 464–469. https://doi.org/10.1038/clpt.2014.147.

Beydoun, M.A., Weiss, J., Obhi, H.K., Beydoun, H.A., Dore, G.A., Liang, H., Evans, M.K., Zonderman, A.B., 2019. Cytokines are associated with longitudinal changes in cognitive performance among urban adults. Brain Behav. Immun. 80, 474–487. https://doi.org/10.1016/j.bbi.2019.04.027.

Biesanz, J.C., Deeb-Sossa, N., Papadakis, A.A., Bollen, K.A., Curran, P.J., 2004. The role of coding time in estimating and interpreting growth curve models. Psychol. Methods 9, 1–30.

Bollen, J., Trick, L., Llewellyn, D., Dickens, C., 2017. The effects of acute inflammation on cognitive functioning and emotional processing in humans: A systematic review of experimental studies. J. Psychosom. Res. 94, 47–55. https://doi.org/10.1016/j.jpsychores.2017.01.002.

Botvinick, M., Braver, T., 2015. Motivation and cognitive control: from behavior to neural mechanism. Annu. Rev. Psychol. 66, 83–113. https://doi.org/10.1146/annurev-psych-010814-015044.

Boyle, C.C., Kuhlman, K.R., Dooley, L.N., Haydon, M.D., Robles, T.F., Ang, Y.-S., Pizzagalli, D.A., Bower, J.E., 2019. Inflammation and dimensions of reward processing following exposure to the influenza vaccine. Psychoneuroendocrinology 102, 16–23. https://doi.org/10.1016/j.psyneuen.2018.11.024.

Boyle, C.C., Bower, J.E., Eisenberger, N.I., Irwin, M.R., 2023. Stress to inflammation and anhedonia: Mechanistic insights from preclinical and clinical models. Neurosci. Biobehav. Rev. 152, 105307. https://doi.org/10.1016/j.neubiorev.2023.105307.

Brydon, L., Harrison, N.A., Walker, C., Steptoe, A., Critchley, H.D., 2008. Peripheral inflammation is associated with altered substantia nigra activity and psychomotor slowing in humans. Biol. Psychiatry 63 (11), 1022–1029. https://doi.org/10.1016/j.biopsych.2007.12.007.

Burrows, K., Stewart, J. L., Kuplicki, R., Figueroa-Hall, L., Spechler, P. A., Zheng, H., ... & Tulsa 1000 Investigators. (2021). Elevated peripheral inflammation is associated

with attenuated striatal reward anticipation in major depressive disorder. Brain, behavior, and immunity, 93, 214-225.
Burton, S., Knibb, G., Jones, A., 2021. A meta-analytic investigation of the role of reward on inhibitory control. Q. J. Exp. Psychol. 74 (10), 1818–1828. https://doi.org/10.1177/17470218211008895.
Calabrese, F., Rossetti, A.C., Racagni, G., Gass, P., Riva, M.A., Molteni, R., 2014. Brain-derived neurotrophic factor: a bridge between inflammation and neuroplasticity. Front. Cell. Neurosci. 8, 430. https://doi.org/10.3389/fncel.2014.00430.
Capuron, L., Ravaud, A., Dantzer, R., 2001. Timing and specificity of the cognitive changes induced by interleukin-2 and interferon-alpha treatments in cancer patients. Psychosom. Med. 63 (3), 376–386. https://doi.org/10.1097/00006842-200105000-00007.
Carty, C.L., Heagerty, P., Nakayama, K., McClung, E.C., Lewis, J., Lum, D., Boespflug, E., McCloud-Gehring, C., Soleimani, B.R., Ranchalis, J., Bacus, T.J., Furlong, C.E., Jarvik, G.P., 2006. Inflammatory response after influenza vaccination in men with and without carotid artery disease. Arterioscler. Thromb. Vasc. Biol. 26 (12), 2738–2744. https://doi.org/10.1161/01.ATV.0000248534.30057.b5.
Chaytor, N., Schmitter-Edgecombe, M., 2003. The ecological validity of neuropsychological tests: a review of the literature on everyday cognitive skills. Neuropsychol. Rev. 13 (4), 181–197. https://doi.org/10.1023/b:nerv.0000009483.91468.fb.
Chiew, K.S., 2021. Revisiting positive affect and reward influences on cognitive control. Curr. Opin. Behav. Sci. 39, 27–33. https://doi.org/10.1016/j.cobeha.2020.11.010.
Chiew, K.S., Braver, T.S., 2011. Positive affect versus reward: emotional and motivational influences on cognitive control. Front. Psychol. 2, 279. https://doi.org/10.3389/fpsyg.2011.00279.
Chiew, K.S., Stanek, J.K., Adcock, R.A., 2016. Reward Anticipation Dynamics during Cognitive Control and Episodic Encoding: Implications for Dopamine. Front. Hum. Neurosci. 10, 555. https://doi.org/10.3389/fnhum.2016.00555.
Christian, L.M., Porter, K., Karlsson, E., Schultz-Cherry, S., Iams, J.D., 2013. Serum proinflammatory cytokine responses to influenza virus vaccine among women during pregnancy versus non-pregnancy. Am. J. Reprod. Immunol. 70 (1), 45–53. https://doi.org/10.1111/aji.12117.
Cohen, O., Dankner, R., Chetrit, A., Luxenburg, O., Langenauer, C., Shinfeld, A., Smolinsky, A.K., 2003. Multidisciplinary intervention for control of diabetes in patients undergoing coronary artery bypass graft (CABG). Cardiovascular Surgery (london, England) 11 (3), 195–200. https://doi.org/10.1016/s0967-2109(03)00019-x.
Criaud, M., Boulinguez, P., 2013. Have we been asking the right questions when assessing response inhibition in go/no-go tasks with fMRI? A meta-analysis and critical review. Neurosci. Biobehav. Rev. 37 (1), 11–23. https://doi.org/10.1016/j.neubiorev.2012.11.003.
Criaud, M., Anton, J.-L., Nazarian, B., Longcamp, M., Metereau, E., Boulinguez, P., & Ballanger, B. (2021). The Human Basal Ganglia Mediate the Interplay between Reactive and Proactive Control of Response through Both Motor Inhibition and Sensory Modulation. Brain Sciences, 11(5). https://doi.org/10.3390/brainsci11050560.
Curran, P.J., Bauer, D.J., 2011. The disaggregation of within-person and between-person effects in longitudinal models of change. Annu. Rev. Psychol. 62, 583–619. https://doi.org/10.1146/annurev.psych.093008.100356.
Daly, M., McMinn, D., Allan, J.L., 2014. A bidirectional relationship between physical activity and executive function in older adults. Front. Hum. Neurosci. 8, 1044. https://doi.org/10.3389/fnhum.2014.01044.
Dantzer, R., O'Connor, J.C., Freund, G.G., Johnson, R.W., Kelley, K.W., 2008. From inflammation to sickness and depression: when the immune system subjugates the brain. Nat. Rev. Neurosci. 9 (1), 46–56. https://doi.org/10.1038/nrn2297.
de Vet, H.C.W., Mokkink, L.B., Mosmuller, D.G., Terwee, C.B., 2017. Spearman-Brown prophecy formula and Cronbach's alpha: different faces of reliability and opportunities for new applications. J. Clin. Epidemiol. 85, 45–49. https://doi.org/10.1016/j.jclinepi.2017.01.013.
DellaGioia, N., Hannestad, J., 2010. A critical review of human endotoxin administration as an experimental paradigm of depression. Neurosci. Biobehav. Rev. 34 (1), 130–143. https://doi.org/10.1016/j.neubiorev.2009.07.014.
Delpech, J.-C., Madore, C., Nadjar, A., Joffre, C., Wohleb, E.S., Layé, S., 2015. Microglia in neuronal plasticity: Influence of stress. Neuropharmacology 96 (Pt A), 19–28. https://doi.org/10.1016/j.neuropharm.2014.12.034.
Diamond, A., 2013. Executive functions. Annu. Rev. Psychol. 64, 135–168. https://doi.org/10.1146/annurev-psych-113011-143750.
Dooley, L.N., Kuhlman, K.R., Robles, T.F., Eisenberger, N.I., Craske, M.G., Bower, J.E., 2018. The role of inflammation in core features of depression: Insights from paradigms using exogenously-induced inflammation. Neurosci. Biobehav. Rev. 94, 219–237. https://doi.org/10.1016/j.neubiorev.2018.09.006.
Draper, A., Koch, R.M., van der Meer, J.W., Aj Apps, M., Pickkers, P., Husain, M., van der Schaaf, M.E., 2018. Effort but not reward sensitivity is altered by acute sickness induced by experimental endotoxemia in humans. Neuropsychopharmacology 43 (5), 1107–1118. https://doi.org/10.1038/npp.2017.231.
Edwards, K.M., Burns, V.E., Reynolds, T., Carroll, D., Drayson, M., Ring, C., 2006. Acute stress exposure prior to influenza vaccination enhances antibody response in women. Brain Behav. Immun. 20 (2), 159–168. https://doi.org/10.1016/j.bbi.2005.07.001.
Eisenberger, N.I., Inagaki, T.K., Mashal, N.M., Irwin, M.R., 2010. Inflammation and social experience: an inflammatory challenge induces feelings of social disconnection in addition to depressed mood. Brain Behav. Immun. 24 (4), 558–563. https://doi.org/10.1016/j.bbi.2009.12.009.
Eisenberger, N.I., Moieni, M., Inagaki, T.K., Muscatell, K.A., Irwin, M.R., 2017. In Sickness and in Health: The Co-Regulation of Inflammation and Social Behavior. Neuropsychopharmacology 42 (1), 242–253. https://doi.org/10.1038/npp.2016.141.
Fard, M.T., Savage, K.M., Stough, C.K., 2022. Peripheral inflammation marker relationships to cognition in healthy older adults - A systematic review. Psychoneuroendocrinology 144, 105870. https://doi.org/10.1016/j.psyneuen.2022.105870.
Faul, F., Erdfelder, E., Lang, A.-G., Buchner, A., 2007. G*Power 3: A flexible statistical power analysis for the social, behavioral, and biomedical sciences. Behav. Res. Methods 39, 175–191.
Feldman, M., Jolink, T.A., Alvarez, G.M., Fendinger, N., Gaudier-Diaz, M., Lindquist, K.A., Muscatell, K., 2022. August 22). Vaccination Model of Inflammation. Retrieved from osf.io/da346.
Feldman, M.J., Jolink, T.A., Alvarez, G.M., Fendinger, N.J., Gaudier-Diaz, M.M., Lindquist, K.A., Muscatell, K.A., 2023. The roles of inflammation, affect, and interoception in predicting social perception. Brain Behav. Immun. 112, 246–253. https://doi.org/10.1016/j.bbi.2023.05.011.
Felger, J.C., Treadway, M.T., 2017. Inflammation effects on motivation and motor activity: role of dopamine. Neuropsychopharmacology 42 (1), 216–241. https://doi.org/10.1038/npp.2016.143.
Freeman, S.M., Aron, A.R., 2016. Withholding a Reward-driven Action: Studies of the Rise and Fall of Motor Activation and the Effect of Cognitive Depletion. J. Cogn. Neurosci. 28 (2), 237–251. https://doi.org/10.1162/jocn_a_00893.
Frenois, F., Moreau, M., O'Connor, J., Lawson, M., Micon, C., Lestage, J., Kelley, K.W., Dantzer, R., Castanon, N., 2007. Lipopolysaccharide induces delayed FosB/DeltaFosB immunostaining within the mouse extended amygdala, hippocampus and hypothalamus, that parallel the expression of depressive-like behavior. Psychoneuroendocrinology 32 (5), 516–531. https://doi.org/10.1016/j.psyneuen.2007.03.005.
Gassen, J., Makhanova, A., Maner, J.K., Plant, E.A., Eckel, L.A., Nikonova, L., Prokosch, M.L., Boehm, G.W., Hill, S.E., 2019a. Experimentally-Induced Inflammation Predicts Present Focus. Adapt. Hum. Behav. Physiol. 5 (2), 148–163. https://doi.org/10.1007/s40750-019-00110-7.
Gassen, J., Prokosch, M.L., Eimerbrink, M.J., Proffitt Leyva, R.P., White, J.D., Peterman, J.L., Burgess, A., Cheek, D.J., Kreutzer, A., Nicolas, S.C., Boehm, G.W., Hill, S.E., 2019b. Inflammation Predicts Decision-Making Characterized by Impulsivity, Present Focus, and an Inability to Delay Gratification. Sci. Rep. 9 (1), 4928. https://doi.org/10.1038/s41598-019-41437-1.
Geier, C.F., Luna, B., 2012. Developmental effects of incentives on response inhibition. Child Dev. 83 (4), 1262–1274. https://doi.org/10.1111/j.1467-8624.2012.01771.x.
Gilbert, S.J., Burgess, P.W., 2008. Executive function. Curr. Biol. 18 (3), R110–R114. https://doi.org/10.1016/j.cub.2007.12.014.
Gorelick, P.B., 2010. Role of inflammation in cognitive impairment: results of observational epidemiological studies and clinical trials. Ann. N. Y. Acad. Sci. 1207, 155–162. https://doi.org/10.1111/j.1749-6632.2010.05726.x.
Grigoleit, J.-S., Oberbeck, J.R., Lichte, P., Kobbe, P., Wolf, O.T., Montag, T., del Rey, A., Gizewski, E.R., Engler, H., Schedlowski, M., 2010. Lipopolysaccharide-induced experimental immune activation does not impair memory functions in humans. Neurobiol. Learn. Mem. 94 (4), 561–567. https://doi.org/10.1016/j.nlm.2010.09.011.
Grigoleit, J.-S., Kullmann, J.S., Wolf, O.T., Hammes, F., Wegner, A., Jablonowski, S., Engler, H., Gizewski, E., Oberbeck, R., Schedlowski, M., 2011. Dose-dependent effects of endotoxin on neurobehavioral functions in humans. PLoS One 6 (12), e28330.
Hall, P.A., Fong, G.T., Epp, L.J., Elias, L.J., 2008. Executive function moderates the intention-behavior link for physical activity and dietary behavior. Psychol. Health 23 (3), 309–326. https://doi.org/10.1080/14768320701212099.
Handke, A., Axelsson, J., Benson, S., Boy, K., Weskamp, V., Hasenberg, T., Remy, M., Hebebrand, J., Föcker, M., Brinkhoff, A., Unteroberdörster, M., Engler, H., Schedlowski, M., Lasselin, J., 2020. Acute inflammation and psychomotor slowing: Experimental assessment using lipopolysaccharide administration in healthy humans. Brain, Behavior, & Immunity Health 8, 100130. https://doi.org/10.1016/j.bbih.2020.100130.
Haroon, E., Raison, C.L., Miller, A.H., 2012. Psychoneuroimmunology meets neuropsychopharmacology: translational implications of the impact of inflammation on behavior. Neuropsychopharmacology 37 (1), 137–162. https://doi.org/10.1038/npp.2011.205.
Hennessy, M.B., Deak, T., Schiml, P.A., 2014. Sociality and sickness: have cytokines evolved to serve social functions beyond times of pathogen exposure? Brain Behav. Immun. 37, 15–20. https://doi.org/10.1016/j.bbi.2013.10.021.
Heymsfield, S.B., Peterson, C.M., Thomas, D.M., Heo, M., Schuna, J.M., 2016. Why are there race/ethnic differences in adult body mass index-adiposity relationships? A Quantitative Critical Review. Obesity Reviews 17 (3), 262–275. https://doi.org/10.1111/obr.12358.
Hueston, C.M., Cryan, J.F., Nolan, Y.M., 2017. Adolescent social isolation stress unmasks the combined effects of adolescent exercise and adult inflammation on hippocampal neurogenesis and behavior. Neuroscience 365, 226–236. https://doi.org/10.1016/j.neuroscience.2017.09.020.
Hunter, R.L., Dragicevic, N., Seifert, K., Choi, D.Y., Liu, M., Kim, H.-C., Cass, W.A., Sullivan, P.G., Bing, G., 2007. Inflammation induces mitochondrial dysfunction and dopaminergic neurodegeneration in the nigrostriatal system. J. Neurochem. 100 (5), 1375–1386. https://doi.org/10.1111/j.1471-4159.2006.04327.x.
Iaboni, F., Douglas, V.I., Baker, A.G., 1995. Effects of reward and response costs on inhibition in ADHD children. J. Abnorm. Psychol. 104 (1), 232–240. https://doi.org/10.1037/0021-843X.104.1.232.
Inagaki, T.K., Muscatell, K.A., Irwin, M.R., Cole, S.W., Eisenberger, N.I., 2012. Inflammation selectively enhances amygdala activity to socially threatening images.

Neuroimage 59 (4), 3222–3226. https://doi.org/10.1016/j.neuroimage.2011.10.090.
Inagaki, T.K., Muscatell, K.A., Irwin, M.R., Moieni, M., Dutcher, J.M., Jevtic, I., Breen, E. C., Eisenberger, N.I., 2015. The role of the ventral striatum in inflammatory-induced approach toward support figures. Brain Behav. Immun. 44, 247–252. https://doi.org/10.1016/j.bbi.2014.10.006.
Janelsins, M.C., Kesler, S.R., Ahles, T.A., Morrow, G.R., 2014. Prevalence, mechanisms, and management of cancer-related cognitive impairment. Int. Rev. Psychiatry 26 (1), 102–113.
Jolink, T.A., Fendinger, N.J., Alvarez, G.M., Feldman, M.J., Gaudier-Diaz, M.M., Muscatell, K.A., 2022. Inflammatory reactivity to the influenza vaccine is associated with changes in automatic social behavior. Brain Behav. Immun. 99, 339–349. https://doi.org/10.1016/j.bbi.2021.10.019.
Jolink, T.A., West, T.N., Alvarez, G.M., Cardenas, M.N., Feldman, M.J., Algoe, S.B., Muscatell, K.A., 2024. Higher interleukin-6 is associated with greater momentary social connection in close relationships in daily life. Psychoneuroendocrinology 164, 107020. https://doi.org/10.1016/j.psyneuen.2024.107020.
Jurgens, H.A., Amancherla, K., Johnson, R.W., 2012. Influenza infection induces neuroinflammation, alters hippocampal neuron morphology, and impairs cognition in adult mice. J. Neurosci. 32 (12), 3958–3968. https://doi.org/10.1523/JNEUROSCI.6389-11.2012.
Jurgens, H.A., Johnson, R.W., 2012. Dysregulated neuronal-microglial cross-talk during aging, stress and inflammation. Exp. Neurol. 233 (1), 40–48. https://doi.org/10.1016/j.expneurol.2010.11.014.
Kaiser, D., Weise, G., Möller, K., Scheibe, J., Pösel, C., Baasch, S., Gawlitza, M., Lobsien, D., Diederich, K., Minnerup, J., Kranz, A., Boltze, J., Wagner, D.-C., 2014. Spontaneous white matter damage, cognitive decline and neuroinflammation in middle-aged hypertensive rats: an animal model of early-stage cerebral small vessel disease. Acta Neuropathol. Commun. 2, 169. https://doi.org/10.1186/s40478-014-0169-8.
Kouneiher, F., Charron, S., Koechlin, E., 2009. Motivation and cognitive control in the human prefrontal cortex. Nat. Neurosci. 12 (7), 939–945. https://doi.org/10.1038/nn.2321.
Koyama, A., O'Brien, J., Weuve, J., Blacker, D., Metti, A.L., Yaffe, K., 2013. The role of peripheral inflammatory markers in dementia and Alzheimer's disease: a meta-analysis. J. Gerontol. A Biol. Sci. Med. Sci. 68 (4), 433–440. https://doi.org/10.1093/gerona/gls187.
Kuhlman, K.R., Robles, T.F., Dooley, L.N., Boyle, C.C., Haydon, M.D., Bower, J.E., 2018. Within-subject associations between inflammation and features of depression: Using the flu vaccine as a mild inflammatory stimulus. Brain Behav. Immun. 69, 540–547.
Kuhlman, K.R., Robles, T.F., Haydon, M.D., Dooley, L., Boyle, C.C., Bower, J.E., 2020. Early life stress sensitizes individuals to the psychological correlates of mild fluctuations in inflammation. Dev. Psychobiol. 62 (3), 400–408. https://doi.org/10.1002/dev.21908.
Kumle, L., Võ, M.-L.-H., Draschkow, D., 2021. Estimating power in (generalized) linear mixed models: An open introduction and tutorial in R. Behav. Res. Methods 53 (6), 2528–2543. https://doi.org/10.3758/s13428-021-01546-0.
Kuo, H.-K., Yen, C.-J., Chang, C.-H., Kuo, C.-K., Chen, J.-H., Sorond, F., 2005. Relation of C-reactive protein to stroke, cognitive disorders, and depression in the general population: systematic review and meta-analysis. Lancet Neurol. 4 (6), 371–380. https://doi.org/10.1016/S1474-4422(05)70099-5.
Kuznetsova A, Brockhoff PB, Christensen RHB (2017). "lmerTest Package: Tests in Linear Mixed Effects Models."_Journal of Statistical Software_, *82*(13), 1-26. doi: 10.18637/jss.v082.i13 <https://doi.org/10.18637/jss.v082.i13>.
Lacourt, T.E., Houtveen, J.H., Veldhuijzen van Zanten, J.J.C.S., Bosch, J.A., Drayson, M. T., Van Doornen, L.J.P., 2015. Negative affectivity predicts decreased pain tolerance during low-grade inflammation in healthy women. Brain Behav. Immun. 44, 32–36. https://doi.org/10.1016/j.bbi.2014.10.003.
Lai, K.S.P., Liu, C.S., Rau, A., Lanctôt, K.L., Köhler, C.A., Pakosh, M., Carvalho, A.F., Herrmann, N., 2017. Peripheral inflammatory markers in Alzheimer's disease: a systematic review and meta-analysis of 175 studies. J. Neurol. Neurosurg. Psychiatry 88 (10), 876–882. https://doi.org/10.1136/jnnp-2017-316201.
Lasselin, J., Treadway, M.T., Lacourt, T.E., Soop, A., Olsson, M.J., Karshikoff, B., Paues-Göranson, S., Axelsson, J., Dantzer, R., Lekander, M., 2017. Lipopolysaccharide alters motivated behavior in a monetary reward task: a randomized trial. Neuropsychopharmacology 42 (4), 801–810. https://doi.org/10.1038/npp.2016.191.
Laurenson, C., Gorwood, P., Orsat, M., Lhuillier, J.-P., Le Gall, D., Richard-Devantoy, S., 2015. Cognitive control and schizophrenia: The greatest reliability of the Stroop task. Psychiatry Res. 227 (1), 10–16. https://doi.org/10.1016/j.psychres.2015.03.004.
Leonardo, S., Fregni, F., 2023. Association of inflammation and cognition in the elderly: A systematic review and meta-analysis. Front. Aging Neurosci. 15, 1069439. https://doi.org/10.3389/fnagi.2023.1069439.
Long, S., Chen, Y., Meng, Y., Yang, Z., Wei, M., Li, T., Ni, J., Shi, J., Tian, J., 2023. Peripheral high levels of CRP predict progression from normal cognition to dementia: A systematic review and meta-analysis. J. Clin. Neurosci. 107, 54–63. https://doi.org/10.1016/j.jocn.2022.11.016.
Madan, C.R., 2017. Motivated cognition: effects of reward, emotion, and other motivational factors across a variety of cognitive domains. Collabra. Psychology 3 (1), 24. https://doi.org/10.1525/collabra.111.
Madison, A.A., Kiecolt-Glaser, J.K., 2022. Are sick people really more impulsive?: Investigating inflammation-driven impulsivity. Psychoneuroendocrinology 141, 105763. https://doi.org/10.1016/j.psyneuen.2022.105763.
Madison, A.A., Andridge, R., Renna, M.E., Sheridan, J.F., Lustberg, M., Ramaswamy, B., Wesolowski, R., Williams, N.O., Sardesai, S.D., Noonan, A.M., Reinbolt, R.E., Cherian, M.A., Malarkey, W.B., Kiecolt-Glaser, J.K., 2023. Inflamed but not impulsive: Acute inflammatory cytokine response does not impact prepotent response inhibition. J. Affect. Disord. 342, 1–9. https://doi.org/10.1016/j.jad.2023.09.008.
McAfoose, J., Baune, B.T., 2009. Evidence for a cytokine model of cognitive function. Neurosci. Biobehav. Rev. 33 (3), 355–366. https://doi.org/10.1016/j.neubiorev.2008.10.005.
McDade, T.W., Miller, A., Tran, T.T., Borders, A.E.B., Miller, G., 2021. A highly sensitive multiplex immunoassay for inflammatory cytokines in dried blood spots. Am. J. Hum. Biol. 33 (6), e23558.
McKim, D.B., Niraula, A., Tarr, A.J., Wohleb, E.S., Sheridan, J.F., Godbout, J.P., 2016. Neuroinflammatory Dynamics Underlie Memory Impairments after Repeated Social Defeat. J. Neurosci. 36 (9), 2590–2604. https://doi.org/10.1523/JNEUROSCI.2394-15.2016.
Meyers, C.A., Abbruzzese, J.L., 1992. Cognitive functioning in cancer patients: effect of previous treatment. Neurology 42 (2), 434–436. https://doi.org/10.1212/wnl.42.2.434.
Migliaccio, R., Tanguy, D., Bouzigues, A., Sezer, I., Dubois, B., Le Ber, I., Batrancourt, B., Godefroy, V., Levy, R., 2020. Cognitive and behavioural inhibition deficits in neurodegenerative dementias. Cortex 131, 265–283. https://doi.org/10.1016/j.cortex.2020.08.001.
Miller, E.M., McDade, T.W., 2012. A highly sensitive immunoassay for interleukin-6 in dried blood spots. Am. J. Hum. Biol. 24 (6), 863–865. https://doi.org/10.1002/ajhb.22324.
Miyake, A., Friedman, N.P., Emerson, M.J., Witzki, A.H., Howerter, A., Wager, T.D., 2000. The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: a latent variable analysis. Cogn. Psychol. 41 (1), 49–100. https://doi.org/10.1006/cogp.1999.0734.
Miyake, A., Friedman, N.P., 2012. The Nature and Organization of Individual Differences in Executive Functions: Four General Conclusions. Curr. Dir. Psychol. Sci. 21 (1), 8–14. https://doi.org/10.1177/0963721411429458.
Mostofsky, S.H., Simmonds, D.J., 2008. Response inhibition and response selection: two sides of the same coin. J. Cogn. Neurosci. 20 (5), 751–761. https://doi.org/10.1162/jocn.2008.20500.
Muscatell, K.A., Inagaki, T.K., 2021. Beyond social withdrawal: New perspectives on the effects of inflammation on social behavior. Brain, Behavior, & Immunity Health 16, 100302. https://doi.org/10.1016/j.bbih.2021.100302.
Nederkoorn, C., Houben, K., Hofmann, W., Roefs, A., Jansen, A., 2010. Control yourself or just eat what you like? Weight gain over a year is predicted by an interactive effect of response inhibition and implicit preference for snack foods. Health Psychol. 29 (4), 389–393. https://doi.org/10.1037/a0019921.
Nosek, B.A., Banaji, M.R., 2001. The Go/No-Go Association Task. Soc. Cogn. 19 (6), 625–666. https://doi.org/10.1521/soco.19.6.625.20886.
O'Connor, M.-F., Irwin, M.R., Wellisch, D.K., 2009. When grief heats up: pro-inflammatory cytokines predict regional brain activation. Neuroimage 47 (3), 891–896. https://doi.org/10.1016/j.neuroimage.2009.05.049.
Padmala, S., Pessoa, L., 2010. Interactions between cognition and motivation during response inhibition. Neuropsychologia 48 (2), 558–565. https://doi.org/10.1016/j.neuropsychologia.2009.10.017.
Papies, E.K., Stroebe, W., Aarts, H., 2008. Healthy cognition: processes of self-regulatory success in restrained eating. Pers. Soc. Psychol. Bull. 34 (9), 1290–1300. https://doi.org/10.1177/0146167208320063.
Pronk, T., Molenaar, D., Wiers, R.W., Murre, J., 2022. Methods to split cognitive task data for estimating split-half reliability: A comprehensive review and systematic assessment. Psychon. Bull. Rev. 29 (1), 44–54. https://doi.org/10.3758/s13423-021-01948-3.
Radin, A.S., Kuhlman, K.R., Boyle, C.C., Haydon, M.D., Bower, J.E., 2021. Using the influenza vaccine as a mild, exogenous inflammatory challenge: When does inflammation peak? Brain, Behavior, & Immunity Health 13, 100239. https://doi.org/10.1016/j.bbih.2021.100239.
Reimann, Z., Miller, J.R., Dahle, K.M., Hooper, A.P., Young, A.M., Goates, M.C., Magnusson, B.M., Crandall, A., 2020. Executive functions and health behaviors associated with the leading causes of death in the United States: A systematic review. J. Health Psychol. 25 (2), 186–196. https://doi.org/10.1177/1359105318800829.
Saleem, M., Herrmann, N., Swardfager, W., Eisen, R., Lanctôt, K.L., 2015. Inflammatory Markers in Mild Cognitive Impairment: A Meta-Analysis. Journal of Alzheimer's Disease 47 (3), 669–679. https://doi.org/10.3233/JAD-150042.
Savi, F.F., de Oliveira, A., de Medeiros, G.F., Bozza, F.A., Michels, M., Sharshar, T., Dal-Pizzol, F., Ritter, C., 2021. What animal models can tell us about long-term cognitive dysfunction following sepsis: A systematic review. Neurosci. Biobehav. Rev. 124, 386–404. https://doi.org/10.1016/j.neubiorev.2020.12.005.
Schmidt, R., Schmidt, H., Curb, J.D., Masaki, K., White, L.R., Launer, L.J., 2002. Early inflammation and dementia: a 25-year follow-up of the Honolulu-Asia Aging Study. Ann. Neurol. 52 (2), 168–174. https://doi.org/10.1002/ana.10265.
Segerstrom, S.C., Hardy, J.K., Evans, D.R., Greenberg, R.N., 2012. Vulnerability, distress, and immune response to vaccination in older adults. Brain Behav. Immun. 26 (5), 747–753. https://doi.org/10.1016/j.bbi.2011.10.009.
Shen, Y., Connor, T.J., Nolan, Y., Kelly, J.P., Leonard, B.E., 1999. Differential effect of chronic antidepressant treatments on lipopolysaccharide-induced depressive-like behavioural symptoms in the rat. Life Sci. 65 (17), 1773–1786. https://doi.org/10.1016/s0024-3205(99)00430-0.
Shields, G.S., Moons, W.G., Slavich, G.M., 2017. Inflammation, Self-Regulation, and Health: An Immunologic Model of Self-Regulatory Failure. Perspect. Psychol. Sci. 12 (4), 588–612. https://doi.org/10.1177/1745691616689091.
Skelly, D.T., Griffin, É.W., Murray, C.L., Harney, S., O'Boyle, C., Hennessy, E., Dansereau, M.-A., Nazmi, A., Tortorelli, L., Rawlins, J.N., Bannerman, D.M.,

Cunningham, C., 2019. Acute transient cognitive dysfunction and acute brain injury induced by systemic inflammation occur by dissociable IL-1-dependent mechanisms. Mol. Psychiatry 24 (10), 1533–1548. https://doi.org/10.1038/s41380-018-0075-8.
Stanford, F.C., Lee, M., Hur, C., 2019. Race, ethnicity, sex, and obesity: is it time to personalize the scale? Mayo Clin. Proc. 94 (2), 362–363. https://doi.org/10.1016/j.mayocp.2018.10.014.
Su, C., Zhao, K., Xia, H., Xu, Y., 2019. Peripheral inflammatory biomarkers in Alzheimer's disease and mild cognitive impairment: a systematic review and meta-analysis. Psychogeriatrics : the Official Journal of the Japanese Psychogeriatric Society 19 (4), 300–309. https://doi.org/10.1111/psyg.12403.
Tsai, M.Y., Hanson, N.Q., Straka, R.J., Hoke, T.R., Ordovas, J.M., Peacock, J.M., Arends, V.L., Arnett, D.K., 2005. Effect of influenza vaccine on markers of inflammation and lipid profile. J. Lab. Clin. Med. 145 (6), 323–327. https://doi.org/10.1016/j.lab.2005.03.009.
van den Boogaard, M., Ramakers, B.P., van Alfen, N., van der Werf, S.P., Fick, W.F., Hoedemaekers, C.W., Verbeek, M.M., Schoonhoven, L., van der Hoeven, J.G., Pickkers, P., 2010. Endotoxemia-induced inflammation and the effect on the human brain. Crit. Care 14 (3), R81. https://doi.org/10.1186/cc9001.
Verbruggen, F., McLaren, R., 2018. Effects of reward and punishment on the interaction between going and stopping in a selective stop-change task. Psychol. Res. 82 (2), 353–370. https://doi.org/10.1007/s00426-016-0827-5.
Verbruggen, F., McLaren, R., 2018. Effects of reward and punishment on the interaction between going and stopping in a selective stop-change task. Psychol. Res. 82 (2), 353–370. https://doi.org/10.1007/s00426-016-0827-5.
Vichaya, E.G., Dantzer, R., 2018. Inflammation-induced motivational changes: Perspective gained by evaluating positive and negative valence systems. Curr. Opin. Behav. Sci. 22, 90–95. https://doi.org/10.1016/j.cobeha.2018.01.008.
Vichaya, E.G., Hunt, S.C., Dantzer, R., 2014. Lipopolysaccharide reduces incentive motivation while boosting preference for high reward in mice. Neuropsychopharmacology 39 (12), 2884–2890. https://doi.org/10.1038/npp.2014.141.
Wang, L., Li, S., Zhou, X., Theeuwes, J., 2018. Stimuli that signal the availability of reward break into attentional focus. Vision Res. 144, 20–28. https://doi.org/10.1016/j.visres.2017.10.008.
Westbrook, A., Frank, M.J., Cools, R., 2021. A mosaic of cost-benefit control over cortico-striatal circuitry. Trends Cogn. Sci. 25 (8), 710–721. https://doi.org/10.1016/j.tics.2021.04.007.
Willette, A.A., Lubach, G.R., Coe, C.L., 2007. Environmental context differentially affects behavioral, leukocyte, cortisol, and interleukin-6 responses to low doses of endotoxin in the rhesus monkey. Brain Behav. Immun. 21 (6), 807–815. https://doi.org/10.1016/j.bbi.2007.01.007.
Wohleb, E.S., Delpech, J.-C., 2017. Dynamic cross-talk between microglia and peripheral monocytes underlies stress-induced neuroinflammation and behavioral consequences. Prog. Neuropsychopharmacol. Biol. Psychiatry 79 (Pt A), 40–48. https://doi.org/10.1016/j.pnpbp.2016.04.013.
Wohleb, E.S., Powell, N.D., Godbout, J.P., Sheridan, J.F., 2013. Stress-induced recruitment of bone marrow-derived monocytes to the brain promotes anxiety-like behavior. J. Neurosci. 33 (34), 13820–13833. https://doi.org/10.1523/JNEUROSCI.1671-13.2013.
Wright, L., Lipszyc, J., Dupuis, A., Thayapararajah, S.W., Schachar, R., 2014. Response inhibition and psychopathology: a meta-analysis of go/no-go task performance. J. Abnorm. Psychol. 123 (2), 429–439. https://doi.org/10.1037/a0036295.
Wright, C.E., Strike, P.C., Brydon, L., Steptoe, A., 2005. Acute inflammation and negative mood: mediation by cytokine activation. Brain Behav. Immun. 19 (4), 345–350. https://doi.org/10.1016/j.bbi.2004.10.003.

Further reading

Aron, A.R., Robbins, T.W., Poldrack, R.A., 2004. Inhibition and the right inferior frontal cortex. Trends Cogn. Sci. 8 (4), 170–177. https://doi.org/10.1016/j.tics.2004.02.010.
Bektas, A., Schurman, S.H., Sen, R., Ferrucci, L., 2018. Aging, inflammation and the environment. Exp. Gerontol. 105, 10–18. https://doi.org/10.1016/j.exger.2017.12.015.
Bezdjian, S., Baker, L.A., Lozano, D.I., Raine, A., 2009. Assessing inattention and impulsivity in children during the Go/NoGo task. Br. J. Dev. Psychol. 27 (Pt 2), 365–383. https://doi.org/10.1348/026151008X314919.
Blackwell, A.D., Garcia, A.R., 2022. Ecoimmunology in the field: Measuring multiple dimensions of immune function with minimally invasive, field-adapted techniques. Am. J. Hum. Biol. 34 (11), e23784.
Cunningham, C., Hennessy, E., 2015. Co-morbidity and systemic inflammation as drivers of cognitive decline: new experimental models adopting a broader paradigm in dementia research. Alzheimer's Research & Therapy 7 (1), 33. https://doi.org/10.1186/s13195-015-0117-2.
d'Avila, J.C., Siqueira, L.D., Mazeraud, A., Azevedo, E.P., Foguel, D., Castro-Faria-Neto, H.C., Sharshar, T., Chrétien, F., Bozza, F.A., 2018. Age-related cognitive impairment is associated with long-term neuroinflammation and oxidative stress in a mouse model of episodic systemic inflammation. J. Neuroinflammation 15 (1), 28. https://doi.org/10.1186/s12974-018-1059-y.
Eikelenboom, P., Hoozemans, J.J., Veerhuis, R., van Exel, E., Rozemuller, A.J., van Gool, W.A., 2012. Whether, when and how chronic inflammation increases the risk of developing late-onset Alzheimer's disease. Alzheimer's Research & Therapy 4 (3), 15. https://doi.org/10.1186/alzrt118.
Friedman, N.P., Haberstick, B.C., Willcutt, E.G., Miyake, A., Young, S.E., Corley, R.P., Hewitt, J.K., 2007. Greater attention problems during childhood predict poorer executive functioning in late adolescence. Psychol. Sci. 18 (10), 893–900. https://doi.org/10.1111/j.1467-9280.2007.01997.x.
Friedman, N.P., Miyake, A., 2017. Unity and diversity of executive functions: Individual differences as a window on cognitive structure. Cortex 86, 186–204. https://doi.org/10.1016/j.cortex.2016.04.023.
Frömer, R., Lin, H., Dean Wolf, C.K., Inzlicht, M., Shenhav, A., 2021. Expectations of reward and efficacy guide cognitive control allocation. Nat. Commun. 12 (1), 1030. https://doi.org/10.1038/s41467-021-21315-z.
Goldberg, E.L., Dixit, V.D., 2015. Drivers of age-related inflammation and strategies for healthspan extension. Immunol. Rev. 265 (1), 63–74. https://doi.org/10.1111/imr.12295.
Herrera, P.M., Van Meerbeke, A.V., Speranza, M., Cabra, C.L., Bonilla, M., Canu, M., Bekinschtein, T.A., 2019. Expectation of reward differentially modulates executive inhibition. BMC Psychology 7 (1), 55. https://doi.org/10.1186/s40359-019-0332-x.
Jin, R., Chan, A.K.Y., Wu, J., Lee, T.M.C., 2022. Relationships between Inflammation and Age-Related Neurocognitive Changes. Int. J. Mol. Sci. 23 (20). https://doi.org/10.3390/ijms232012573.
Kohls, G., Schulte-Rüther, M., Nehrkorn, B., Müller, K., Fink, G.R., Kamp-Becker, I., Herpertz-Dahlmann, B., Schultz, R.T., Konrad, K., 2013. Reward system dysfunction in autism spectrum disorders. Soc. Cogn. Affect. Neurosci. 8 (5), 565–572. https://doi.org/10.1093/scan/nss033.
Marsland, A.L., Petersen, K.L., Sathanoori, R., Muldoon, M.F., Neumann, S.A., Ryan, C., Flory, J.D., Manuck, S.B., 2006. Interleukin-6 covaries inversely with cognitive performance among middle-aged community volunteers. Psychosom. Med. 68 (6), 895–903. https://doi.org/10.1097/01.psy.0000238451.22174.92.
McDade, T.W., 2014. Development and validation of assay protocols for use with dried blood spot samples. Am. J. Hum. Biol. 26 (1), 1–9. https://doi.org/10.1002/ajhb.22463.
McDade, T.W., Williams, S., Snodgrass, J.J., 2007. What a drop can do: dried blood spots as a minimally invasive method for integrating biomarkers into population-based research. Demography 44 (4), 899–925. https://doi.org/10.1353/dem.2007.0038.
Metti, A.L., Aizenstein, H., Yaffe, K., Boudreau, R.M., Newman, A., Launer, L., Gianaros, P.J., Lopez, O.L., Saxton, J., Ives, D.G., Kritchevsky, S., Vallejo, A.N., Rosano, C., 2015. Trajectories of peripheral interleukin-6, structure of the hippocampus, and cognitive impairment over 14 years in older adults. Neurobiol. Aging 36 (11), 3038–3044. https://doi.org/10.1016/j.neurobiolaging.2015.07.025.
Metti, A.L., Cauley, J.A., 2012. How predictive of dementia are peripheral inflammatory markers in the elderly? Neurodegenerative Disease Management 2 (6), 609–622. https://doi.org/10.2217/NMT.12.68.
Meule, A., 2017. Reporting and Interpreting Task Performance in Go/No-Go Affective Shifting Tasks. Front. Psychol. 8, 701. https://doi.org/10.3389/fpsyg.2017.00701.
Morrisette-Thomas, V., Cohen, A.A., Fülöp, T., Riesco, É., Legault, V., Li, Q., Milot, E., Dusseault-Bélanger, F., Ferrucci, L., 2014. Inflamm-aging does not simply reflect increases in pro-inflammatory markers. Mech. Ageing Dev. 139, 49–57. https://doi.org/10.1016/j.mad.2014.06.005.
Muscatell, K.A., Moieni, M., Inagaki, T.K., Dutcher, J.M., Jevtic, I., Breen, E.C., Irwin, M. R., Eisenberger, N.I., 2016a. Exposure to an inflammatory challenge enhances neural sensitivity to negative and positive social feedback. Brain Behav. Immun. 57, 21–29. https://doi.org/10.1016/j.bbi.2016.03.022.
Muscatell, K.A., Eisenberger, N.I., Dutcher, J.M., Cole, S.W., Bower, J.E., 2016b. Links between inflammation, amygdala reactivity, and social support in breast cancer survivors. Brain Behav. Immun. 53, 34–38. https://doi.org/10.1016/j.bbi.2015.09.008.
Pawelec, G., Goldeck, D., Derhovanessian, E., 2014. Inflammation, ageing and chronic disease. Curr. Opin. Immunol. 29, 23–28. https://doi.org/10.1016/j.coi.2014.03.007.
Shen, X.-N., Niu, L.-D., Wang, Y.-J., Cao, X.-P., Liu, Q., Tan, L., Zhang, C., Yu, J.-T., 2019. Inflammatory markers in Alzheimer's disease and mild cognitive impairment: a meta-analysis and systematic review of 170 studies. J. Neurol. Neurosurg. Psychiatry 90 (5), 590–598. https://doi.org/10.1136/jnnp-2018-319148.
Shields, G.S., Kuchenbecker, S.Y., Pressman, S.D., Sumida, K.D., Slavich, G.M., 2016. Better cognitive control of emotional information is associated with reduced pro-inflammatory cytokine reactivity to emotional stress. Stress (amsterdam, Netherlands) 19 (1), 63–68. https://doi.org/10.3109/10253890.2015.1121983.
Skogstrand, K., Ekelund, C.K., Thorsen, P., Vogel, I., Jacobsson, B., Nørgaard-Pedersen, B., Hougaard, D.M., 2008. Effects of blood sample handling procedures on measurable inflammatory markers in plasma, serum and dried blood spot samples. J. Immunol. Methods 336 (1), 78–84. https://doi.org/10.1016/j.jim.2008.04.006.
Weaver, J.D., Huang, M.H., Albert, M., Harris, T., Rowe, J.W., Seeman, T.E., 2002. Interleukin-6 and risk of cognitive decline: MacArthur studies of successful aging. Neurology 59 (3), 371–378. https://doi.org/10.1212/wnl.59.3.371.
Yang, S., Luo, W., Zhu, X., Broster, L.S., Chen, T., Li, J., Luo, Y., 2014. Emotional content modulates response inhibition and perceptual processing. Psychophysiology 51 (11), 1139–1146. https://doi.org/10.1111/psyp.12255.
Yee, J.R., Prendergast, B.J., 2010. Sex-specific social regulation of inflammatory responses and sickness behaviors. Brain Behav. Immun. 24 (6), 942–951. https://doi.org/10.1016/j.bbi.2010.03.006.
Zhang, K., Zhu, Y., Zhu, Y., Wu, S., Liu, H., Zhang, W., Xu, C., Zhang, H., Hayashi, T., Tian, M., 2016. Molecular, functional, and structural imaging of major depressive disorder. Neurosci. Bull. 32 (3), 273–285. https://doi.org/10.1007/s12264-016-0030-0.