Charting the brain networks of impulsivity: Meta-analytic synthesis, functional connectivity modelling and neurotransmitter associations

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Abstract

Impulsivity is a multidimensional construct that plays a crucial role in human behaviour and is believed to be a transdiagnostic marker of several psychiatric disorders. However, given its multifaceted nature, investigations of its neural correlates are challenging. In this study, we used a comprehensive multi-modal approach to investigate the functional network organisation of two domains in which impulsivity manifests: decision-making and action control. Within-domain ALE meta-analyses of task-based fMRI studies identified two distinct and non-overlapping functional systems: one located in the default-mode network, associated with value-based judgments and goal-directed decision-making, and the other distributed across higher-order networks associated with cognitive control. These systems were organised into four specialised communities of default-mode, cingulo-insular, frontoparietal, and temporal regions and their integration was associated with serotonin receptor density. Our findings reinforce insights from previous behavioural research and provide substantial evidence for the multidimensional nature of impulsivity on the neural level. This highlights the necessity for a comprehensive dimensional ontology on all levels of investigation to address impulsivity in a transdiagnostic manner.

Introduction

Impulsivity has been defined as the “predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the individual or to others” (Moeller et al., 2001). Impulsive behaviours are a pervasive part of life for many individuals, from reckless driving (Teese & Bradley, 2008) or reactive aggression (Gvion & Apter, 2011) to smoking (Sharma et al., 2014) or thrill-seeking (Whiteside & Lynam, 2001; Cyders et al., 2007). Thus, impulsivity plays a crucial role in the human condition, being strongly intertwined with cognitive control and decision-making (Dalley et al., 2011). Heightened impulsivity is believed to be a hallmark of several psychiatric disorders such as attention-deficit/hyperactivity disorder (ADHD), substance abuse, and bipolar disorder (Moeller et al., 2001), which has informed theories of impulsivity as a transdiagnostic marker (Berlin & Hollander, 2014; Amlung et al., 2019). Therefore, understanding the neural mechanisms behind impulsivity is of high research and societal value.

Behavioural and theoretical investigations of impulsivity indicate it is a multidimensional psychological construct (Bari & Robbins, 2013; Caswell et al., 2015; Dalley et al., 2011; Dalley & Robbins, 2017; Dick et al., 2010; MacKillop et al., 2016; Reynolds et al., 2006, 2008), and some authors even argue there is no single umbrella construct of impulsivity at all (Cyders, 2015; Strickland & Johnson, 2020). Proposed models vary, resulting in a lack of consensus on the number and characteristics of the constituent dimensions. Furthermore, trait-based models of impulsivity such as the UPPS-P (Cyders et al., 2007; Whiteside & Lynam, 2001) often appear to be unrelated to assessments of behavioural performance, yielding largely independent bodies of evidence (Sharma et al., 2014; Strickland & Johnson, 2020). Within most performance-based models, impulsivity is believed to manifest as suboptimal decision-making due to discounting of delayed consequences and the failure to inhibit prepotent response tendencies. The subjective decrease in reward value as a function of the delay in obtaining that reward has been labelled as delay consequence sensitivity (DCS; (Strickland & Johnson, 2020), ‘impulsive choice’ (Hamilton, Mitchell, et al., 2015; Winstanley et al., 2006) or ‘impulsive decision-making’ (Sharma et al., 2014). It is classically investigated using the delay discounting paradigm. Failures of response inhibition, the

capacity to inhibit a prepotent response tendency, have been otherwise referred to as ‘impulsive action’ (Winstanley et al., 2006) or ‘rapid-response impulsivity’ (Hamilton, Littlefield, et al., 2015). In humans, it is typically investigated using go/nogo, stop-signal or 5-choice serial reaction time tasks.

In the last few decades, there has been broad interest in understanding the neural mechanisms of impulsivity, with studies reporting associations with brain activity (Christakou et al., 2011; Sripada et al., 2011; DeVito et al., 2013; Wilbertz et al., 2014; Wang, Shen, et al., 2017; Anandakumar et al., 2018), connectivity (Wang, Zhou, et al., 2017; H. Cai et al., 2020) and neurochemistry (de Boer & Koolhaas, 2005; Winstanley et al., 2005; Koffarnus et al., 2011). However, given the multidimensional nature of impulsivity, comprehensive investigations of its neural correlates are challenging. Moreover, results across studies are difficult to compare, as the primary outcome measures are often correlations with impulsive traits that show little overlap with performance-based assessments of impulsivity. As impulsive individuals display altered behavioural responses on DCS and response inhibition tasks, neural correlates can be investigated already on the level of activity in those behavioural tasks without the need for correlational analyses. Here, many studies have explored task-based brain activations during response inhibition (Aron & Poldrack, 2006; Sebastian et al., 2013) and DCS (McClure et al., 2004) as well as associations with DCS-related processes such as the subjective valuation of rewards (Kable & Glimcher, 2007). Partially echoing behavioural findings, these studies point to two largely distinct functional systems associated with response inhibition and DCS (Bari & Robbins, 2013; Dalley et al., 2011), with potentially overlapping regions across domains within the prefrontal cortex (PFC) (Hamilton, Littlefield, et al., 2015; Hamilton, Mitchell, et al., 2015; Cieslik et al., 2015; Zhang et al., 2017; Schüller et al., 2019; Noda et al., 2020). Specifically, evidence suggests that the multiple-demand network, sometimes referred to as the frontoparietal or cognitive control network, subserves inhibitory control exerted to prevent premature responding (Duncan, 2010; Cieslik et al., 2015; Zhang et al., 2017). Conversely, the default-mode network (DMN) or the valuation system together with the dorsolateral prefrontal cortex is believed to underlie DCS (Owens et al., 2017; Schüller et al., 2019; Noda et al., 2020). However, direct comparisons of brain activation during response inhibition and DCS are lacking (see Wang et al. (2016) for a comparison with respect to resting-state connectivity and grey matter volume). Thus, the architecture of the functional brain networks

linked to the different impulsivity dimensions and their interactions remains poorly understood.

A large body of theoretical work considers impulsivity as a form of a trade-off between self-control and impulsive systems (Whiteside & Lynam, 2001; Dalley et al., 2011; Sharma et al., 2014). Behaviourally, impulsive responding during tasks probing inhibition and discounting is characterised by commission errors or fast reaction time (Bari & Robbins, 2013; Ioannidis et al., 2019) and steeper discounting of future rewards (Frost & McNaughton, 2017), respectively. However, given the trade-off between control and error-sensitive systems on the neural level in cognitive control (Duckworth et al., 2018), a comprehensive account of the neural mechanisms behind impulsivity ought to capture both regions related to 'impulsive' error responses as well as those linked to 'controlled' correct responses within each domain. In the literature, successful inhibition has been associated with the anterior insula, medial frontal cortex and right frontoparietal regions (Zhang et al., 2017; Cieslik et al., 2023). Conversely, the posterior medial frontal cortex covering the pre-supplementary motor area (pre-SMA) and anterior midcingulate cortex (aMCC), dorsal posterior cingulate cortex, and thalamus are believed to play an important role in error monitoring as activity in these regions has been reliably found during errors of commission (Ullsperger et al., 2014; Cieslik et al., 2023). Functional dissociations between impulsive and controlled responding during the delay discounting task are less clear. The ventral striatum, ventromedial PFC and anterior cingulate cortex (ACC) have been implicated in choices of smaller sooner (SS) rewards or steeper discounting; conversely, dorsolateral PFC and right parietal regions have been associated with choices of larger later rewards (LL) and shallower discounting (Schüller et al., 2019; Noda et al., 2020).

While neuroimaging evidence points to two largely distinct functional systems associated with response inhibition and DCS, investigations on the neurochemical level are more mixed, with several neurotransmitter systems believed to play a significant role in modulating impulsivity (Chamberlain & Sahakian, 2007; Dalley et al., 2011; Dalley & Robbins, 2017). Psychostimulant drugs used to treat ADHD such as methylphenidate block the reuptake of dopamine and norepinephrine. In most patients, they substantially reduce symptoms and can improve response inhibition even in healthy individuals (Aron & Poldrack, 2006; Hanwella et al., 2011; Nagashima et al., 2014). Functionally, these improvements may be

partly ascribed to increased right inferior frontal and insula activation (Rubia et al., 2014). In rodents, atomoxetine, a selective norepinephrine reuptake inhibitor reduces delay discounting and enhances inhibition (Robinson et al., 2008). Outside psychostimulants, dopamine is classically associated with addiction (Berke & Hyman, 2000; Wise & Robble, 2020) and has been suggested as a major candidate for passing reward prediction errors within the valuation system (Nasser et al., 2017). Findings from the animal literature show that lesions to the nucleus accumbens - a dopamine-rich nucleus - increase impulsivity on DCS tasks and may also impair response inhibition (Basar et al., 2010). Finally, there is some evidence for the involvement of serotonin in response inhibition, which is impaired following serotonin depletion (Worbe et al., 2014). It has also been inversely related to aggression, a behavioural manifestation of impulsivity, with serotonin 5HT1A/1B receptor agonists reducing aggressive behaviour(de Boer & Koolhaas, 2005; Duke et al., 2013; da Cunha-Bang & Knudsen, 2021).

Here we aimed to delineate a comprehensive brain network associated with impulsivity using coordinate-based ALE meta-analyses (Turkeltaub et al., 2002; Eickhoff et al., 2009, 2012) to synthesise the pertinent neuroimaging literature. We focus on two cognitive-behavioural dimensions that show consensus across most performance-based models of impulsivity and are most commonly investigated in the neuroimaging literature: delayed consequence sensitivity and response inhibition. To this end, we investigate both the activity associated with impulsive responding (commission errors or choices of SS rewards) and non-impulsive, ‘controlled’ responding (successful inhibition or choices of LL rewards) within each dimension. To capture other relevant processes involved in the execution of the delay discounting task, we also included associations with subjective value (Kable & Glimcher, 2007). Next, we characterised the network organisation using resting-state functional connectivity and graph-theoretical methods in two independent large-scale datasets to uncover the functional architecture of the impulsivity networks. Finally, given the widespread use of neurotransmitter-acting medication to treat conditions with impulsive symptoms (Chamberlain & Sahakian, 2007), we investigated if the impulsivity network function could theoretically be modulated by neurochemistry. To this end, we explored associations between network organisation and receptor density of neurotransmitter systems associated with impulsivity (dopamine, serotonin and norepinephrine) obtained from PET imaging.

Results

Meta-analysis

Delayed-Consequence Sensitivity

Analysis of experiments investigating DCS revealed significant findings only for impulsive responding (i.e. impulsive decision-making: choices of SS over LL and correlation with discounting factor k) and subjective value (correlation and parametric modulation) contrasts. Impulsive responding (Fig 1A) led to consistent activation of the ventromedial prefrontal cortex (VMPFC), left frontal pole (FP), ACC and bilateral ventral caudate extending to the nucleus accumbens hereafter referred to as ventral striatum (VS) (Haber, 2011). Analysis of experiments correlating activity with subjective value revealed convergence in a largely overlapping network (Fig 1B). Conjunction analysis revealed that left VMPFC, bilateral VS and right ACC were common in both meta-analyses. Conversely, contrast analyses showed that only FP was specific to impulsive responding, while subcallosal cingulate cortex (scACC) and posterior cingulate cortex (PCC) were specific to subjective value (S1 and S2 Figs). There were no converging clusters for experiments testing controlled responding (choices of LL over SS). The exclusion of studies that correlated measures of impulsivity such as the discount rate k (thus including only the ‘pure’ SS > LL and LL > SS contrasts) revealed similar results (S3 Fig).

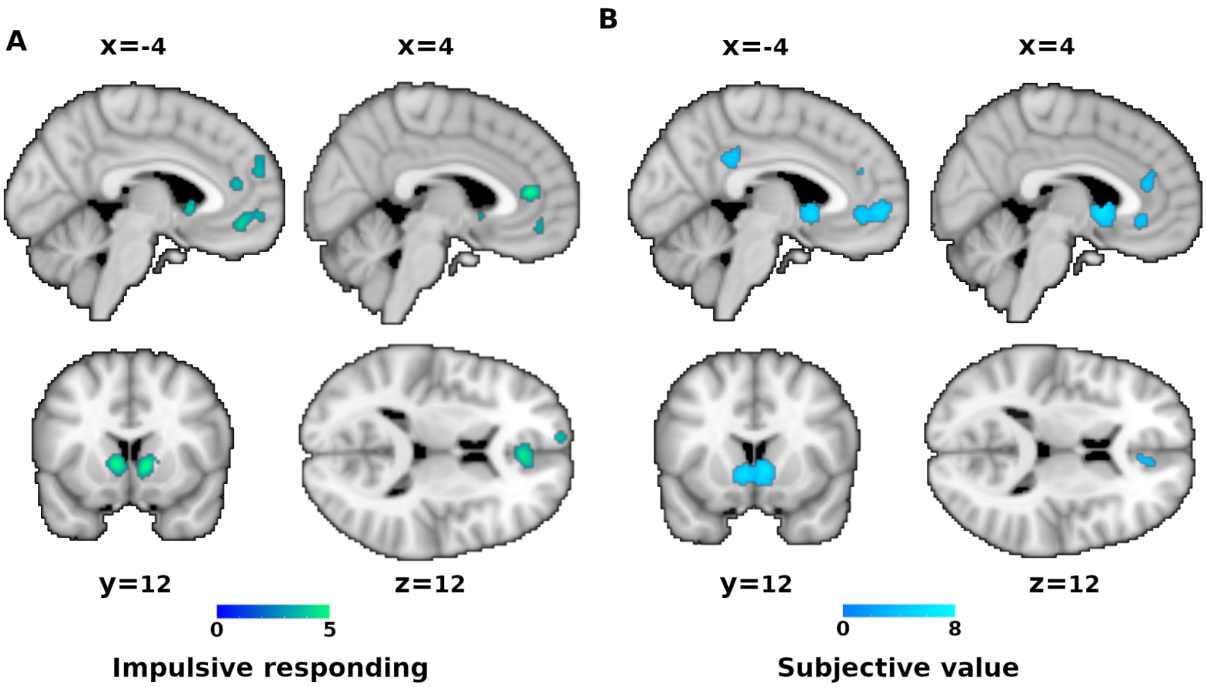

Fig 1. Delayed-consequence sensitivity. Results of the meta-analysis on brain activity correlates of (A) impulsive responding and (B) subjective value. Colour codes z-score.

Response Inhibition

Due to the low number of published experiments that directly contrasted impulsive and controlled responding (i.e. inhibition failure vs. success), a meta-analysis of this contrast was not possible (Eickhoff et al., 2016). We, therefore, computed two meta-analyses of experiments examining brain activation during failed or successful inhibition against baseline and subsequently tested for differences as well as commonalities between them on the meta-analytic level (see Methods for further details). Results of the individual analyses of failed or successful inhibition against baseline can be found in S4 Fig 4. These analyses revealed a widespread network of insular, frontoparietal and subcortical regions in line with previous findings (Cieslik et al., 2023).

The meta-analytic contrast analysis revealed stronger convergence for impulsive responding (failed vs. successful inhibition) in preSMA, aMCC, the right anterior section of the superior frontal gyrus (aSFG) and right supramarginal gyrus (SMG) (Fig 2A orange-yellow). Stronger convergence for controlled responding (successful vs. failed inhibition) was found across the lateral frontal and dorsal premotor cortex (dPMC) in addition to the right temporal and parietal regions, right anterior insula (aI) and left putamen (Fig 2A blue). Fig 2B illustrates the conjunction analysis across the meta-analyses of failed and successful inhibition against baseline.

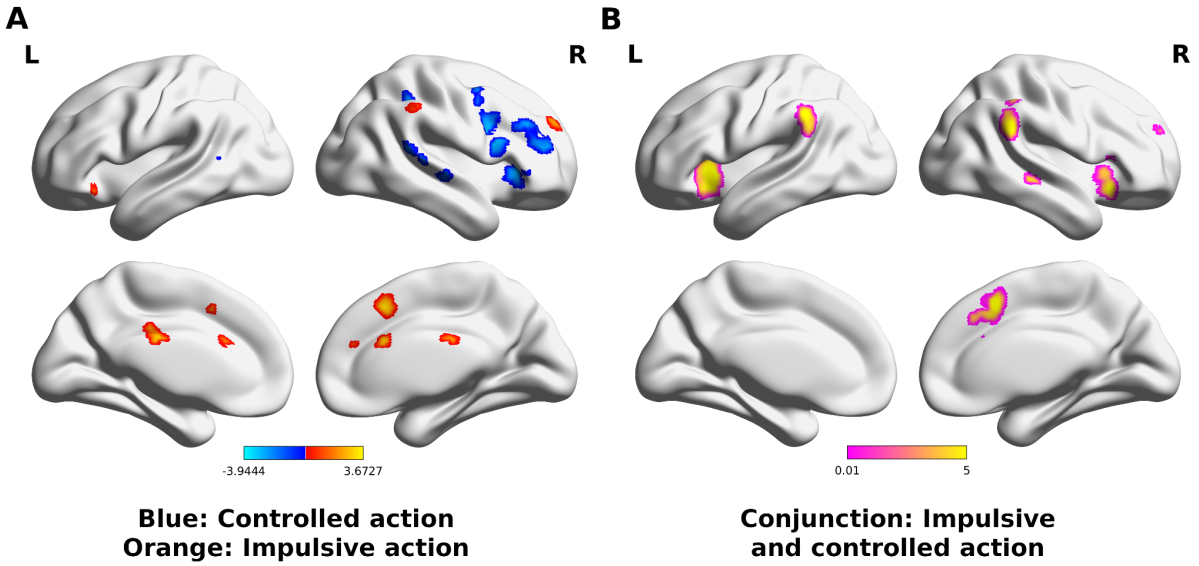

Fig 2. Response Inhibition. Results of (A) meta-analytic contrast and (B) conjunction analyses of successful inhibition > baseline and failure of inhibition > baseline contrast meta-analyses.

Network characterisation

To explore the functional organisation of the resulting meta-analytic regions, we investigated their functional connectivity profiles, community structure and neurochemical properties. The nodes that were used for these analyses are displayed in Fig 3. For MNI coordinates and complete regions labels see Table 1. An overlay with Yeo et al.'s (2011) resting state networks (Fig 3B) shows that nodes from the DCS meta-analyses were primarily located within medial DMN. Combined controlled and impulsive responding nodes were mostly found in the dorsal attention network, while the remaining nodes were distributed over frontoparietal and ventral attention networks (Fig 3B). The extracted meta-analytic nodes and all result maps are available in the ANIMA database: https://anima.fz-juelich.de/studies (Reid et al., 2016).

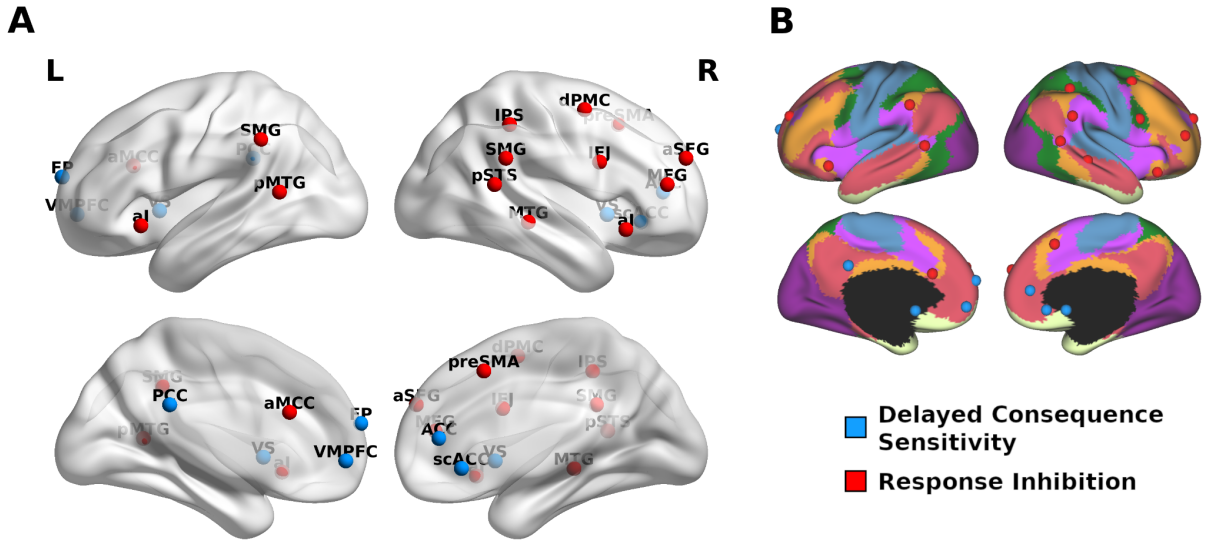

Fig 3. Nodes of the Impulsivity Network. (A) impulsivity network nodes: Delayed consequence sensitivity in blue and response inhibition in red. Panel (B) displays Impulsivity network nodes overlaid over Yeo et al., (2011) resting-state networks: visual (purple), somatomotor (blue), dorsal attention (green), ventral attention (pink), limbic (white), frontoparietal (orange) and default mode (red) networks.

Table 1

Meta-analytic nodes

Region (abbreviation)	Hemisphere	MNI Coordinate		
		x	y	z
Ventral striatum (VS)	Left	-8	10	0
Ventromedial prefrontal cortex (VMPFC)	Left	-4	44	-8
Ventral striatum (VS)	Right	10	12	-2
Anterior cingulate cortex (ACC)	Right	8	42	10
Frontal pole (FP)	Left	-10	62	18
Subcallosal cingulate cortex (scACC)		2	30	-6
Posterior cingulate cortex (PCC)		-2	-38	28
Anterior insula (AI)	Left	-38	20	-8
Anterior insula (AI)	Right	32	22	-10
Pre-supplementary motor area (preSMA)	Right	4	18	48
Supramarginal gyrus (SMG)	Right	60	-42	28
Supramarginal gyrus (SMG)	Left	-60	-44	36
Middle temporal gyrus (MTG)	Right	54	-30	-6
Superior frontal gyrus (SFG)	Right	24	54	28
Anterior midcingulate cortex (aMCC)		0	24	24
Inferior frontal junction (IFJ)	Right	48	8	26
Middle frontal gyrus (MFG)	Right	40	44	14
Posterior superior temporal sulcus (pSTS)	Right	58	-48	14
Intraparietal sulcus (IPS)	Right	40	-40	46
Posterior middle temporal gyrus (pMTG)	Left	-58	-52	12
Dorsal premotor cortex (dPMC)	Right	38	0	54

Abbreviations: MNI – Montreal Neurological Institute

Community Structure

To detect communities within the impulsivity network we used the Louvain community detection algorithm (Blondel et al., 2008), which divides a network into non-overlapping groups of nodes. Using estimates of resting-state FC between all network nodes from 528 participants of the publicly available Nathan Kline Institute dataset (eNKI) (Nooner et al.,

2012) as edges, this approach yielded a four-community solution (Fig 4A). Repeating this procedure 1000 times, we observed a strong convergence across solutions suggesting that our four-community solution was not restricted to a local maximum in the solution space (Fig 4B). To evaluate the robustness of our findings further, we repeated the community detection analysis using a different set of 316 unrelated subjects from the Human Connectome Project dataset (HCP) (Van Essen et al., 2013) and found an identical community structure (S5 Fig).

The first, fronto-medial community consisted of all DCS nodes in the network (VS, VMPFC, ACC, frontal pole, PCC and scACC). Regions related to response inhibition were subdivided into three different communities. In the order of appearance in Fig 4, the first of these comprised mostly regions of the so-called salience network (Seeley et al., 2007), i.e. bilateral aI and aMCC as well as right SFG. The next community spanned mainly right-lateralized frontoparietal regions (IFJ, MFG, dPMC and IPS) as well as preSMA. The last community consisted of temporoparietal regions (bilateral SMG, MTG, and pSTS). Interestingly, the cingulo-insular community was the only community to display positive coupling with regions of both the DCS and response inhibition networks.

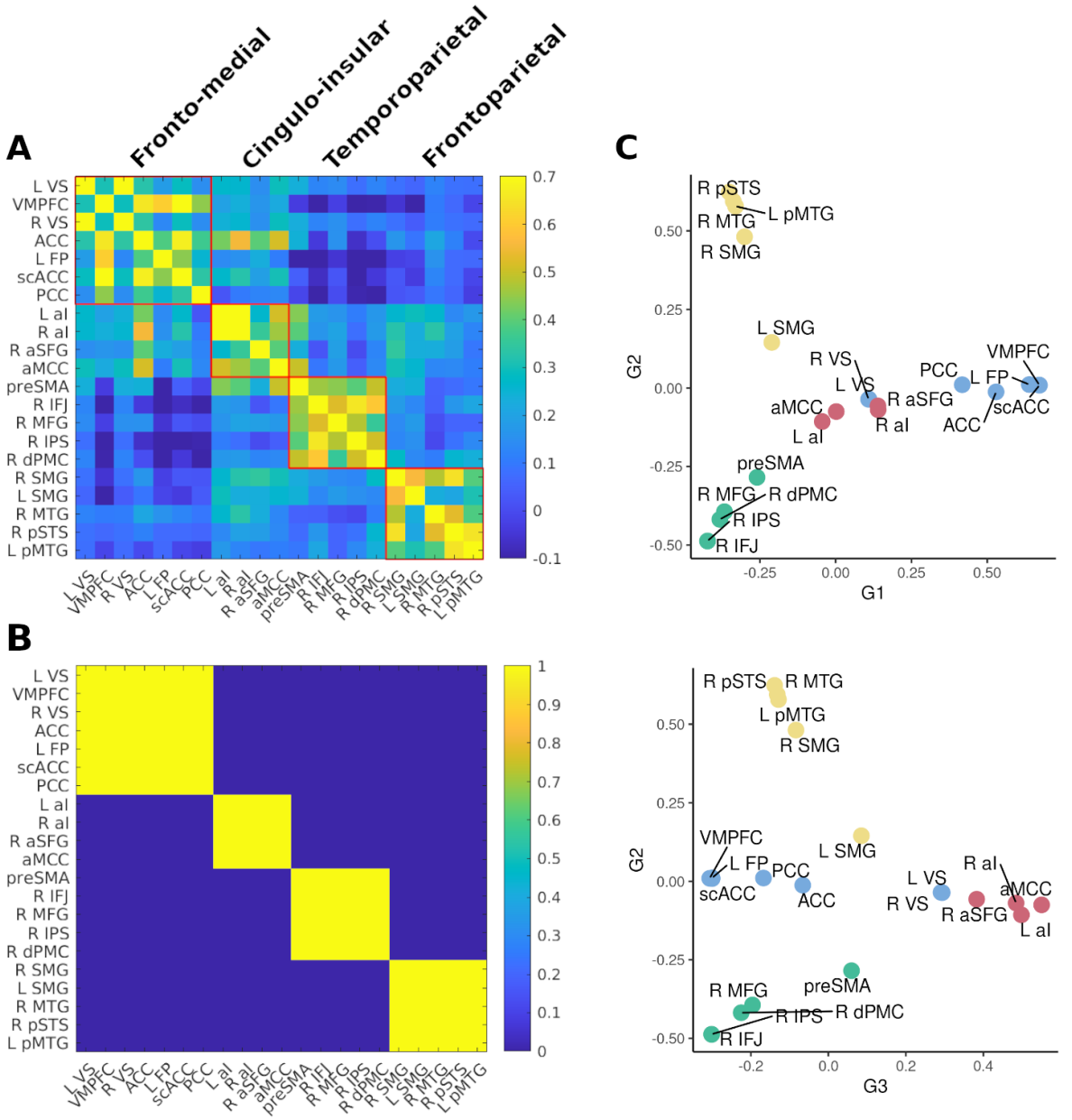

Fig 4. Impulsivity Network Communities. Panel A shows connectivity-based communities in the discovery sample (eNKI). The agreement matrix in panel B displays the consensus across 1000 repetitions of the community detection. Legend refers to the proportion of overlapping community solutions. Seed-voxel connectivity gradients are displayed in panel C. For a 3D depiction of the three components in C see: https://github.com/MartinGell/Impulsivity_networks

Finally, we investigated the robustness of the resulting communities by a complementary whole-brain analysis. Here, principal component analysis of the pairwise similarity between maps of seed-to-voxel connectivity of the meta-analytic nodes was used to explore the dimensions along which they were organised in relation to the rest of the brain (for scree plot see S6 Fig). The initial three components that explained the most variance showed loadings that were in strong agreement with our community detection results suggesting the node-to-brain interactions paralleled node-to-node relationships (Fig 4C). The first principal component showed that DCS nodes (except VS) displayed affinity in their connectivity with the rest of the brain while being dissimilar to the response inhibition regions. Similar properties were observed for the cingulo-insular, frontoparietal and temporoparietal communities along the second and third gradient revealing the closeness of within-community nodes in their whole-brain connectivity profiles. Results did not differ with varying sparsity or decomposition parameters.

Network Organisation Related to Receptor Density

Finally, we examined if network organisation was associated with neurotransmitters related to impulsivity across domains (Dalley & Robbins, 2017). In particular, given our systems approach, we were interested if the interactions between network nodes within and between communities are related to dopamine and serotonin receptor density as well as norepinephrine transporter density derived from PET imaging. Network organisation was assessed using two graph-theoretical measures: (i) within-module degree z-score, a measure of how well a node is connected to other nodes in its community and (ii) participation coefficient, a measure of how well a node is connected to other modules (Guimerà & Nunes Amaral, 2005). Only serotonin 5HT1a receptor density showed a positive relation to within-module degree z-score in both samples (eNKI: $\rho = 0.49$, $p = 0.014$; HCP: $\rho = 0.64$, $p = 0.002$), suggesting that node-wise serotonin expression was related to within-module integration (Fig 5A).

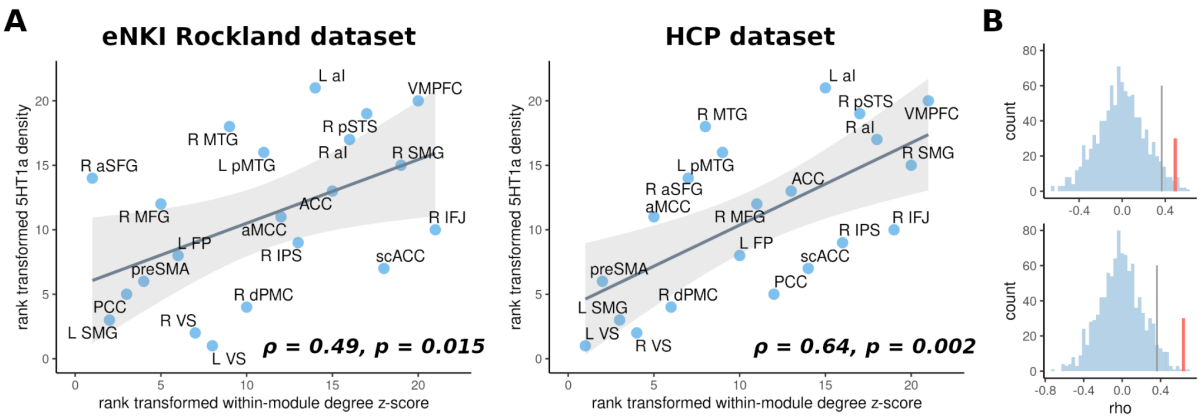

Fig 5. Association of community organisation with serotonin receptor 5HT1a. Panel A shows the relationship between within-module degree z-score (high scores indicate within-network integrator regions) and 5HT1a receptor in the discovery (left) and replication sample (right). Panel B displays permutation-derived null-distributions of correlation coefficients (Spearman's rho) between receptor density and within-module degree z-score in the discovery sample (top) and replication sample (bottom). Observed correlation is marked with a red line and the significance level of 0.05 is indicated by a grey line.

Discussion

The present study investigated brain networks associated with two dimensions of impulsivity: response inhibition and DCS. Using ALE meta-analyses of task-based fMRI studies we provide evidence for two distinct functional systems: one centred in the medial prefrontal cortex, ventral striatum and posterior cingulate cortex involved in DCS, and the second covering right lateral frontal cortex, temporoparietal regions, anterior insula and anterior midcingulate cortex subserving response inhibition. Community detection based on resting-state functional connectivity between all meta-analytically derived nodes in two large independent samples revealed four functional communities. The fronto-medial community included all DCS regions corroborating their dissociation from the other system. Response inhibition, in turn, was fractionated into three networks spanning frontoparietal,

temporoparietal and cingulo-insular regions. Lastly, the integration of individual nodes within those communities calculated in two independent datasets was associated with serotonin receptor density.

Two Systems

The results of our meta-analyses indicate that response inhibition and DCS domains of impulsivity differ not only in terms of their behavioural manifestations (Sharma et al., 2014; Stahl et al., 2014; MacKillop et al., 2016) but also on the neural level. Mirroring the behavioural dichotomy, we found two distinct sets of regions involved in each of the domains. The network of regions associated with DCS was mainly localised within the DMN (Raichle, 2015; Owens et al., 2017), while response inhibition covered the multiple-demand network (Duncan, 2010; Müller et al., 2015; Langner et al., 2018). These findings directly support theoretical accounts proposing separate functional systems for individual impulsivity domains (Strickland & Johnson, 2020) and are in agreement with findings on delay discounting (Frost & McNaughton, 2017; Noda et al., 2020) and response inhibition (Zhang et al., 2017; Cieslik et al., 2023). The current work now provides a more fine-grained overview by differentiating controlled and impulsive processing within each dimension and considering both in the final network definition.

Within the framework of response inhibition, impulsivity has been described as an impairment in executive functioning (with inhibitory control being one of the major executive functions), while from the delayed-gratification perspective, impulsivity has been more associated with motivational processes that underlie decision-making (Bari & Robbins, 2013; Stahl et al., 2014). In line with this, the regions related to DCS identified here, especially vmPFC and ventral striatum, have been previously implicated in value-based decision-making (Haber & Knutson, 2010; Rangel & Hare, 2010). Similarly, regions related to response inhibition have been classically associated with executive functions (Duncan, 2010; Langner et al., 2018; Camilleri et al., 2018). These functional differences echo behavioural findings. Performance on response inhibition and delay discounting tasks are differentially related to treatment outcomes of impulsivity-related disorders (Sheffer et al.,

2012; Stevens et al., 2014), impulsive behaviours such as reactive aggression or drug-taking (Sharma et al., 2014) and to pharmacological intervention (Winstanley et al., 2004; Worbe et al., 2014). For instance, after reviewing the literature, Stevens and colleagues (2014) concluded that retention and treatment success in addiction, a condition believed to be strongly related to impulsivity (de Wit, 2009; Dick et al., 2010), was likely related to performance in monetary incentive delay tasks, but not to commission errors in response inhibition. The present findings, therefore, show that behavioural differentiation between the two dimensions is also mirrored on the neural level by the involvement of two distinct neurocognitive systems.

Four Communities

Activity within the response inhibition and DCS networks has been linked to both behavioural (Aron & Poldrack, 2006; Hariri et al., 2006; Wang et al., 2016) and clinical (Stevens et al., 2014) variability. However, to develop markers of psychopathology, interactions within and between large-scale systems are essential (Castellanos et al., 2013; Bassett et al., 2018). Moreover, co-occurring deficits in both response inhibition and steeper delay discounting within the same individual in conditions like addiction and ADHD are not uncommon (Bickel et al., 2012; Castellanos-Ryan et al., 2014; Ioannidis et al., 2019; Yücel et al., 2019). Thus, the two networks identified here cannot account for most impulsivity-related variability in isolation. A systems perspective that considers both within- and between-system interactions may be necessary to bridge this gap. To this end, we used resting-state functional connectivity between the meta-analytic nodes as well as between the nodes and the rest of the brain to identify their community organisation based on their intrinsic coupling patterns. Supporting our meta-analytic findings, the fronto-medial community comprised all DCS regions, suggesting tight integration. Conversely, response inhibition regions split into three communities (cingulo-insular, temporoparietal and frontoparietal) that strongly resemble previous reports (Camilleri et al., 2018; Langner et al., 2018).

The frontoparietal community corresponded to regions within the dorsal attention (IFJ, dPMC, IPS) and frontoparietal (preSMA, MFG) resting state networks (Yeo et al., 2011). The

dorsal attention network is believed to subserve top-down control of visuospatial attention (Corbetta & Shulman, 2002) including attentional shifting (Kelley et al., 2008), while the preSMA has been implicated in cognitive control (Cole et al., 2013) and motor preparation (Kennerley et al., 2004). Directing attention to expected and relevant stimuli and intentionally enhancing the processing of these stimuli when they occur subserved by the DAN may thus enable the appropriate initiation or inhibition of actions when appropriate (such as when a stop or no-go signal appears). With the exception of the right MTG (located in the DMN), the temporoparietal community (bilateral SMG and STS) covered regions located in the posterior ventral attention network. The TPJ, which covers most of the community, has been argued to underlie contextual updating more generally (Geng & Vossel, 2013) and updating responses from action execution to action inhibition during the stop-signal task more specifically (Cieslik et al., 2015). Thus, inefficient updating or transfer of updated information to motor regions via preSMA may result in slower responses or failures of inhibition commonly observed in high-impulsive individuals (Bari & Robbins, 2013).

The last community displayed tight interactions between the anterior insula, aMCC and aSFG, which have been previously described as the salience network (SN) (Seeley et al., 2007; Gordon et al., 2017). The SN has been associated with detecting important or salient stimuli (Seeley et al., 2007), and is believed to initiate control signals and facilitate switching between higher-order networks (Menon & Uddin, 2010; Goulden et al., 2014). We observed positive associations between the cingulo-insular community and both the frontoparietal and temporoparietal communities supporting its role as a control element within the response inhibition network (for a similar account, see Camillieri et al., 2017). In action inhibition specifically, such top-down signals likely originate from the aMCC which has been previously linked to error monitoring (Ullsperger et al., 2014) and may be crucial to inhibitory planning in the preSMA that displayed a strong association with it. Taken together, by facilitating attention, control, updating and action planning, the three communities together likely produce the required behaviour: to enact or inhibit an impulsive response tendency.

The cingulo-insular community also displayed a positive association with the DCS subsystem. These results are in line with models of the salience network as a control element mediating the dynamic interactions between DMN and frontoparietal networks to facilitate goal-directed behaviour (Menon, 2011). Similarly, the cingulo-insular community

may play a role in coordinating the fronto-medial and frontoparietal communities. Aberrant interactions between the frontoparietal networks, DMN and SN (i.e. the triple network model) (Menon, 2011) have been proposed to underlie a number of psychiatric disorders. It is thus not unlikely that impulsivity, itself a transdiagnostic marker (Berlin & Hollander, 2014), is related to the functional integrity of the cingulo-insular, fronto-medial and frontoparietal communities. Supporting this, connectivity between these large-scale systems has been already associated with discounting rate (Chen et al., 2018), ADHD (W. Cai et al., 2018), addiction (Wang, Shen, et al., 2017; Zhang & Volkow, 2019) and impulsive symptoms in Parkinson's disease (Koh et al., 2020). Similarly, findings of aberrant connectivity between the dlPFC (part of the frontoparietal subsystem) and ventral striatum (part of the delay sensitivity subsystem) in substance use disorder (Jollans et al., 2016; Ersche et al., 2020) and pathological gambling (Koehler et al., 2013) may be in part explained by a dysfunctional salience control subsystem. As such, inappropriate disengagement of either the frontoparietal or fronto-medial communities during task execution may result in apparent connectivity changes between them and affect behaviour (Liang et al., 2016; Shine & Poldrack, 2018). Taken together, we propose the multidimensional construct of impulsivity is associated with a broad network including default mode, frontoparietal, temporal and subcortical regions that can be distinguished into four communities. Interactions between these communities suggest that the entire network is ultimately involved in the final behavioural phenotype of impulsivity.

Neurochemistry

To investigate the biological relevance of the identified community organisation, we explored the relationship between integration and segregation of the impulsivity network with the receptor/transporter density of three impulsivity-related transmitter systems of the brain. These analyses revealed that within-community integrator regions display a higher density of the serotonin 5HT1a receptor, suggesting that integration within communities may be modulated by available serotonin. Evidence of serotonin involvement in different impulsivity dimensions is mixed, with the strongest evidence implicating it in response inhibition (Dalley & Robbins, 2017). There is ample evidence for an inverse association between serotonin

levels and aggression, a behavioural manifestation of impulsivity (Duke et al., 2013; Carhart-Harris & Nutt, 2017; da Cunha-Bang & Knudsen, 2021). Specifically, 5HT1A/1B receptor agonists have been shown to reduce aggressive behaviour in many species including humans (Cleare & Bond, 2000; Sperry et al., 2003; de Boer & Koolhaas, 2005; Popova et al., 2007), while a reduction in firing has been associated with increased aggression (Audero et al., 2013). Activation within regions that exhibited high within-community integration like the anterior insula and medial PFC has been previously proposed to regulate aggression (Blair et al., 2021). The present findings, therefore, indicate that serotonergic modulation of behaviours such as aggression might be associated with facilitated integration within communities. Interestingly, neither the norepinephrine transporter nor dopamine receptor density was found to be related to functional network organisation. Our results thus indicate that the mechanism of action of norepinephrine and dopamine on function may not be through altering network integration or segregation, warranting further investigation.

Limitations and Outlook

The present investigation focused on neural responding during the execution of cognitive tasks measuring impulsivity. It, therefore, does not warrant any conclusions on the relationship between brain activity and self-report measures of impulsivity, as questionnaire-derived trait assessments often demonstrate limited correlations with performance-based assessments of impulsivity (Sharma et al., 2014). Future work may investigate whether individual differences in trait impulsivity relate to the network identified here. In the present work, we focused on the two best-characterised dimensions of impulsivity that were also most commonly investigated with fMRI. Some models suggest sustained attention (the ability to keep one's attention focused over time) and risk-taking as additional components of impulsivity (Strickland & Johnson, 2020); however, there is substantial variance in proposed behavioural assessments. A meta-analysis of fMRI studies investigating sustained attention by Langner & Eickhoff (2013) has reported activations in regions largely overlapping with those identified here in the response inhibition network. Risky behaviours rarely play a substantial role in theoretical models of impulsivity and have

been measured using the probability discounting task and Balloon Analog Risk Task (Lejuez et al., 2002). fMRI investigations during these tasks have revealed regions within the DCS network and parts of the multiple demand network (Peters & Büchel, 2009; Schonberg et al., 2012; Miedl et al., 2012; Seaman et al., 2018), suggesting overlapping activation with regions found in our meta-analyses. Therefore, the network described here may provide a largely comprehensive description of the neurocircuitry associated with the multidimensional construct of impulsivity.

Conclusions

Taken together, our findings reinforce insights from previous behavioural research and provide substantial evidence for the multidimensional nature of impulsivity on the neural level. In particular, we identified and characterised two non-overlapping neurocognitive systems linked to processes underlying impulsive and controlled decision-making and action control. Each of these was centred in a distinct large-scale network of brain organisation. The first was located in the default-mode network associated with value-based judgements and goal-directed decision-making, the second was distributed across higher-order networks related to executive functions of action selection, planning and updating. These systems were found to be organised into four specialised communities of medial frontal, cingulo-insular, frontoparietal and temporal regions. Interactions between the communities and their coordination may affect the impulsivity of our behaviour and decision-making, with the modulation of community integration by serotonin emerging as a possible mechanism. Overall, our findings underscore the necessity for a comprehensive dimensional ontology encompassing symptoms, cognitive processes, and neural systems to effectively address impulsivity in a transdiagnostic manner (Berlin & Hollander, 2014). The research domain criteria framework of the NIH (Insel et al., 2010) has already taken steps in such a direction, with reward valuation and response selection/inhibition forming two separate components – but only the latter refers to impulsivity. Such developments, however, have yet to penetrate clinical research and practice.

Methods

Meta-analysis

We performed a literature search using PubMed (https://www.ncbi.nlm.nih.gov/pubmed) and Web of Science (https://webofknowledge.com) for articles published until the 10th March 2021 that investigated brain activation related to either a DCS or response inhibition with fMRI or PET. Additionally, reference tracing of systematic reviews and meta-analyses (on the topics of impulsivity more broadly), as well as response inhibition and delay discounting, specifically was done. The search terms were selected in keeping with the 'pure measures' of impulsivity within each dimension as suggested by Strickland et al. (2020). For DCS, these were: "delay discounting", "temporal discounting", "delayed reward" as well as each of the keywords separately. The database for response inhibition studies using the go/nogo and stop-signal paradigms in adults was obtained from a recent meta-analysis by Cieslik et al. (2023). We enriched this database by adding studies with adolescent participants for which we used the same search terms as presented in Cieslik et al. (2023), namely: "stop signal task", "go no-go task", "go nogo task", "response inhibition", "inhibition", "action withholding", "action cancellation", "action inhibition", "motor inhibition" and "inhibitory control".

We included only results from peer-reviewed fMRI or perfusion PET experiments reporting results of whole-brain group analyses as coordinates in a standard neuroanatomical reference space (Talairach/Tournoux or Montreal Neurological Institute). Results from region-of-interest (ROI) analyses and studies with partial brain coverage were excluded. Only data from healthy participants (including healthy control groups from patient studies) with mean age >= 12 (with an absolute minimum age of individual participants no lower than 10) were retained. Studies with pharmacological interventions, connectivity-based analyses and single-subject reports were excluded. For studies reporting more than one eligible experiment obtained in the same sample, the reported coordinates were pooled to form a single experiment when included in the same meta-analysis (i.e. coordinates from go/nogo and stop-signal tasks in the same subject group were pooled). If each experiment included a different set of participants, coordinates were not pooled. In cases where different studies or

experiments reported results from partly overlapping samples such as in Kable et al. (2007) and Kable et al. (2010), coordinates were pooled to form a single experiment and the smaller sample size of the two original experiments was used as the input to the analysis. In cases where any of the above criteria were unclear from screened publications, the corresponding authors were contacted. Lastly, authors of clinical studies that passed our inclusion criteria but reported pooled activation for clinical and healthy control groups were contacted for data from the healthy control group only. Of these, three authors responded and are indicated in the table of included studies in the supplementary material For a reporting checklist detailing analysis and study selection choices as suggested by Müller et al. (2018), see supplementary table S1.

Our contrasts of interest were, in general, analyses contrasting impulsive with non-impulsive, 'controlled' behaviour and vice versa, as impulsivity in pertinent paradigms is behaviourally expressed by a higher frequency of 'impulsive responding' such as commission errors (failure to inhibit action when necessary) or choices of smaller but sooner rewards (over larger but later ones). To differentiate the two types of contrasts, we refer to contrasts reflecting impulsive behaviour as 'impulsive responding' and to the reverse contrasts reflecting non-impulsive behaviour as 'controlled responding'. Experiments reporting relative deactivations were interpreted as results of the opposite contrast to that specified (e.g., deactivation observed in a smaller sooner > larger later rewards contrast was interpreted as activation associated with larger later > smaller sooner rewards) unless otherwise specified in the respective publication. A detailed description of the selected contrasts for each impulsivity dimension is provided below. After the exclusion of unsuitable studies (see Fig. 1), the final sample consisted of 46 studies reporting 47 experiments on delayed-consequence sensitivity (21 reporting impulsive, 26 controlled responding and 24 subjective value) and 101 studies reporting 104 experiments on response inhibition (26 reporting impulsive and 96 controlled responding). Details on all studies included can be found in the supplementary material.

1. Delayed-consequence sensitivity

Experiments were separated into 3 categories: impulsive responding, controlled responding and subjective value and separate meta-analyses were calculated for each category. For

impulsive responding, results of smaller sooner (SS) > larger later (LL) rewards, immediate > delayed choice, and $\beta > \delta$ contrasts were selected, while for controlled responding the opposite contrasts were included, namely: LL > SS, delay > immediate and $\delta > \beta$. β is theorised to reflect an 'impatient system' and is usually coded in fMRI paradigms as blocks of trials where immediate rewards are possible, while δ represents the 'patient system' and is coded as blocks of choices where only delayed choices occur (Laibson, 1997; McClure et al., 2004). We further included contrasts that tested for across-participant correlations between brain activity and the temporal discount parameter *k* (or similar constructs reflecting the degree to which individuals discount future rewards). As higher *k* indicates stronger impulsive tendencies, positive correlations were included in the meta-analysis of impulsive responding and negative correlations in the analysis of controlled responding. Lastly, choices between SS and LL rewards are highly influenced by the perceived subjective value of the rewards, which is believed to track the valuation processes during delay discounting tasks (Kable & Glimcher, 2007; Schüller et al., 2019). Therefore, parametric modulation and correlation of activity with subjective value were coded as a third category of experiments.

2. Response Inhibition

Following the guidelines for performing well-powered fMRI meta-analyses (Eickhoff et al., 2016; Müller et al., 2018), we were not able to find a suitable amount of experiments reporting results of the direct comparison between impulsive and controlled responding (with only 15 for impulsive > controlled and 7 experiments for controlled > impulsive). We, therefore, selected experiments contrasting against control conditions not reflecting impulsivity like 'Go' conditions (no need for inhibition) or rest/fixation and then calculated the contrast of interest (impulsive vs. controlled) on the meta-analytical level. In particular, experiments that contrasted brain activation during commission errors or successful inhibition against baseline (Go, fixation or rest) were included. First, we calculated separate meta-analyses for impulsive responding > baseline and controlled responding > baseline, respectively. Next, we compared impulsive and controlled responding by calculating meta-analytic contrasts and conjunction analyses (for further details, see section Activation Likelihood Estimation below).

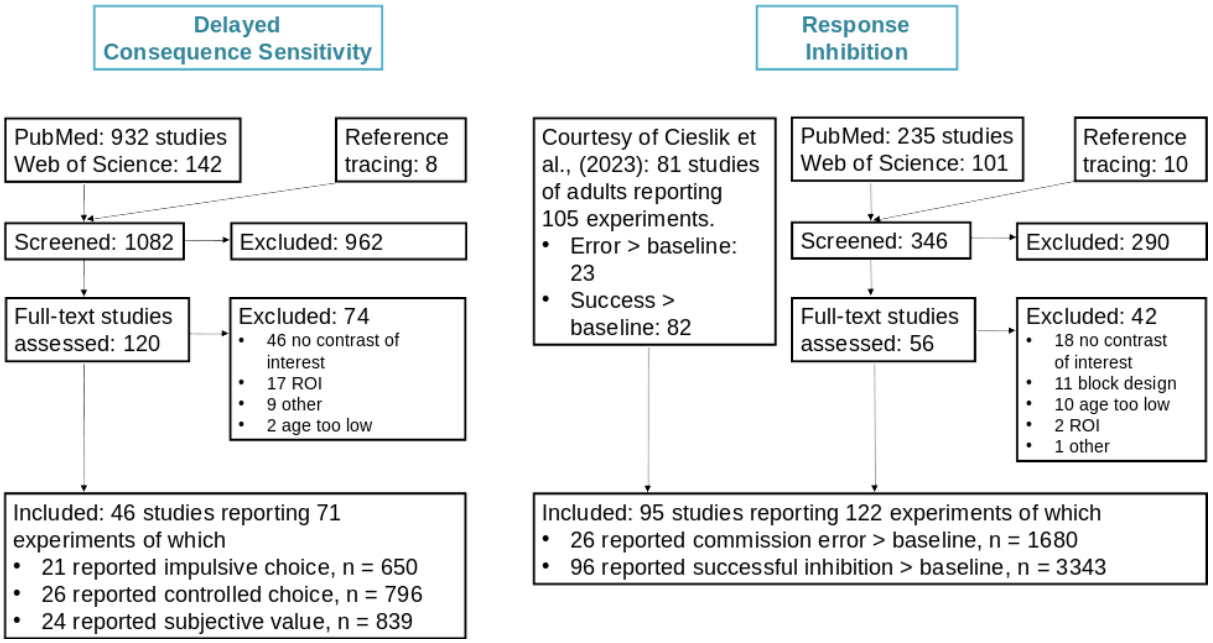

Figure 6. Flow Diagram of Study Selection for the Meta-analyses. The total sample size for each contrast is denoted with n. In the flow diagram, 'study' refers to a publication and by 'experiment' the specific contrast reported. In case a study reported multiple contrasts within the same category (e.g. stop signa), it was counted as one experiment.

Activation likelihood estimation (ALE)

All meta-analyses were performed using the ALE algorithm for coordinate-based meta-analysis of neuroimaging studies (Turkeltaub et al., 2002; Eickhoff et al., 2009, 2012) that was implemented using in-house Matlab (version 2017a) tools. The analyses were executed as described previously (Kogler et al., 2020) and according to the best-practice guidelines for neuroimaging meta-analyses (Müller et al., 2018). The ALE algorithm aims to identify brain areas where activity across many experiments converges more strongly than would be expected from a random spatial association. Briefly, to reflect the spatial

uncertainty of activations, each activation focus was modelled as a centre of a 3D Gaussian probability distribution based on empirical data of between-template and between-subject variance. The between-subject variance was weighted by the number of participants in the respective experiment. For a given experiment, the probability distributions of each focus were then combined and a union over all experiments‘ activation maps were computed. This yielded a voxelwise estimated activation likelihood map (i.e. a map of ALE scores), which describes the degree of spatial convergence across all experiments. Lastly, in order to identify ‘true‘ convergence, the ALE scores were compared to an analytically derived null distribution (Eickhoff et al., 2012) reflecting random spatial associations between activation maps for all experiments. Results were thresholded at $p < .05$ (family-wise error-corrected at cluster level with voxel-level cluster inclusion threshold at $p < .001$; Eickhoff et al., 2016).

Meta-analytic contrast and conjunction analyses

Contrast and conjunction analyses were calculated between meta-analytic results within each behavioural dimension (i.e. for impulsive vs. controlled) to directly compare impulsive and controlled responding for response inhibition and simplify peak extraction (see below). Commonalities between the two meta-analyses were assessed via conjunction analysis, which identifies voxels with significant convergence in both meta-analyses, calculated as the intersection of the cFWE-thresholded result maps. A cluster extent threshold of at least 5 voxels was applied to the resulting conjunction maps.

For contrast analyses, the voxel-wise differences between ALE scores of two meta-analyses were calculated and compared to a null distribution of difference scores. This null distribution was derived by pooling all experiments from the two meta-analyses and randomly dividing them into two groups of the same sample size as the original sets. This procedure was repeated 25,000 times to yield an empirical null distribution of ALE-score differences which the observed difference in ALE scores was tested against. The resulting voxel-wise nonparametric p values were thresholded at $p < 0.05$, cluster extent threshold of at least 5 voxels. While for delayed consequence sensitivity the number of included experiments was quite similar, for response inhibition the meta-analyses of controlled versus impulsive responding were unbalanced (96 vs 26 experiments). To accommodate for the higher power

of the controlled responding > baseline meta-analysis, we employed a subsampling procedure described in detail in the supplementary methods.

Peak extraction

Next, we created a network comprising all regions involved in response inhibition (impulsive and controlled responding) and DCS (impulsive responding and subjective value). We thus combined the peaks of all meta-analytical networks into one single network. Peaks were extracted from the conjunction and contrast analyses between the meta-analyses of each behavioural task dimension (as described above). Thus, the peaks were on the one hand based on those regions that were found to be involved in more than one meta-analysis as well as those that showed stronger convergence in one compared to another meta-analysis (within the DCS and response inhibition domain, respectively). Peaks that lay in grey matter were thus extracted from the respective conjunction and contrast maps using fsl5 (Smith et al., 2004) [cluster] command with the minimum distance between peaks set to 15 mm. For peaks coming from different maps (for example conjunction and contrast maps) that were less than 15 mm apart from each other, we included only the peak with the higher z-score (Nostro et al., 2018). The extracted meta-analytic nodes and all result maps are available in the ANIMA database (Reid et al., 2016): https://anima.fz-juelich.de/studies

Follow-up connectivity analyses for network characterisation

Participants

For all connectivity modelling, two different datasets were used: one served as the discovery sample and one for replicating results. The discovery sample was chosen based on its cross-sectional design reflecting the sample used in the meta-analysis. This allowed us to

use the same cut-off age range of 10 to 75 years present in the meta-analysis. Written consent from all subjects and ethics approval was obtained locally at both sites. Joint reanalysis of the anonymized data was approved by the ethics committee of the Heinrich Heine University Düsseldorf (study ID: 2018-317-RetroDEuA).

As a discovery sample we used the the extended Nathan Kline Institute Rockland dataset (Nooner et al., 2012). This amounted to resting-state and anatomical (f)MRI data of 608 healthy subjects (395 female) aged 10-75 years. Only data from participants who had completed the full 10 min of scanning without excessive movement (defined here as mean framewise displacement of ≤ 0.5 mm) were included in further analyses, resulting in a final sample of n = 528 healthy subjects (338 female, age: 10-75 years). We used whole-brain T1 anatomical MPRAGE images (TR = 1900 ms; 1 mm isotropic voxels) and resting-state fMRI (rsfMRI) multiband echo-planar imaging (EPI) scans (TR = 1400 ms; 2 mm isotropic voxels; duration = 10 minutes; 440 volumes), acquired on a 3-T Siemens Magnetom scanner.

For replication, the minimally preprocessed data of a sample of unrelated healthy subjects (n = 339, 184 female, aged 22-35 years) were obtained from the full release of the Human Connectome Project dataset (Van Essen et al., 2013). We excluded participants with incomplete resting-state scans or excessive movement (mean framewise displacement of > 0.2 mm as used previously by e.g., Yang et al., 2016) resulting in a final sample of n = 336 subjects (183 females, age: 22-35 years). The rsfMRI HCP scanning protocol involved acquiring whole-brain multiband gradient-echo EPI volumes on a 3-T Siemens “Connectome Skyra” scanner (TR = 720 ms, 2 mm isotropic voxels). Four rsfMRI sessions with 1,200 volumes in total (14 min and 24 s) were acquired over two consecutive days, with one left-to-right (LR) and one right-to-left (RL) encoding direction acquired on each day. For the purposes of replicating our findings based on the eNKI sample, only data from the first session on the first day was used (so-called “rest1LR”).

Preprocessing

The eNKI data were preprocessed using fMRIPrep version 20.1.1 (Esteban et al., 2018; fMRIPrep 2020), which is based on Nipype version 1.5.0 (Gorgolewski et al., 2011; Nipype 2017). For a detailed description of each step, see supplementary methods. Briefly, this included skull-stripping, head-motion correction and slice-time correction. The BOLD images were then coregistered to the native space of the subjects' T1w image, normalised to MNI space and motion-corrected.

The HCP data used here were minimally preprocessed. The preprocessing pipeline has been described in detail elsewhere (Glasser et al., 2016). Briefly, this included gradient distortion correction, image distortion correction, registration to subjects' T1w image and to MNI standard space followed by intensity normalisation of the acquired rsfMRI images, and ICA FIX denoising (Salimi-Khorshidi et al., 2014).

For both datasets, additional denoising steps were undertaken using fMRIPrep output files or data provided by the HCP and in-house scripts in MATLAB (version 2019b). First, we regressed mean time courses of 2 tissue classes (white matter and cerebrospinal fluid) and the global signal which has been shown to reduce motion-related artefacts (Ciric et al., 2017). Next, data were linearly detrended, bandpass-filtered at 0.01 – 0.1 Hz and spatially smoothed using a Gaussian kernel of FWHM = 5 mm.

Community detection and network measures

After averaging the time series from all grey-matter voxels within 5-mm spheres around the meta-analytically derived coordinates, node-to-node functional connectivity was calculated as the Pearson correlation between the time courses of each node. The resulting connectivity matrix for each participant was z-scored using Fisher's-z transformation and averaged across all participants. We employed the Louvain algorithm (Blondel et al., 2008),

a stochastic method, for identifying distinct communities within a network by optimising Q, a modularity score (Betzel, 2020). For this, we used the community_louvain.m function from the Matlab-based Brain Connectivity Toolbox (Rubinov & Sporns, 2010). The averaged connectivity matrix between all meta-analytic nodes was used as the input. We finetuned the community assignment by using the communities resulting from applying the algorithm to the connectivity matrix as an additional input and repeated the procedure until Q remained constant. Given the greedy stochastic nature of the algorithm (Good et al., 2010), community assignment was evaluated by repeating the procedure 1000 times to obtain an agreement matrix. To evaluate the node roles in the final community partition, we calculated the participation coefficient (participation_coef_sign.m) and within-module degree z-score (module_degree_zscore.m). The participation coefficient identifies if a node‘s connections are distributed across communities or clustered within a community and reflects between-module integration at high values and segregation at low values. The within-module degree z-score describes the connectedness of a node to its own community relative to other nodes in the same community and thus reflects within-module integration.

Seed-voxel connectivity gradients

While the above-described community detection identifies communities based on node-to-node connectivity profiles, we additionally investigated network organisation based on the ‘node-to-rest of the brain’ connectivity profiles (i.e. seed-to-voxel correlations). In order to determine if nodes located in the same community displayed similar connectivity to the rest of the brain, we identified principal axes of variation in the connectivity profiles across all nodes. This technique was recently used to determine spatial variation in both node-to-node (Margulies et al., 2016) and seed-to-voxel (J. Zhang et al., 2019) connectivity, as well as structural characteristics such as microstructure (Paquola et al., 2019) across the cortex. Seed-to-voxel connectivity was calculated as Pearson correlation between the mean time courses of each node and all remaining grey-matter voxels in the brain, resulting in one connectivity map for each node per subject. Maps for each node were Fisher Z-transformed before averaging across participants. Next, we constructed a node-by-node similarity matrix, by transforming the averaged (3D) seed-to-voxel connectivity map of each node into a vector

and correlating the resulting vectors from each node (resulting in a 21x21 matrix). To this matrix, we then applied principal component analysis using the BrainSpace toolbox (Vos de Wael). Only the top 20% of node similarities were retained (i.e. sparsity parameter). The remaining parameters were kept the same as in previous work by Marguilles et al. (2016), with α set to 0.05. We repeated the gradient decomposition using diffusion map embedding (Coifman et al., 2005) and varying levels of sparsity (30% and 40%) in order to confirm our results were not subject to the choice of dimensionality reduction algorithm or parameters.

PET-based receptor density analysis

To investigate the relationship between neurotransmitter receptor/transporter density and community organisation, we used PET-derived whole-brain maps available in the JuSpace toolbox (Dukart et al., 2021) available online (https://www.fz-juelich.de/inm/inm-7/EN/Resources/_doc/JuSpace.html?nn=2463520). For the analysis, we only used receptor and transporter maps for neurotransmitters theoretically related to impulsivity: serotonin, norepinephrine and dopamine (Chamberlain & Sahakian, 2007; Dalley et al., 2011; Dalley & Robbins, 2017). In particular, for serotonin, we utilised the 5HT1a, 5HT1b, 5HT2a and serotonin transporter maps (SERT) (Savli et al., 2012), norepinephrine transporter map NAT (Hesse et al., 2017) for norepinephrine, and D1 (Kaller et al., 2017), D2 (Alakurtti et al., 2015) and dopamine transporter maps (Dukart et al., 2018) for dopamine. All PET maps were acquired from healthy volunteers and rescaled to a minimum of 0 and a maximum of 100, for further details, see Dukart et al. (2021).

First, all the above PET maps were resampled from 3 mm isotropic voxels to 2 mm isotropic voxels using the fsl5 [flirt] command. For each node, we then averaged the receptor density values in all grey matter voxels within 5 mm diameter spheres around each coordinate. Next, the node-wise receptor density was correlated (using Spearman rank correlation) with within-module degree z-score and participation coefficient derived from the community organisation. Correlations that displayed at least moderate effect size (>+-0.3) in both our discovery and replication datasets were then tested against a spatially informed null model for significance using permutation testing. To this end, we created 1000 random networks by

randomly sampling coordinates from a conservative grey-matter mask. To mirror the spatial properties of our impulsivity network in the randomly sampled networks, we restricted the minimum, mean and maximum Euclidean distance between the sampled nodes to be within 1 standard deviation from the impulsivity network‘s minimum, mean and maximum values, respectively. We then calculated Spearman rank correlation between receptor density in nodes of each random network and our empirically derived measures of integration and segregation to estimate a null distribution. The empirical rank correlation was then compared to the estimated null. Correlation coefficients higher than 95% of the random correlations were interpreted as significant. Scripts used for generating random networks are available at https://github.com/MartinGell/random_nets

Code availability

All scripts and resources utilised in the analysis reported here can be accessed in a public repository on github at https://github.com/MartinGell/Impulsivity_networks. All data used is freely available from https://db.humanconnectome.org/app/template/Login.vm and http://fcon_1000.projects.nitrc.org/indi/enhanced/data.html.

Declaration of interests

The authors declare no competing interests.

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Contributions

Martin Gell: Conceptualization, Investigation, Formal analysis, Writing - Original Draft, **Robert Langner**: Methodology, Project administration, Writing - Review & Editing, **Vincent Küppers**: Validation, Data Curation, **Edna C. Cieslik**: Methodology, Resources, **Theodore D. Satterthwaite**: Resources, Supervision, **Simon B. Eickhoff**: Conceptualization, Software, Resources, Supervision, **Veronika I. Müller**: Conceptualization, Methodology, Writing - Review & Editing, Supervision

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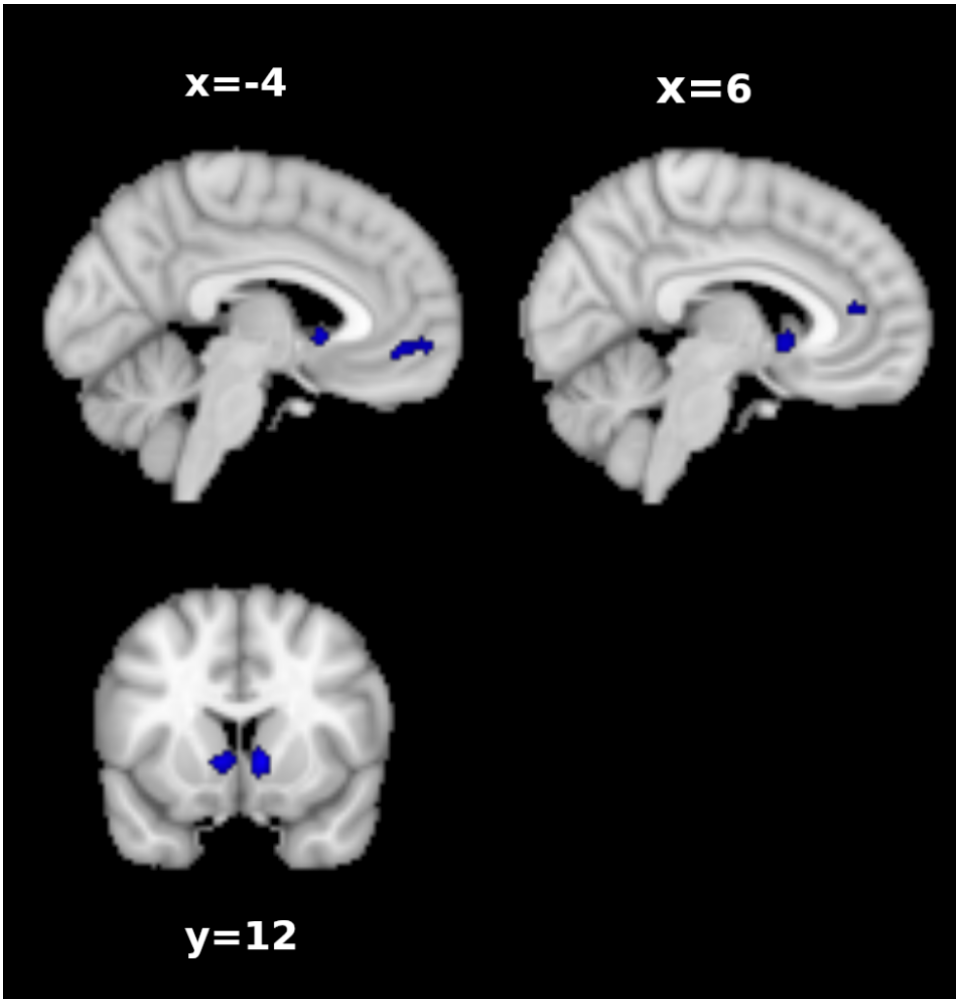
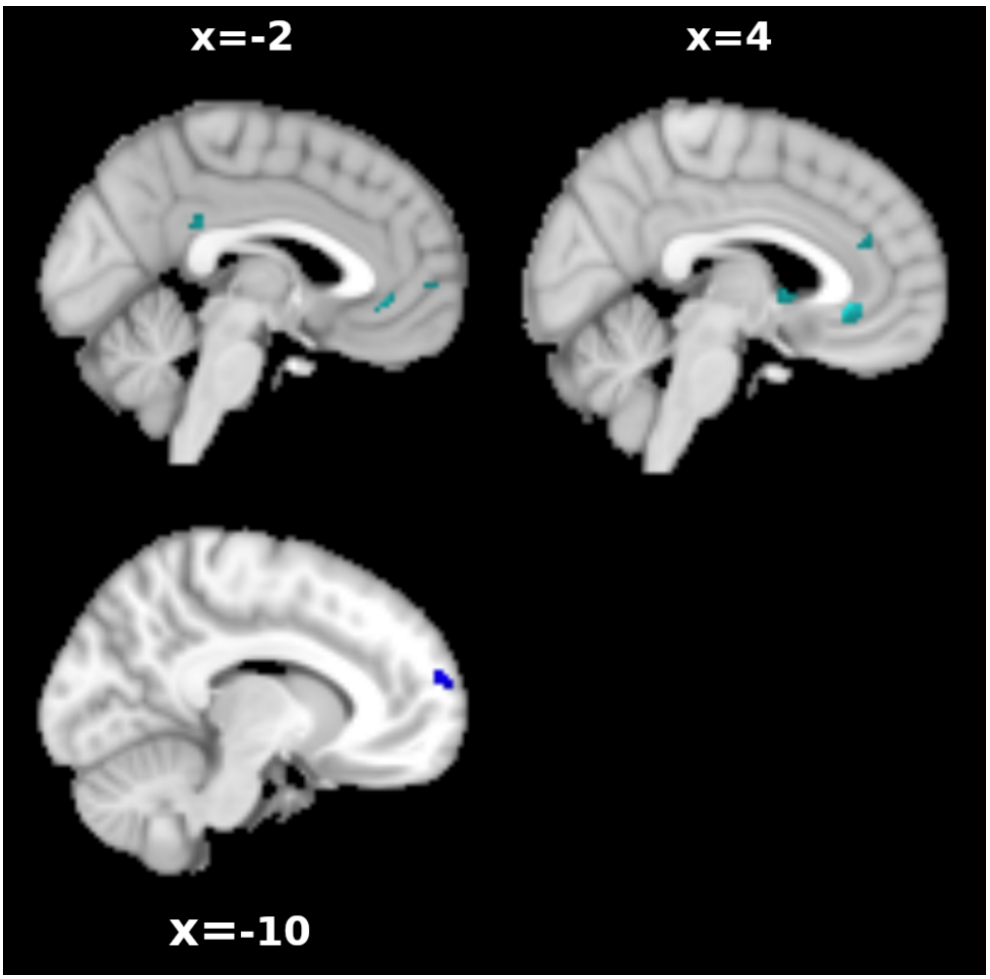
Supplementary Figures and Tables to Gell et al., 2022

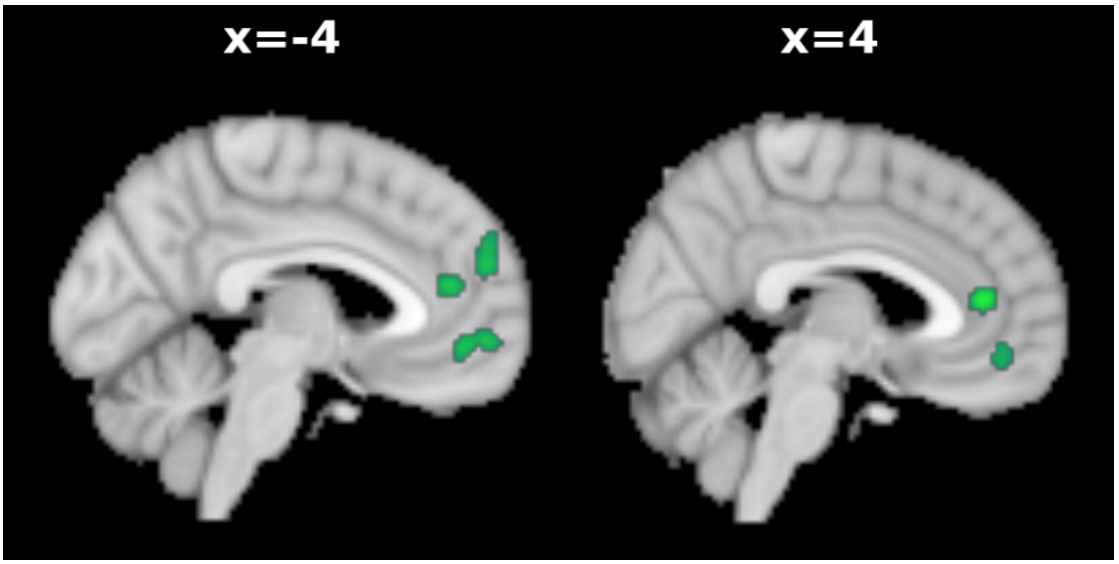
1. Supplementary Figures



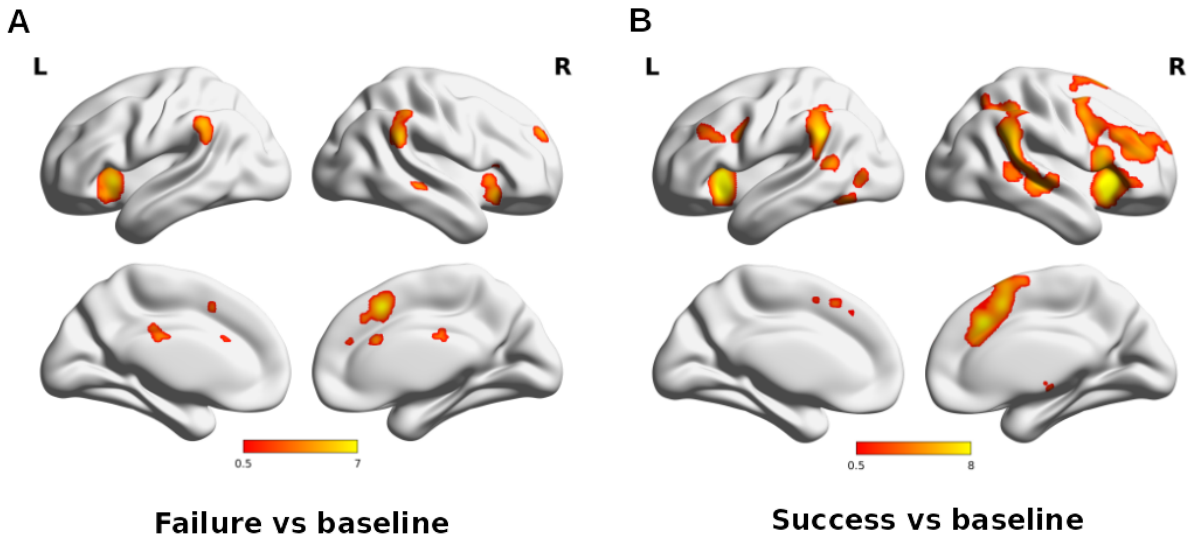
Supplementary Figure 1. Conjunction of impulsive responding and subjective value meta-analyses in DCS.



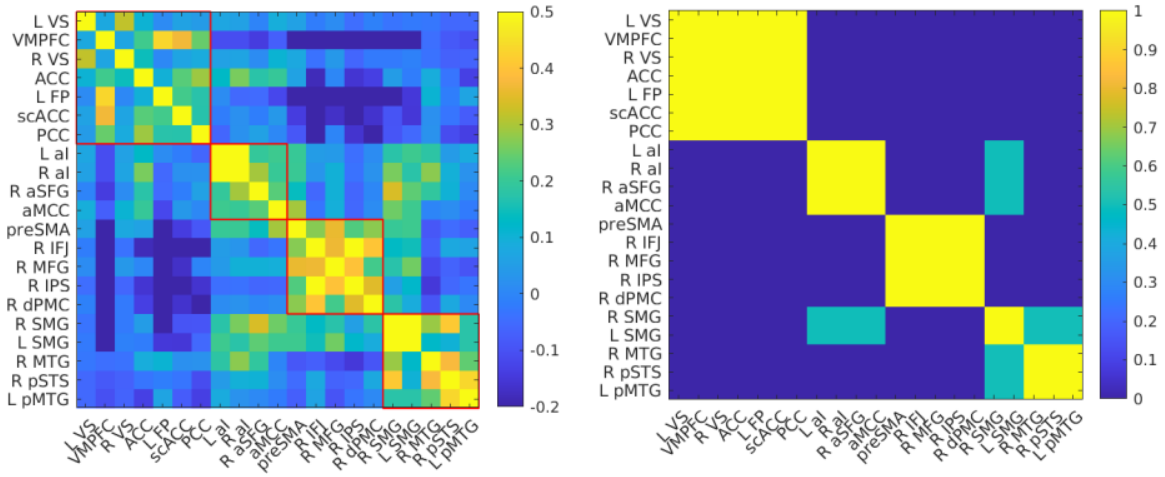
Supplementary Figure 2. Contrast analysis of impulsive responding and subjective value meta-analyses in DCS. Impulsive responding > subjective value in blue and opposite contrast in green.



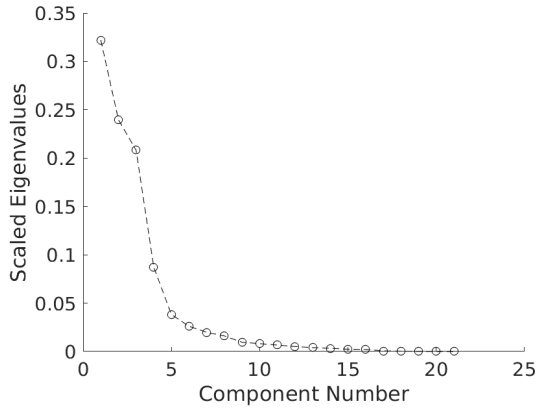
Supplementary Figure 3. Meta-analysis of impulsive responding contrast in DCS without studies correlating activity with k.



Supplementary Figure 4. Results of failure of inhibition against baseline (A) and successful inhibition against baseline (B) meta-analysis.



Supplementary Figure 5. Community detection results in the replication sample. Resulting communities on the left and agreement matrix on the right side (1 = all 1000 repeats yielded the same clustering solution).



Supplementary Figure 6. Scree plot showing the eigenvalues of each component.

Supplementary Tables

Supplementary table 1: Checklist for Neuroimaging Meta-Analyses by Müller et al. (2018)

The research question was specifically defined	YES, and it included the following contrasts: 1) Impulsive choice (smaller sooner > larger later, immediate > delay, β > δ, correlation with k) 2) Controlled choice (larger later > smaller sooner, delay > immediate, δ > β, correlation with k) 3) Subjective value (parametric modulation of and correlation with subjective value) 4) Impulsive action (commission error > go/baseline/rest) 5) Controlled action (successful inhibition > go/baseline/rest) The specific contrasts are reports in the method section
The literature search was systematic	YES, it included the following keywords in the following databases: 1. Delay consequence sensitivity “delay discounting” or “temporal discounting” or “delayed reward” and “fMRI” or “functional magnetic resonance imaging” 2. Response inhibition “stop signal task” or “go nogo task” or “response inhibition” or “inhibition” or "action withholding" or "action cancellation" or "action inhibition" or "motor inhibition" or "inhibitory control" and “fMRI” or “functional magnetic resonance imaging” Databases: PubMed, Web of Science
Detailed inclusion and exclusion criteria were applied	YES, and reasons of non-standard criteria were: Inclusion of: fMRI studies, healthy participants, mean age >= 12, contrast of interest, no region of interest, no pharmacological interventions or connectivity-based analyses
Sample overlap was taken into account	YES, using the following method: In cases of partly overlapping subject groups (e.g. as in the case of Kable 2007 and Kable 2010), coordinates were pooled to form a single experiment and the smaller N of the two original experiments was used as the input to ALE.

All experiments used the same search coverage (state how brain coverage was assessed and how small volume corrections and conjunctions were taken into account)	YES, the search coverage was the following: - whole-brain coverage only - exclusion of ROI studies
Studies are converted to a common reference space	YES, using the following conversion(s): Coordinates reported in Talairach space were converted to MNI space (Lancaster et al., 2007)
Data extraction was conducted by two investigators (ideal case) or double-checked by the same investigator (state how double-checking was performed)	YES, the following authors: - MG, VM, EC checked inclusion criteria - MG extracted coordinates - MG and VK extracted other info: age, sex, sample size, contrast, space - VK and VM double-checked the following data: inclusion criteria, extracted coordinates, all other infos
The paper includes a table with at least the references, basic study description (e.g., for fMRI tasks, stimuli), contrasts and basic sample descriptions (e.g., size, mean age and gender distribution, specific characteristics) of the included studies, source of information (e.g., contact with authors), reference space	YES, and also the following data: - If further information was received by the authors
The study protocol and all analyses was planned before- hand, including the methods and parameters used for inference, correction for multiple testing, etc.	YES. The meta-analysis was not pre-registered; however, all analyses including methods and recommended parameters used for inference were planned before starting the literature search
The paper includes meta-analytic diagnostics	No diagnostics are reported given that all analyses included at least 21 experiments and a cFWE correction. As shown in Eickhoff 2016 results are robust with regard to being driven by only a few experiments.

Supplementary Methods to Gell et al., 2022

1. Main Effect Thresholding and Masking

In order to account for the higher power of the controlled action > baseline/go meta-analysis (i.e. large discrepancy in the number of included studies: 26 in impulsive action vs 103 in controlled action) we used a subsampling procedure. This was achieved by iteratively sub-sampling 26 experiments from the full controlled action database 10 000 times. In each iteration a meta-analysis was calculated and for each voxel an absence or presence of a significant main effect was recorded. Thus a frequency of significant results across the 10 000 iterations for each voxel was obtained. The resulting probabilistic map represents the number of times each voxel participated in a main effect over the permutation procedure as a proportion of the total number of permutations. Finally, this probabilistic map was thresholded at the 90th percentile to remove voxels with a very low probability of participating in a main effect and used to mask the controlled choice main effect map before computing the contrast (see figure x in supplement for illustration of this procedure). An illustration of this procedure is displayed below.

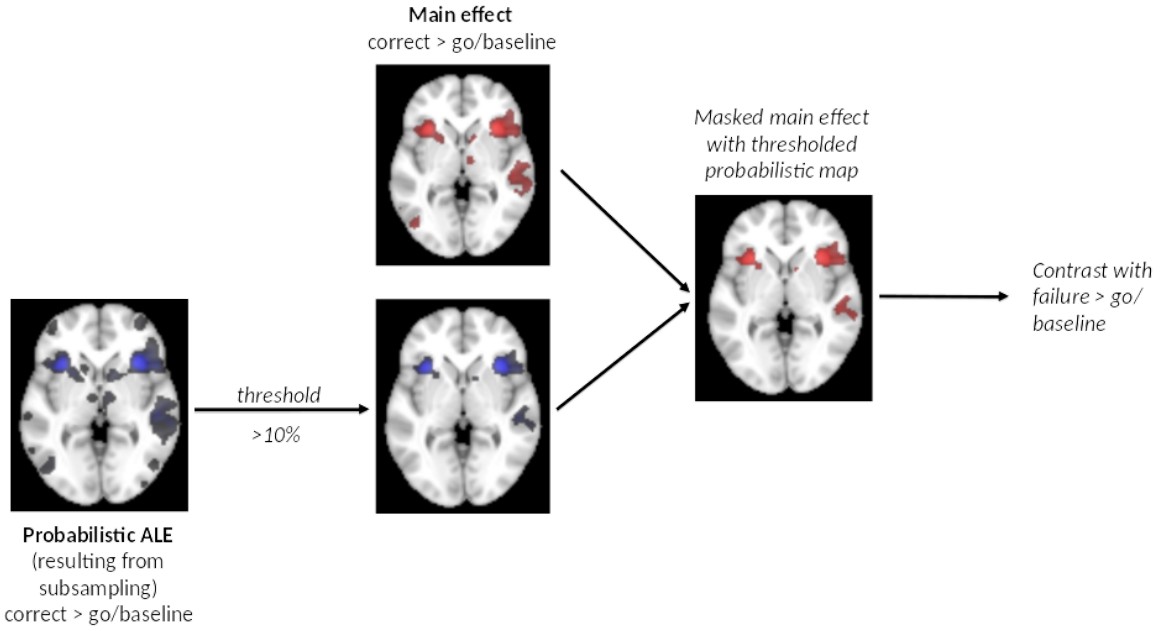


Figure S1. Main effect masking procedure

2. fMRIPrep preprocessing

Anatomical data preprocessing

T1-weighted (T1w) images were corrected for intensity non-uniformity (INU) with `N4BiasFieldCorrection` (Tustison et al., 2010), distributed with ANTs 2.2.0 (Avants, Epstein,

Grossman, and Gee, 2008). The T1w-reference was then skull-stripped with a *Nipype* implementation of the `antsBrainExtraction.sh` workflow (from ANTs), using OASIS30ANTs as target template. Brain tissue segmentation of cerebrospinal fluid (CSF), white-matter (WM) and gray-matter (GM) was performed on the brain-extracted T1w using `fast` [FSL 5.0.9, Zhang, Brady, and Smith, 2001)].

A T1w-reference map was computed after registration of 2 T1w images (after INU-correction) using `mri_robust_template` [FreeSurfer 6.0.1, Reuter, Rosas, and Fischl, 2010)]. Brain surfaces were reconstructed using `recon-all` [FreeSurfer 6.0.1, Dale, Fischl, and Sereno, 1999)], and the brain mask estimated previously was refined with a custom variation of the method to reconcile ANTs-derived and FreeSurfer-derived segmentations of the cortical gray-matter of Mindboggle (Klein et al., 2017). Volume-based spatial normalization to two standard spaces (MNI152NLin6Asym, MNI152NLin2009cAsym) was performed through nonlinear registration with `antsRegistration` (ANTs 2.2.0), using brain-extracted versions of both T1w reference and the T1w template. The following templates were selected for spatial normalization: *FSL's MNI ICBM 152 non-linear 6th Generation Asymmetric Average Brain Stereotaxic Registration Model* [Evans, Janke, Collins, and Baillet, 2012), RRID:SCR_002823; TemplateFlow ID: MNI152NLin6Asym], *ICBM 152 Nonlinear Asymmetrical template version 2009c* [Fonov, Evans, McKinstry, Almli and Collins, et al., 2009), RRID:SCR_008796; TemplateFlow ID: MNI152NLin2009cAsym].

Functional data preprocessing

For the BOLD runs found per subject (across all tasks and sessions), the following preprocessing was performed. First, a reference volume and its skull-stripped version were generated using a custom methodology of *fMRIPrep*. Head-motion parameters with respect to the BOLD reference (transformation matrices, and six corresponding rotation and translation parameters) are estimated before any spatiotemporal filtering using `mcflirt` [FSL 5.0.9, (Jenkinson, Bannister, Brady, and Smith, 2002)]. BOLD runs were slice-time corrected using `3dTshift` from AFNI 20160207 (Cox and Hyde, 1997). Susceptibility distortion correction (SDC) was omitted. The BOLD reference was then co-registered to the T1w reference using `bbregister` (FreeSurfer) which implements boundary-based registration (Greve and Fischl, 2009). Co-registration was configured with six degrees of freedom. The BOLD time-series were resampled onto the following surfaces (FreeSurfer reconstruction nomenclature): *fsaverage*.

The BOLD time-series (including slice-timing correction when applied) were resampled onto their original, native space by applying the transforms to correct for head-motion. These resampled BOLD time-series will be referred to as *preprocessed BOLD in original space*, or just *preprocessed BOLD*. The BOLD time-series were resampled into standard space, generating a *preprocessed BOLD run in MNI152NLin6Asym space*. First, a reference volume and its skull-stripped version were generated using a custom methodology of *fMRIPrep*. *Grayordinates* files (Glasser et al., 2013) containing 91k samples were also generated using the highest-resolution ``fsaverage`` as intermediate standardized surface space.

Automatic removal of motion artifacts using independent component analysis [ICA-AROMA, Pruim et al., (2015)] was performed on the *preprocessed BOLD on MNI space* time-series after removal of non-steady state volumes and spatial smoothing with an isotropic, Gaussian kernel of 6mm FWHM (full-width half-maximum). Corresponding "non-aggresively" denoised runs were produced after such Smoothing. Additionally, the "aggressive" noise-regressors were collected and placed in the corresponding confounds file. Several confounding time-series were calculated based on the *preprocessed BOLD*: framewise displacement (FD), DVARS and three region-wise global signals. FD was computed using two formulations following Power (absolute sum of relative motions, Power et al., (2014)) and Jenkinson (relative root mean square displacement between affines, Jenkinson et al. (2002)). FD and DVARS are calculated for each functional run, both using their implementations in *Nipype* [following the definitions by Power et al., (2014)]. The three global signals are extracted within the CSF, the WM, and the whole-brain masks.
Additionally, a set of physiological regressors were extracted to allow for component-based noise correction [*CompCor*, (Behzadi, Restom, Liau and Liu, 2007)]. Principal components are estimated after high-pass filtering the *preprocessed BOLD* time-series (using a discrete cosine filter with 128s cut-off) for the two *CompCor* variants: temporal (tCompCor) and anatomical (aCompCor). tCompCor components are then calculated from the top 5% variable voxels within a mask covering the subcortical regions. This subcortical mask is obtained by heavily eroding the brain mask, which ensures it does not include cortical GM regions. For aCompCor, components are calculated within the intersection of the aforementioned mask and the union of CSF and WM masks calculated in T1w space, after their projection to the native space of each functional run (using the inverse BOLD-to-T1w transformation). Components are also calculated separately within the WM and CSF masks. For each CompCor decomposition, the *k* components with the largest singular values are retained, such that the retained components' time series are sufficient to explain 50 percent of variance across the nuisance mask (CSF, WM, combined, or temporal). The remaining components are dropped from consideration.

The head-motion estimates calculated in the correction step were also placed within the corresponding confounds file. The confound time series derived from head motion estimates and global signals were expanded with the inclusion of temporal derivatives and quadratic terms for each (Satterthwaite et al., 2013). Frames that exceeded a threshold of 0.5 mm FD or 1.5 standardised DVARS were annotated as motion outliers. All resamplings can be performed with *a single interpolation step* by composing all the pertinent transformations (i.e. head-motion transform matrices, susceptibility distortion correction when available, and co-registrations to anatomical and output spaces). Gridded (volumetric) resamplings were performed using `antsApplyTransforms` (ANTs), configured with Lanczos interpolation to minimize the smoothing effects of other kernels (Lanczos, 1964). Non-gridded (surface) resamplings were performed using `mri_vol2surf` (FreeSurfer).

Many internal operations of *fMRIPrep* use *Nilearn* 0.6.2 (Abraham et al., 2014), mostly within the functional processing workflow. For more details of the pipeline, see [the section corresponding to workflows in *fMRIPrep*'s documentation](https://fmriprep.readthedocs.io/en/latest/workflows.html "FMRIPrep's documentation").

The above boilerplate text was automatically generated by fMRIPrep with the express intention that users should copy and paste this text into their manuscripts *unchanged*. It is released under the [CC0](https://creativecommons.org/publicdomain/zero/1.0/) license.

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Included Studies for Delay Consequence Sensitivity

Impulsive Decision-making					
Author	N	Contrast	Space	Were in-scanner presented rewards adapted based on subjective value?	Source (of coordinates)
Albrecht et al., 2011	28	immediate > delay	TAL	not subject adapted	Table S4
Albrecht et al., 2013	30	now > later	TAL	not subject adapted	Table S4
Banich et al., 2013	29	now > later	MNI	not subject adapted	Table 2
Christakou et al., 2011	40	immediate > delay	TAL	subject adapted	Table 1
de Water et al., 2017	58	correlation of sensitivity to immediate reward in immediate > delay	MNI	not subject adapted	Table S5
Deshpande et al., 2019	54	immediate and delay > delay and delay	MNI	subject adapted	Table S
Elton et al., 2017	95	positive correlation with impulsive choice ration (similar to k) in immediate & delay > control	MNI	not subject adapted	Table 1
Eppinger et al., 2012	30	beta (immediate & delay > all choice options)	TAL	not subject adapted	In text, figure 2
Faralla et al., 2015	28	(immediate > delay) > control	MNI	not subject adapted	Table 5
Hamilton et al. 2020	27	SS > LL	MNI	subject adapted	neurovault
Kable and Glimcher 2010	22	immediate and delay > delay and delay	MNI	subject adapted	Table 1

Luo et al., 2009	37	immediate > delay	MNI	subject adapted	Table 2
Mavrogiorgou et al., 2016	20	immediate > delay	MNI	subject adapted	Table 2
McClure et al., 2004	14	beta (immediate & 0.5*delay)	MNI	not subject adapted	Table S1
Norman et al., 2017	20	immediate > delay	TAL	subject adapted	Table S1
Pine et al., 2009	24	correlation with discount factor D	MNI	not subject adapted	Table S5
Samanez-Larkin et al., 2011	25	beta	TAL	not subject adapted	Table 1
Sripada et al., 2011	20	immediate and delay > delay and delay	MNI	not subject adapted	Table 1
Wittmann et al., 2010	13	immediate > delay	TAL	not subject adapted	table S7
Xu et al., 2009	18	immediate and delay > delay and delay	MNI	not subject adapted	table S2
Zhuang et al., 2020	16	sooner > delayed (main effect of delay decision)	MNI	not subject adapted	Table 2

Controlled Decision-making					
Author	N	Contrast	Space	Were in-scanner presented rewards adapted based on subjective value?	Source (of coordinates)
Banich et al., 2013	29	later > now	MNI	not subject adapted	Table 2
Christakou et al., 2011	40	delay > immediate	TAL	subject adapted	Table 1
de Water et al., 2017	58	delay > immediate	MNI	not subject adapted	Table S4
	58	impatience during delay > immediate	MNI	not subject adapted	Table S5
Deshpande et al., 2019	54	delay and delay > immediate and delay	MNI	subject adapted	Table S
Elton et al., 2017	95	neg corr. with impulsive choice ratio	MNI	not subject adapted	Table 1
Eppinger et al., 2012	30	delta (delay & immediate)	TAL	not subject adapted	In text, Figure 2
Faralla et al., 2015	28	(delay > immediate) > control	MNI	not subject adapted	Table 6
Hamilton et al. 2020	27	LL > SS	MNI	subject adapted	neurovault
Hill et al., 2017	25	delay > immediate	MNI	subject adapted	Table S2
Kable and Glimcher 2007	10	delay > immediate	TAL	subject adapted	Table S5
Kable and Glimcher 2010	22	delay and delay > immediate and delay	TAL	subject adapted	Table 1: reverse contrast
King et al., 2016	36	LL > SS	MNI	subject adapted	authors
Laube et al., 2020	48	LL > SS	MNI	subject adapted	In text, Figure 3

Luo et al., 2009	37	delay > immediate	MNI	subject adapted	Table 2
Luo et al., 2012	21	LL > SS	MNI	subject adapted	Table 1
McClure et al., 2004	14	delta (delay & immediate > baseline)	MNI	not subject adapted	Table S2
Miedl et al., 2015	15	delay > immediate	TAL	not subject adapted	Table 4
Norman et al., 2017	20	delay > immediate	TAL	subject adapted	Table S1
O'Connell et al., 2018	26	delay > immediate	MNI	subject adapted	Figure S3
Samanez-Larkin et al., 2011	25	delta	TAL	not subject adapted	Table 1
Sripada et al., 2011	20	delay > immediate	MNI	not subject adapted	Table 1
Van den Bos et al., 2014	22	LL > SS	MNI	subject adapted	Table 2
Waegeman et al., 2014	41	delay > immediate	MNI	not subject adapted	Table 2
Wang et al., 2017	21	delay > immediate	MNI	not subject adapted	Table 3
Wittmann et al., 2007	13	delay > immediate	TAL	not subject adapted	Table 1
Wittmann et al., 2010	13	delay > immediate	TAL	not subject adapted	Table S7
Zhuang et al., 2020	16	delayed > sooner (main effect of delay decision)	MNI	not subject adapted	Table 2

Subjective value					
Author	N	Contrast	Space	Were in-scanner presented rewards adapted based on subjective value?	Source (of coordinates)
Castrellon et al., 2019	21	subjective value (parametric modulation)	MNI	not subject adapted	Table 2
Cox and Kable 2014	20	subjective value (parametric modulation)	MNI	subject adapted	Table 1
Eppinger et al., 2017	50	correlation with subjective value	TAL	subject adapted	In text, Figure 5
Hare et al., 2014	25	correlation with discounted stimulus value (SV)	MNI	not subject adapted	Table 3
Jimura et al., 2013	43	correlation with subjective value	TAL	subject adapted	Table 1
Kable and Glimcher 2007	10	correlation with subjective value, discount rate	TAL	subject adapted	Table S5
Kable and Glimcher 2010	22	correlation with subjective value	TAL	subject adapted	Table 1
King et al., 2016	36	subjective value (parametric modulation)	MNI	subject adapted	authors
Lempert et al., 2017	35	subjective value (parametric modulation)	MNI	not subject adapted	Table 2
Liu et al., 2012	19	correlation with subjective value	TAL	subject adapted	Table 1
Luo et al., 2012	21	subjective value, stochasticity, discounting factor (k)	MNI	subject adapted	Table S2
Massar et al., 2015	23	correlation with subjective value	TAL	subject adapted	Table 1
Murawski, et al., 2012	13	subjective value > baseline	MNI	subject adapted	Table S5

O'Connell et al., 2018	26	subjective value (parametric modulation)	MNI	subject adapted	Figure S3
Peters and Büchel 2009	22	correlation with subjective value	MNI	subject adapted	Table S2
Peters and Büchel 2010	30	correlation with subjective value	MNI	subject adapted	In text
Pine et al., 2009	24	correlation with subjective value (discounted utility)	MNI	not subject adapted	Table S6
Prevost et al., 2010	18	correlation with subjective value	MNI	subject adapted (force)	Table 1
Ripke et al., 2012	27	subjective value (parametric modulation)	MNI	subject adapted	Tables S1 and S3
Samanez-Larkin et al., 2011	25	correlation with subjective value	TAL	not subject adapted	Table S2
Sasse et al., 2017	22	subjective value (parametric modulation)	MNI	subject adapted	Table 3
Seaman et al., 2018	75	correlation with subjective value	MNI	not subject adapted	Table 3
Sripada et al., 2011	20	correlation with subjective value	MNI	not subject adapted	Table 1
Wang et al., 2014	28	subjective relative value, corr. with value of immediate option	MNI	not subject adapted (adapted from pilot study matching in age and gender)	Table 1
Wiehler et al., 2017	23	subjective value (parametric modulation)	MNI	subject adapted	authors

Included Studies for Response Inhibition

Successful Inhibition Database					
Author	n	contrast	space	paradigm	type of error
Aron and Poldrack, 2006	13	Correct Stop > Correct Go	MNI	stop signal task	SM Table 2
Berkmann et al., 2014	60	Correct Stop > Correct Go	MNI	stop signal task	Table 2
Bobb et al., 2011	13	Correct Stop > Correct Go	MNI	stop signal task	Table 2
Boecker et al., 2011	15	Correct Stop > Correct Go	MNI*	stop signal task	SM Table 1
Boehler et al., 2010	15	Correct Stop > Go	MNI	stop signal task	Table 3
Cai and Leung, 2009	12	Correct Stop > Correct Go	MNI	stop signal task	Table 1
Cai and Leung, 2011	23	Correct Stop > Go	MNI	stop signal task	SM Table 1
Cai et al. 2014	19	Correct Stop > Correct Go	MNI	stop signal task	Table 2
Chen et al., 2015	25	Correct NoGo > Go	MNI	go/no-go	Table 2
Chevrier et al., 2007	14	correct stop > go	TAL	stop signal task	Table 1
Chikara et al., 2018	20	Correct Stop > Correct Go	MNI	stop signal task	Table 3A
Chikazoe et al., 2009a	22	Correct Stop > Correct uncertain go trials	MNI	stop signal task	Table 2
Chikazoe et al., 2009b	25	correct No-Go > infrequent correct Go	MNI	go/no-go	Table 1
				go/no-go	Table 2

Congdon et al., 2014	62	Correct Stop > Correct Go	MNI	stop signal task	Table 2
Coxon et al., 2016_1	20	Correct Stop > Correct Go	MNI	stop signal task	Table 2 (young)
Coxon et al., 2016_2	20	Correct Stop > Correct Go	MNI	stop signal task	Table 2 (old)
Czapla et al., 2017	21	Correct NoGo > Correct Go	MNI	go/no-go	SM Table 4
				go/no-go	SM Table 5
Dambacher et al., 2015	15	Correct NoGo > Go	TAL	go/no-go	Table 1
Fassbender et al., 2004	18	Correct Inhibitions > tonic activation	TAL	go/no-go	Table 2
Fauth-Bühler et al., 2012	18	successful stop > baseline	MNI	stop signal task	from authors
Fedota et al., 2015	16	Correct NoGo > Correct Go	MNI	go/no-go	Table 1
Fuentes-Claramonte et al., 2016	57	Correct NoGo > Correct frequent Go	MNI	go/no-go	Table 2
		Correct NoGo > Correct Infrequent Go		go/no-go	Table 2
Ganos et al., 2014	15	Correct Stop > Go	MNI	stop signal task	SM Table 2
Garavan et al., 1999	14	correct Inhibition > active baseline	TAL	go/no-go	Table 1
Garavan et al., 2002	14	correct Inhibition > active baseline	TAL	go/no-go	Table 1
Garavan et al., 2003	16	correct Inhibition > active baseline	TAL	go/no-go	Table 1
Geng et al., 2009	16	Correct NoGo > Correct Go	MNI	go/no-go	Table 3
Ghahremani et al., 2012	18	Correct Stop > Correct Go	MNI	stop signal task	Table 3

Harle et al., 2016	34	Correct Stop > Correct Go	TAL	stop signal task	SM Table 2
Hester et al., 2004a	15	successful inhibition vs. Baseline	TAL	go/no-go	Table 1
Hester et al., 2004b	15	Successful inhibition vs go	TAL	go/no-go	Table 3
Hough et al., 2016	22	Correct NoGo > Correct Go	MNI	go/no-go	SM Table
Hsu et al., 2017	20	Correct NoGo > Go	MNI	go/no-go	Table 2
Hughes et al., 2012	10	Correct stop > baseline	MNI	stop signal task	Table 4
Hughes et al., 2013	15	Correct stop > baseline	MNI	stop signal task	Table 3
Jahfari et al., 2011	20	Correct Stop > Go	MNI	stop signal task	Table 4
Jahfari et al., 2012	16	Correct Stop > Go	MNI	stop signal task	Table 5
Jahfari et al., 2015	23	Correct Stop > Go	MNI	stop signal task	Table 3
Kaladjian et al., 2007	21	correct No-Go > Correct Go	TAL	go/no-go	Table 3
Kaladjian et al., 2009a	20	correct No-Go > Correct Go	TAL	go/no-go	Table 3
Kaladjian et al., 2009b	10	correct No-Go > Correct Go	TAL	go/no-go	Table 3
		correct No-Go > Correct Go		go/no-go	Table 3
Kelly et al., 2004	15	Successful inhibition vs baseline	TAL	go/no-go	Table1
Kenner et al., 2010	24	Correct Stop > Correct Go	MNI	stop signal task	SM Table 3
Kiehl et al., 2000	14	correct Inhibition > active baseline	MNI	go/no-go	Table 1
Ko et al., 2014	23	Correct NoGo > Go	MNI	go/no-go	Table 2

Ko et al., 2016	32	Correct Stop > Correct Go	MNI	stop signal task	Table 1A
		Correct Stop > Correct Go		stop signal task	Table 1B
Köhler et al., 2018	33	Correct NoGo > Correct Go	MNI	go/no-go	SM Table 1
Lavallee et al., 2014	21	Correct Stop > Go	MNI	stop signal task	Table 2
Liddle et al. , 2001	16	correctNoGo > correctGo	MNI	go/no-go	Table 3
Lorenz et al., 2015	38	Corect Stop > Go	MNI	stop signal task	SM Table 1
Marco-Pallarés et al., 2008	10	correct Stop > correct Go	MNI	stop signal task	Table 1
Mazzola-Pomietto et al., 2009	16	correct No-Go > Correct Go	TAL	go/no-go	Table 3
Mohammadi et al., 2015	17	Correct Stop > Go	MNI	stop signal task	SM Table 2
Montojo et al., 2013	30	Correct Stop > Correct Go	MNI	stop signal task	Table 2
Nakata et al., 2008	15	Correct NoGo > Correct Go	TAL	go/no-go	Table 5
O'Connor et al., 2012	18	Correct NoGo > active baseline	MNI	go/no-go	Table 1
Rae et al., 2014	17	Stop specified correct > go specified correct	MNI	stop signal task	SM Table 2
		Stop select correct > go select correct		stop signal task	SM Table 2
Rodriguez-Pujadas et al., 2014	33	Correct Stop > Correct Go	MNI*	stop signal task	Table 2
Rothmayr et al., 2011	12	correct No-Go > Correct Go	MNI	go/no-go	Table 2
Rubia et al., 2006_1	21	successful NoGo > successful Go	TAL	go/no-go	Table 2

Rubia et al., 2006_2	25	successful no-go > successful go	TAL	go/no-go	Table 2
Schel et al., 2014	24	Correct Stop > Correct Go	MNI	stop signal task	Table 4
Sebastian et al., 2012	24	Correct NoGo > Correct Go	MNI	go/no-go	Table 5
		Correct Stop > Correct Go		stop signal task	Table 5
Sebastian et al., 2013a	48	Correct NoGo > active baseline including go	MNI	go/no-go	SM Table 1
		Correct Stop > Correct Go		stop signal task	SM Table 1
Sebastian et al., 2013b_1	24	Correct NoGo > active baseline including go	MNI	go/no-go	Table 3
		Correct Stop versus correct Go		stop signal task	Table 3
Sebastian et al., 2013b_2	21	correct NoGo > correct congruent go	MNI	go/no-go_hybrid response interference	Table 2
		Correct Stop versus correct congruent Go		stop signal task_hybrid response interference	Table 2
Sebastian et al., 2017	80	Correct Stop > Correct Go	MNI	stop signal task	from authors
Sharp et al., 2010	26	Correct Stop > Correct Go	MNI	stop signal task	SM Table 1
Steele et al. 2014	102	Correct NoGo > Correct Go	MNI	go/no-go	Table 2
Swann et al. 2012	16	Correct Stop > Correct Go	MNI	stop signal task	SM Table 2
Tabu et al., 2011	13	Correct Stop > Correct Go	MNI	stop signal task	text, page 280

Tabu et al., 2012	13	Correct Stop > Correct Go	MNI	stop signal task	SM Table 1 (hand task)
		Correct Stop > Correct Go		stop signal task	SM Table 1 (foot task)
Van der Meer et al., 2013	19	Correct Stop > Correct Go	MNI	stop signal task	SM Table 3A
Van Eijk et al., 2015_1	18	Correct NoGo > Correct Go	MNI	go/no-go	Table 4
		Correct Stop > Correct Go		stop signal task	Table 4
van Eijk et al., 2015_2	25	Correct NoGo > Correct congruent Go	MNI	go/no-go_hybrid response interference	Table 5
		Correct stop > correct congruent go		stop signal task_hybrid response interference	Table 5
Walther et al., 2010	17	correct No-Go > Correct Go	MNI	go/no-go	Table 1
Wilbertz et al., 2014	49	Correct Stop > Go	MNI	stop signal task	SM Table 2
Xu et al., 2015	18	Correct Stop > Go	MNI	stop signal task	Table 2
Xu et al., 2017	21	Correct Stop > Correct Go	MNI*	stop signal task	SM Table 1
Xue et al., 2008	15	Correct Stop > Correct Go	MNI	stop signal task	SM Table 1 (manual)
		Correct Stop > Correct Go		stop signal task	SM Table 1 (letter naming)

Zandbelt and Vink, 2010	24	Correct Stop > Go	MNI	stop signal task	SM Table 3B
Zandbelt et al. 2011	22	Correct Stop > Go	MNI	stop signal task	SM Table 11
Zheng et al., 2008	18	Correct Stop > Go	TAL	go/no-go	Table 1
Lock et al., 2011	13	correct No-Go > Correct Go	TAL	go/no-go	Table 2
Nosarti et al., 2006	14	(Correct no-go – go) – (correct 'odd' (aka go acting as a control for low freq of nogo) – go)	TAL	go/no-go	In text pg. 268
Durston et al., 2006	11	Successful No-go > Go	MNI	go/no-go	Table 2
He et al., 2014	30	Correct No-go > Go	MNI	go/no-go	Table 3
Schulz et al., 2004	9	Correct nogo > Correct Go	TAL	go/no-go	Table 2
Lee et al., 2018	34	Correct No-go > Go	MNI	go/no-go	Table 3
Heitzeg et al., 2010	20	Correct No-go > Go	MNI	go/no-go	Table 2
Mulder et al., 2008	12	Correct No-go > Go	MNI	go/no-go	Table 2
White et al., 2014	1133	stop success > baseline	MNI	stop signal task	SM Table 3
Lim et al., 2015	27	Stop Success > Go	MNI	stop signal task	SM Table
Bennett et al., 2009	11	Correct No-go – Correct go	TAL	go/no-go	Table 2
Cascio et al., 2015	37	Correct No-Go > Correct Go	MNI	go/no-go	Table 3
Rubia et al., 2013	66	Stop Success > Go	TAL	stop signal task	Table 2

Failure of Inhibition Database					
Author	N	contrast	space	paradigm	source of data
Boecker et al., 2011	15	failed stop - Go	MNI	stop signal task	SM Table 2
Boehler et al., 2010	15	unsuccessful stop > go	MNI	stop signal task	SM Table 4 and 3
Chen et al., 2015	25	failed nogo - go	MNI	go/no-go	Table 2
Chevrier et al., 2007	14	unsuccessful stop vs baseline	TAL	stop signal task	Table 1
Dambacher et al., 2015	15	False alarm > go	TAL	go/no-go	Table 1
Garavan et al., 2002	14	error vs. Baseline	TAL	go/no-go	Table 1
Garavan et al., 2003	16	error vs. Baseline	TAL	go/no-go	Table 1
Harle, 2016	34	stop error > go	TAL	stop signal task	SM Table 3
Hester et al., 2004	15	error vs. Baseline	TAL	go/no-go	Table 2
Hester et al., 2005	13	error vs correct go	TAL	go/no-go	Table 1
Hough et al., 2015	22	unsuccessful inhibition vs baseline	MNI	go/no-go	SM Table 5
Hsu et al., 2017	20	unsuccessful inhibition vs go	MNI	go/no-go	Table 3
Hughes et al., 2012	10	stop failure vs baseline	MNI	stop signal task	Table 4
Hughes et al., 2013	15	stop failure vs baseline	MNI	stop signal task	Table 3

Jahfari, 2011	20	failed stop > go	MNI	stop signal task	Table 4
Jahfari, 2012	16	failed stop > go	MNI	stop signal task	Table 5
Kiehl et al., 2000	14	error vs baseline (error of comission)	MNI	go/no-go	Table 1
Ko et al. 2014	23	unsuccessful inhibition - go	MNI	go/no-go	Table 3
Mohammadi et al., 2015	17	unsuccessful stop > go	MNI	stop signal task	SM Table 3
Rubia et al., 2003	20	unsuccessful stop vs go	TAL	stop signal task	Table 1
Sharp et al., 2010	26	incorrect stop vs correct go	MNI	stop signal task	SM Table 4
Steele et al., 2014	102	error vs correct go	MNI	go/no-go	Table 1 and 3
Xu et al., 2017	21	failed stop > go	TAL	stop signal task	SM Table 2
White et al., 2014	1133	stop failure > baseline	MNI	stop signal task	SM Table 3
Lim et al., 2015	27	stop failure > go	MNI	stop signal task	SM Table
Halari et al., 2009	21	stop failure – go	TAL	stop signal task	Table 3

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