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Impulsive Responses to Positive and Negative Emotions: Parallel Neurocognitive Correlates and their Implications

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Abstract

Theory about the conceptual basis of psychiatric disorders has long emphasized negative emotionality. More recent ideas emphasize roles for positive emotionality and impulsivity as well. This article examines impulsive responses to positive and negative emotions, which have been labelled as urgency. Urgency is conceptually and empirically distinct from other forms of impulsivity. A large body of work indicates that Urgency is more robustly related to psychopathology than are other forms of impulsivity. Researchers have considered four neurocognitive models of urgency: excessive emotion generation, poor emotion regulation, risky decision-making, and poor cognitive control. Little evidence supports emotion generation or risky decision-making as the core issues driving urgency. Rather, urgency appears related to dysfunction in key hubs implicated in the integration of cognitive control and emotion regulation (e.g., the orbitofrontal cortex and anterior insula), expressed as response inhibition deficits that emerge most robustly in high arousal contexts. These neurocognitive processes appear remarkably parallel for positive and negative urgency. We provide methodological suggestions and theoretical hypotheses to guide future research.

Keywords

impulsivity; emotion; urgency; psychopathology; cognition; imaging

This article focuses on trait-like tendencies to respond impulsively to emotion states, referred to as emotion-related impulsivity. This literature first centered on impulsive reactions to negative emotions (1) but later broadened to incorporate impulsive reactions to positive emotions (2–3). Work in this domain rapidly demonstrated the power of integrating the study of emotion with that of impulse. The focus on this nexus has emerged in parallel with a growing body of work in neuroscience on the interface of emotion and cognitive

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control (4–6). Collectively, this work is beginning to transform psychopathology research to focus on integrating these domains (7–8).

Even though this section of the issue focuses on positive emotions, we consider impulsive responses to both positive and negative emotions, because the body of work pertaining to negative emotion is much larger, and the parallel findings provide a foundation for understanding responses to positive emotion. Indeed, one of our conclusions is that the valence of the emotion is often less important than the impulsive responsivity per se.

Definitions and Measures

Close study of emotion-related impulsivity began with the development of the Urgency scale in 2001, now referred to as Negative Urgency (NU). NU items cover impulsive responses to negative emotion (e.g., “When I am upset I often act without thinking.”) NU is one subscale of the UPPS, which is an abbreviation for Urgency, (low) Perseverance, (low) Premeditation, and Sensation-Seeking (1). NU is not merely a tendency toward negative emotionality, in that it shows divergent validity with such constructs as distress tolerance and neuroticism (8–9). The idea that some people experience impulsivity triggered by emotions rapidly became influential.

NU references mostly negative emotions. The Positive Urgency (PU) measure, developed a few years later, references impulsiveness during states of positive emotions (3). Whether measured using self-report or interview, NU and PU are highly correlated (*r*’s = .46 to .49, *N*’s = 183, 1886) and form a higher-order factor (10, replicated in 11). Hence, problems with impulsive responding to emotion appear not to be specific to the valence of emotion. Rather people who have problematic responsivity to negative emotions tend to report problematic responses to positive emotions.

Urgency is consistently found to be distinct from other forms of impulsivity that do not reference emotion, including self-reported lack of perseverance or premeditation, r’s < .35 [2, 10–11], and laboratory tasks involving impulsivity measured outside of emotion contexts (12–13). This suggests that problems of constraint over emotion are separable.

Links of Emotion-Related Impulsivity with Psychopathology

Across hundreds of studies, NU relates robustly to a broad range of psychopathologies (14). Beyond effects for externalizing disorders (*r*=.34, or Hedges g =.74), NU also correlates with depressive symptoms and diagnoses (14–19), (*r*=.45, Hedges g = 1.00[14]), and these effects withstand control for comorbid externalizing syndromes (20). NU also is elevated among persons with schizophrenia (21). Beyond syndromes, NU is related to interview and self-report measures of aggression (20–22), suicidal ideation and attempts (14,23–25), and self-harm (24–28).

The development of the PU scale was inspired by clinical observations that many impulsive symptoms involve positive affectivity, such as drinking and gambling (3). Indeed, PU appears significantly more elevated than NU for persons with a history of mania (29). Nonetheless, effect sizes for PU are comparable to those of NU across a range of

internalizing and externalizing disorders (14,17,20, 30–31). Effect sizes for PU even parallel those of NU for lifetime major depressive disorder (32–33). High PU is also related to suicidality and self-harm (14). The counterintuitive findings linking depression and suicidality to impulsive reactions to positive emotions point to the importance of poor constraint over emotion as the core concern.

These findings do not appear to be artifactual (8). Despite concerns that the effects could reflect some form of bias in self-evaluation, parent-, interviewer-, and self-ratings of Urgency each show robust effects on psychopathology (2,34–35,14). Contrary to the idea that ratings simply reflect memories of symptoms, NU predicts onset and worse course of alcohol and smoking problems (36–37), as well as risk of suicide attempt (38) and self-harm in longitudinal studies (39). PU also predicts a worse course of illegal drug use, risky sexual behavior, and alcohol use (40–43). Also supporting scale validity, laboratory and experience sampling studies confirm that the scales predict emotion-related changes in behavior. That is, NU predicts symptom worsening (e.g., dietary lapses, drinking) during negative emotion states (44–46), and PU predicts more alcohol use during positive emotion states (47).

Emotion-related impulsivity appears more predictive than studying either emotion or impulsivity in isolation. Effect sizes for NU and PU are much larger than those for other forms of impulsivity measured on the UPPS [mean r<.15 for Premeditation, Perseverence, and Sensation-Seeking] (14). The observed effect sizes for NU with internalizing and externalizing disorders (Hedges g = .75 to 1.00) are also substantively larger than those observed for behavioral impulsivity measures, such as the go/no-go task (generally < .50) (48) or delay discounting tasks (r with addictive behavior = .14) (49). Effects of NU and PU consistently emerge after controlling for other forms of impulsivity or emotionality (3,8,14,44).

The robust and transdiagnostic mental health effects have led to the argument that emotion-related impulsivity may help explain the p-factor, a general vulnerability to psychopathology (50). Other risk factors, such as approach motivation and threat sensitivity, may shape which specific symptoms emerge in the context of this failure of constraint (see 51).

Neurocognitive Correlates of Urgency

In the search for basic mechanisms, researchers have considered four domains: emotion generation, emotion regulation, risky decision-making, and response inhibition. We consider behavioral and neuroimaging findings jointly within domain (52). We highlight the few studies probing how emotion and cognition interact, using valenced stimuli or mood inductions. We also integrate relevant findings from task-free methodologies (e.g. positron emission topography (PET), structural MRI, and resting-state functional connectivity (RSFC).

Figure 1 provides a schematic of key neural nodes involved in the generation and control of emotion. Although each region is clearly involved in many task contexts, the figure highlights key functions implicated in the interface of emotion and impulsivity. We attend closely to findings regarding the orbitofrontal cortex (OFC) and anterior insula (AI), as they

are integrative hubs activated by emotion generation, emotion regulation, and cognitive control tasks (5,52–54).

The locus coeruleus (LC) exerts neuromodulatory effects across this network. The LC is the dominant source of norepinephrine (NE) in the brain, and dynamic fluctuations in NE correlate with subjective arousal (55), which is present with intense positive and negative emotions (56).

Given the history of inflated effect sizes for neuroimaging correlates of individual difference variables (57–58), we prioritize replicated findings and theoretically-guided patterns, not effect sizes. Tables 1–4 provide the broader set of imaging and urgency studies.

Emotion Generation.

Persons with NU and PU do not show elevated subjective, facial behavior, psychophysiological, or cortisol responses to standardized emotion-relevant stimuli or mood induction procedures (12,44,46–47,59), stressors such as speech tasks (60,61), or failed trials on cognitive laboratory tasks (62).

As shown in Table 1, several fMRI studies have considered passive observations of valenced stimuli. Both of the studies of PU were limited by small samples and by use of stimuli that evoked more negative than positive emotion (63–64). Unsurprisingly, PU was not related significantly to neural activation profiles in these studies. Consistent with the null effects for behavioral reactivity, NU related to increased amygdala activity in only one of three fMRI studies to probe passive observation of valenced stimuli (63). NU, however, did correlate with higher GABA-A binding availability in the amygdala (65), which has been related to heightened behavioral reactivity to emotion and acute stress (66). In contrast to the mostly null findings regarding emotion generation or amygdala reactivity, all three passive observation studies found that NU related to increased OFC activation to valenced (vs. neutral) stimuli (63,64,67).

Potential monetary reward may evoke more positive emotion than positive pictures do, of import for PU. In one large study (N=100), high NU and PU both related to increased ventral striatum (VS) and ventrolateral prefrontal cortex (VLPFC) activation during uncertain monetary reward anticipation (68). Findings with different imaging modalities also implicate the VS. NU and PU related to D2/D3 receptor binding potential in VS (69). NU (PU not measured) also correlated with GABA-A receptor availability and gray matter volume in the VS (65,70).

Taken together, Urgency appears linked to OFC responses to valenced stimuli and VS and VLPFC responses to reward expectancy. OFC and VLPFC are implicated in many task contexts, including representation, monitoring, and updating of stimulus-outcome relationships in the context of goal pursuit (53,71,75). Aberrant function in these regions may reflect greater subjective evaluation of cues of potential threat and reward, which could influence updating and learning processes.

Emotion Regulation.

Rather than emotion reactivity, high NU relates to poor emotion regulation, including less reappraisal and perspective-taking, and more rumination (76). Two fMRI studies have used a cognitive reappraisal task (77). In one, NU correlated with higher amygdala activation when directed to reappraise, suggesting less down-regulation of emotion (78). In the other study, profiles differed by group, but in aggregate, NU correlated with increased dorsolateral prefrontal cortex (DLPFC) activation during the sustain condition (79), suggesting ineffective recruitment of cognitive control networks when instructed to modulate emotion. One report linked NU to lower GABA levels in DLPFC (80), which could contribute to these DLPFC inefficiencies during emotion regulation. No data is available concerning PU and neural correlates of emotion regulation.

Risky Decision-Making.

More than 100 behavioral studies have considered risky decision-making tasks such as the Balloon Analog Risk Task (81), the Iowa Gambling Task (82), and Delay of Gratification tasks. NU and PU are not significantly related to such measures, r's ≤.13, in meta-analyses (12–13), or recent findings (83–88 but see 89).

Evidence is mixed about whether higher arousal conditions trigger risky decision-making for those with high Urgency. In one meta-analysis, NU correlated with less ability to delay gratification for actual payments, r=.24, but not for hypothetical rewards, r=.03 (13 but see 90–91 for nonreplications). In one study, PU related to greater risk taking after (though not before) a mood induction (47 but see 61,88 for nonreplications).

In the fMRI studies of risky decision making, higher anterior insula (AI) activation, consistent with greater prioritization of interoceptive cues (54), was correlated with NU (92–94) and PU (92). Consistent with the importance of emotional arousal in Urgency, higher AI activation was observed in decisions involving higher arousal or risk, such as sex vs. conversation (92) and uncertain vs. certain decisions (93).

In sum, although Urgency does not consistently relate to performance on the laboratory measures being used to assess risky decision-making. fMRI findings link Urgency to elevated AI during risky decision-making in higher arousal/risk conditions. Given evidence that AI lesions relate to diminished risky decision-making (95), dynamic fluctuations in AI might help explain the rash behavior observed with high PU and NU.

Response Inhibition.

Consistent with theory (96–98), findings of meta-analyses indicate that NU and PU relate to poorer performance on tasks involving response inhibition, including go/no-go, go-stop, and antisaccade. These effects appear specific, and are not observed with other facets of executive function, including attention, planning, or time estimation tasks (12, 13, 88). One meta-analysis showed correlations with shifting tasks (13), consistent with models of response inhibition and shifting as closely related facets of executive function (99).

Focusing on response inhibition, the correlations with Urgency are not universally observed. These effects are generally small among student and community samples, mean r=.11 for

NU, and r=.14 for PU, but more robust in the 4 available clinical samples, r=.34 for NU (no PU effects available; 88). Response inhibition deficits might be present only for those with severe urgency. In two studies with college students, we found the expected curvilinear pattern, in which little link with response inhibition was observed at the low end of Urgency, but response inhibition deficits emerged at higher levels (33,88). These effects were observed for PU and a composite of PU and NU (33,88).

Some have suggested the importance of distinguishing between early- and late-stage response inhibition, which differentially involve withholding initiation of a prepotent response versus stopping a response already underway (27–28). In one meta-analysis, NU related to deficits on late-stage response inhibition (stop-signal) task but not early-stage response inhibition (13). Nonetheless, when both tasks were administered in the same sample, poorer performance on both the early and late response inhibition measures correlated with PU and NU (28).

Given that emotion triggers symptoms for those with highUurgency, we hypothesized that small increases in arousal might trigger declines in response inhibition for those with high urgency. We used pupil dilation, which is innervated by the LC and correlates closely with subjective arousal (55), to measure trial-by-trial arousal during a response inhibition (antisaccade) task. Persons with higher emotion-related impulsivity showed decay in response inhibition after even minor increases in arousal (pupil); those with low emotion-related impulsivity did not (62). Hence emotional arousal (tied to positive and negative emotions) may interfere with response inhibition for those with high emotion-related impulsivity. Declines in cognitive control with extreme elevations of NE are normative (100–104); those with higher urgency may show more fragility in response inhibition with these dynamic fluctuations in NE.

Consistent with the role of emotional arousal, fMRI findings have linked NU and PU to activation of regions involved in response inhibition only when valenced stimuli were incorporated into the task, and not in response to neutral stimuli (105–108). In the two fMRI studies of early-stage response inhibition that included valenced stimuli, NU and PU related to increased activity in cognitive control regions, including AI, dorsal caudate, and VLPFC (106), as well as supplementary eye field during an antisaccade task (107). Activation of these regions was correlated to NU in the presence of negative stimuli (106), and to PU in the presence of positive stimuli (107).

In contrast, in an fMRI study of late response inhibition, NU related to *less* activity in VLPFC (108). Although more studies are needed to test the replicability of this divergence, these results suggest a compensatory response that enables early-stage response inhibition, and diminished activation to late-stage response inhibition task demands in people with high urgency.

Intriguingly, VS activation after a rewarded “Stop” trial in this study correlated with better task performance for people with lower, but not higher Urgency (108). This highlights reinforcement signaling in the VS, alongside NE-based arousal, as a dynamic circuit that

integrates with response inhibition networks and may be disrupted in those with high Urgency.

Of clinical relevance, atypical activations of canonical response inhibition regions appear to statistically mediate relationships of NU and PU with real-world outcomes, such as alcohol consumption and risky sexual behavior (105–106). In addition, in structural MRI studies (Table 3), NU was tied to decreased grey matter volume or cortical thickness in several cognitive control and integration hub regions shown in Figure 1, complementing the pattern of results from fMRI (21,109–1110).

Functional Connectivity.

RSFC, a technique to index synchrony between brain regions in the absence of task performance, has been interpreted as the intrinsic and modal functional state of neural networks (111). Because motion may produce systematic Type 1 errors in RSFC (112–114), we only include studies that took recommended steps to reduce these artifacts (115). As shown in Table 2, RSFC findings published before newer standards for cluster-defining primary thresholds in multiple comparisons corrections (116,117) had less conservative analyses (21).

Multiple studies indicate that NU and PU are related to weaker RSFC involving cognitive control regions, but the specific profiles vary across studies. NU and PU have been related to weaker RSFC of cognitive control regions with OFC (21,118) and with default-mode network (DMN) (118,119). Findings regarding other RSFC correlates of Urgency have been even more mixed, including those involving emotion generation regions (118,120,121). Perhaps different network disturbances can contribute to Urgency in an equifinal way. It is also possible that RSFC is too unconstrained, and standard correlational and “seed-to-voxel” approaches may capture only a small slice of the complex functional connectivity profile in any one study.

Treatment Development

Multiple authors have emphasized the need to develop treatments to address Urgency (122,123). Urgency, however, may be a difficult treatment target. For example, high NU predicted poorer outcome across six studies of cognitive therapy for alcohol use disorder (124). Similarly, NU predicted less ability to implement coping to improve diet after a single-session intervention (125). These findings suggest that interventions may need to be tailored to address NU. We briefly consider treatment development work targeting emotion regulation, cognitive control, and their combination.

Several findings show promise for emotion regulation approaches. In secondary analyses of interventions focused on improving emotion regulation, NU predicted positive outcomes (126). One study tested a 9-week dialectical behavior therapy group to enhance emotion regulation in a school setting as a preventative approach to tendencies to engage in rash behavior in response to emotion. Pre-post scores on a measure of risky behavior during positive and negative emotion states declined, particularly for students with higher NU scores at baseline (127). In another study, college students received a single session of

emotion management training or impulsivity reduction training. Emotion management training was more powerful than impulsivity training in reducing NU scores (128).

Cognitive training also appears promising. In one study, adults with high emotion-related impulsivity were assigned to either immediate cognitive training (response inhibition on the Go/No-go task and working memory on the PASAT) or a waitlist. Emotion-related impulsivity scores declined after 6 sessions of training, but not in the waitlist condition (129).

One intervention for emotion-related impulsivity and aggression targeted emotion and cognitive control. To address emotionality, participants were taught to identify anger and related triggers, and then learned self-calming skills such as relaxation. To remediate deficits in cognitive control during high arousal, participants were taught to pre-plan how they would cope with anger using implementation intentions, a well-validated approach to diminishing impulsive actions (130). Aggression and self-harm declined significantly in the active intervention but not the waitlist condition (131).

Overall, these early treatment development findings support the idea that addressing problems of executive control during periods of high arousal can help alleviate emotion-related impulsivity and its consequences. This fits with the mechanisms being identified in the basic research on emotion-related impulsivity. Given the transdiagnostic effects of emotion-related impulsivity, one goal will be to test whether targeting emotion-related impulsivity can reduce symptoms transdiagnostically.

Conclusion

The Urgency scales are well-validated as related to a broad range of psychopathologies and behavioral problems, with effect sizes larger than those for measures of emotionality or impulsivity alone. Despite a long history of focus on negative emotionality, the effects for PU are as large as those for NU.

Work on the neurocognitive correlates of Urgency is accelerating, and mostly falls within four domains: emotion generation, emotion regulation, risky decision-making and response inhibition. Some models have achieved little support. Urgency does not appear related to emotion generation, across behavioral, psychophysiological or neural indices. Behavioral performance on the tasks used to capture risky decision-making does not appear to be consistent correlates of Urgency, although elevated anterior insula is observed during such tasks, particularly when higher risk decisions are being made.

Across behavior and imaging methods, evidence supports difficulties in response inhibition (but not facets of executive function such as attention problems) for both PU and NU. Given that reappraisal rests on similar cognitive control circuitry to response inhibition (132), it is unsurprising that Urgency also relates to less use of this emotion regulation strategy and atypical neural responses when instructed to regulate emotion. Smaller effect sizes are observed for healthy (as compared to clinical) samples in behavioral studies of response inhibition (88). Consistent with basic findings, early intervention work suggests the merit of targeting response inhibition and emotion regulation deficits.

In addition to canonical cognitive control regions, key regions at the interface of cognition × emotion, OFC and AI, are tied to Urgency across task contexts. OFC and AI lesions have both been tied to impulsive action (71,95,133). Structural deficits in OFC were observed in one severe clinical sample. RSFC findings, albeit more mixed and more evident in severe clinical samples, also highlight the importance of weakened communication between OFC and key regions involved in cognitive control. Together, findings suggest these regions play a central role in the phenotype.

The stronger clinical versus community profile fits models of primary (generalized and structural) versus secondary (triggered and dynamic) cognitive control deficits (134). There is no clear evidence of reproducible structural correlates in community samples. Rather it appears that constraint circuits are perturbed by high emotion/arousal. That is, some perturbations may magnify deficits among those with high Urgency in the community samples—response inhibition deficits appear to decay with valenced stimuli, mood inductions, and increases in norepinephrine levels (pupil), and fail to show normative improvement with higher ventral striatum activation. AI, which has been related to response inhibition (135) appears to be more powerfully related to Urgency in high risk contexts, possibly indicating increased prioritization of interoceptive emotion cues. More research is needed to understand how these dynamic fluctuations interact with abnormal function of cognitive control regions and OFC.

Despite the importance of Urgency in mental health outcomes, behavioral and imaging research remains limited. Many studies rely on behavioral tasks with poor reliability (136–137). Latent models are needed to control for task unreliability and overlap in functions probed by the behavioral measures, as a way to evaluate specificity of executive function (99). Neuroimaging studies of PU are sparse—in each neurocognitive domain, only one PU study used adequate positive stimuli. Many fMRI studies rely on small sample sizes. Many RSFC findings have not replicated, perhaps because the standard correlational statistics used do not capture dynamic processes of urgency. Community samples may not include many highly impulsive individuals; future community-based studies will need to adequately sample those with higher Urgency, and to consider potential curvilinear effects in which neurocognitive deficits may only be observed as Urgency is more severe.

Taking into account the strengths and limitations, the profile of findings suggests several hypotheses for future research:

1. In community samples, behavioral and neural correlates of NU and PU will be more robustly observed when the system is perturbed, through techniques such as experimental manipulation of emotional arousal.
2. The dynamic nature of the deficits calls for imaging techniques designed to capture temporally instable processes, such as dynamic functional connectivity.
3. Findings regarding the OFC and AI suggest the importance of using tasks that effectively probe the function of these regions, such as updating and shifting stimulus-outcome operations during goal pursuit, and responses to interoceptive cues of arousal.

Although we hope these ideas will help guide future research, it may be that more than one process will culminate, in an equifinal manner, in Urgency. Indeed, promising but currently underexplored findings of reinforcement signaling in VS, GABA binding in amygdala, and RSFC of DMN may prove key in future models. Given the complex nature of the neural circuits underpinning the deficits, best-suited techniques will be those that assess the architecture of multiple brain circuits, such as graph theory and structural equation modeling (138,139).

In sum, poor control over positive (as well as negative) emotions appears to be a major risk factor for a very broad range of psychopathologies. This suggests that positive affectivity is as important for understanding psychopathology as is negative affectivity. PU correlates highly with NU, and the behavioral and neural correlates of PU and NU are largely parallel. Given the overlap, those studying problematic outcomes of emotionality would do well to consider the idea that the real culprit may be the loss of control over high emotion states generally, rather than positive or negative emotion specifically. The PU and NU scales may provide a quick tool to capture problems of cognitive control and behavioral problems in contexts of high emotional arousal, and appear related to disturbances in OFC, AI, and cognitive control circuitry in such contexts. As such, these brief, easily administered scales may provide a method to bridge these neural indicators to transdiagnostic psychopathology outcomes in future research.

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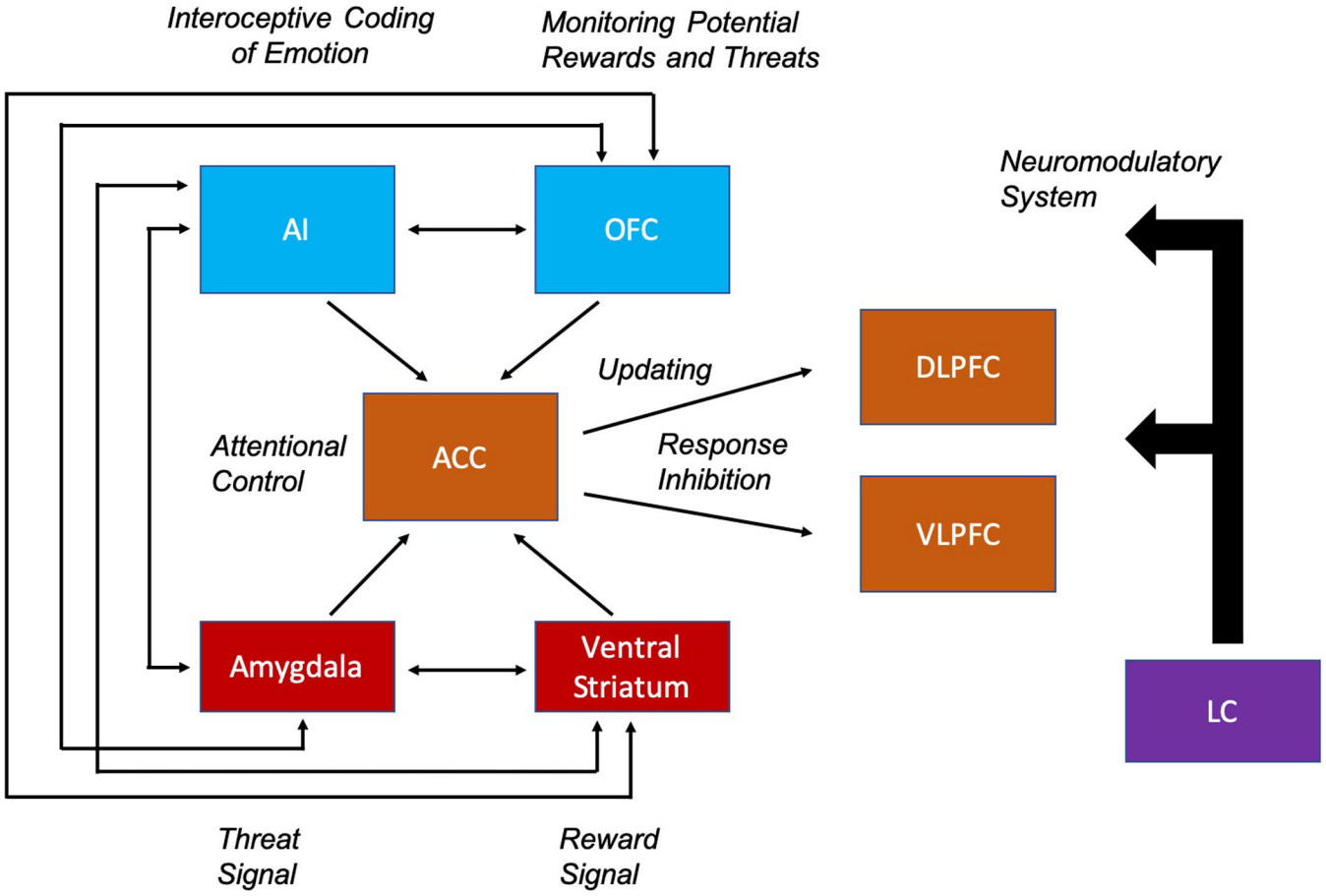

Figure 1.

Key neural regions involved in the integration of emotion and cognition, adapted from Pessoa, 2009 (142). This includes regions that contribute to emotion generation (143,144) (Red) and cognitive control regions (Orange) involved in attentional control, response inhibition, and the updating of information (145–147). Regions that integrate affective and motivational signals (Blue) are hubs in both the emotion generation and control processes (53,54,71). Neuromodulators (Purple), such as NE projecting from the LC may affect the rest of the network via state-dependent shifts in arousal (55). Arrows represent the hypothesized functional interactions among regions of interest.

Table 1.

Summary of Task-Based fMRI Studies

Study	Task	Emotion Stimuli	Sample Size by Group	Findings with High Negative Urgency	Findings with High Positive Urgency	Other Findings
Emotion Generation						
Cyders et al., 2014 (64)	Passive Smelling	Alcohol/Juice/Sham (olfactory)	HC: 27	Alcohol > Sham: ↑OFC	Null	
Cyders et al., 2015 (63)	Passive Viewing	IAPS (Positive, Negative, Neutral)	HC: 27[a]	Negative > Neutral: ↑lateral OFC, ↑ amygdala	Null	NU mediated relationship between amygdala & OFC and risk-taking
Eiler et al., 2014 (67)	Passive Smelling	Food/Non-food (olfactory)	Normal Weight: 25 Obese: 25	**Normal Weight**: Null **Obese** (Food > No Food): ↑OFC	*N/A*	
Chase et al., 2017(69)	Card Guessing	Expectancy Cue	Psychological Distress: 48 HC: 52	**Whole Sample** (Uncertain Reward Anticipation): ↑VLPFC, ↑ventral striatum	Whole Sample (Uncertain Reward Anticipation): ↑VLPFC, ↑ventral striatum	
Emotion Regulation						
Albein-Urios et al., 2012 (79)	Cognitive Reappraisal (83)	IAPS (Negative, Neutral)	HC: 18 CD: 17	**Whole sample** (Maintain > Observe): ↑ DLPFC **HC** (Reappraise> Maintain): ↓VLPFC ⇔amygdala **CD** (Maintain > Observe): ↑DLPFC ⇔ AI/OFC	*N/A*	
Albein-Urios et al., 2013 (78)	Cognitive Reappraisal (83)	IAPS (Negative, Neutral)	HC: 21 CD: 17 CD+PD: 18	**HC**: Null **CD**: Null **CD + PD** (Reappraise > Maintain): ↑amygdala	*N/A*	
Risky Decision-Making						
Smith et al., 2018 (92)	Virtual Dating	Avatars of virtual dates	HC: 107	Sexual > Conversational Decision: ↑AI	Sexual Decision > Conversation Decision: ↑ AI	
Xiao et al., 2013 (93)	Iowa Gambling Task	Win/Loss Feedback	Never Drink: 14 Binge Drink: 14	**Whole sample** (IGT > Control task): ↑ AI, ↓OFC	*N/A*	
Xue et al., 2010 (94)	Modified Cups Task	Win/Loss Feedback	HC: 14	Trial after Safe Bet: ↑ AI	*N/A*	↑AI activation on current trial predicted risky decision on the subsequent trial
Response Inhibition						

Study	Task	Emotion Stimuli	Sample Size by Group	Findings with High Negative Urgency	Findings with High Positive Urgency	Other Findings
Barkley-Levenson et al., 2018 (105)	Color-Word Stroop	None	No History of Risky Sex: 33 History Risky Sex: 72	Null	Null	ACC[b], DLPFC[b], frontal pole[b] & AI activation mediated relationship between NU and risk-taking
Chester et al., 2016 (106)	Go/NoGo – IAPS images displayed behind targets	IAPS (Positive, Negative, Neutral)	High Urgency: 40 Low Urgency: 38	NoGo > Go on Negative Trials: ↑AI, dorsal striatum, VLPFC NoGo > Go on Neutral and Positive Trials: Null	*N/A*	AI mediated relationship between NU and alcohol consumption
Tervo-Clemmens et al., 2017 (107)	Antisaccade	Reward Cue (50% of trials)	HC: 116	Null	Rewarded Antisaccade: ↑SEF Unrewarded Antisaccade: Null	
Wilbertz et al., 2014 (108)	Stop Signal Task	Reward Stop Signal (50% of Stop trials)	High Impulsivity: 24 Low Impulsivity: 25	Rewarded Stop > Go: ↓VLPFC Unrewarded Stop > Go: ↓VLPFC	*N/A*	↑VS after successful reward trial predicted increased performance for low and medium, but not high Urgency participants.

ACC, anterior cingulate cortex; AI, anterior insula; CD, cocaine dependent; DLPFC, dorsolateral prefrontal cortex; HC, healthy control; IAPS, International Affective Picture System; IGT, Iowa Gambling Task; NU, negative urgency; OFC, orbitofrontal cortex; PD, personality disorder (cluster B); SEF, supplementary eye field; VLPFC, ventrolateral prefrontal cortex; VS, ventral striatum.

[a] Same sample used in Cyders et al., 2014 and Cyders et al., 2015

[b] These regions also mediated the relationship found between Positive Urgency and risk-taking.

↑ Represents a positive correlation with Urgency

↓ Represents a negative correlation with Urgency

⇔ Represents bidirectional functional connectivity

N/A – Study did not include Positive Urgency in analyses

Table 2.

Summary of Resting-State Functional Connectivity Studies

Study	Sample Size by Group	Scan Time	Motion Correction	Correction for Multiple Comparisons	Findings with High Negative Urgency	Findings with High Positive Urgency
Hoptman et al., 2014 (21)	HC: 31 SCZ: 33	6 min	Motion correction, denoising, mean FD included as covariate	Primary threshold: z=2.3 Cluster-level correction: p=0.05 using GRF	**SCZ**: ↓ lateral OFC → MFG (left) ↓ medial OFC → SFG ↓ medial OFC → rACC ↓ rACC → SFG ↑ lateral OFC → IFG/MFG (right) ↑ lateral OFC → PCC ↑ medial OFC → precuneus ↑ medial OFC → cuneus ↑ frontal pole → SPL **HC**: ↓ lateral OFC → insula ↓ frontal pole → insula ↓ frontal pole → postcentral gyrus ↑ frontal pole → putamen ↑ lateral OFC → STG	**SCZ**: ↓ rACC → frontal pole ↑ frontal pole → occipital gyrus **HC**: ↓ lateral OFC → insula ↑ rACC → IPL ↑ rACC→ parahippocampal gyrus ↑ frontal pole → putamen
Golchert et al., 2017 (119)	HC: 112 (MPI-S) HC: 92 (NKI-RS)	MPI-S: 15.5 min NKI-RS: 10 min	Motion correction, denoising, mean FD included as covariate	Primary threshold: z=3.1 Cluster-level correction: pFWE < .05	Null	↓ subgenual ACC → retrosplenial cortex
Zhao et al., 2017 (120) *	HC: 85	10.83 min	Motion correction, scrubbing FD > 0.3mm	Primary threshold: p<0.001 Cluster-level correction: pFWE < 0.05	↓ PCC/precuneus → VTA, thalamus, LN, medial GP, putamen, SN and caudate ↓ PCC/precuneus → precentral gyrus	
Zhu et al., 2017 (118)	HC: 26 AD: 25	5 min	Motion correction, scrubbing FD > 0.5mm or > 5% change global signal intensity	Primary threshold: pFWE < 0.05; Results correction: p < 0.05 bonferroni	**AD**: ↓ amygdala ⇔ striatum ↓ OFC ⇔ ECN ↓ OFC ⇔ DMN **HC**: Null	**AD**: Null **HC**: ↓ anterior DMN ⇔ anterior DMN
Um et al., 2019 (121)	HC: 62 TU: 34	5 min	Motion correction, denoising, motion parameters included as covariates, scrubbing FD > 0.3mm	Primary threshold: p = .001; Cluster-level threshold pFWE < .05.	**Whole group**: ↑ AI → dACC **TU**: ↓ NAcc → dACC, DLPFC **HC**: ↑ NAcc → dACC, DLPFC	*N/A*

ACC, anterior cingulate cortex; AD, alcohol dependence; AI, anterior insula; dACC, dorsal anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; DMN, default mode network; ECN, executive control network; FD, framewise displacement; FWE, family-wise error rate; GP, globus pallidus; HC, healthy control; IFG, inferior frontal gyrus; IPL, inferior parietal lobule LN, lentiform nucleus; MFG, middle frontal gyrus; MPI-S, Max Planck Institute Sample; NAcc, nucleus accumbens; NKI-RS, Nathan Kline Institute – Rockland Sample; OFC, orbitofrontal cortex; PCC, posterior cingulate cortex; rACC, rostral anterior cingulate cortex; SCZ, schizophrenia; SFG, superior frontal gyrus; SN, substantia nigra; SPL, superior parietal lobule; STG, superior temporal gyrus TU, tobacco user; VTA, ventral tegmental area.

* Used an Urgency score that combined PU and NU.

↑ Represents a positive correlation with Urgency

↓ Represents a negative correlation with Urgency

⇔ Represents bidirectional functional connectivity

→ Represents directional (seeded) functional connectivity

N/A – Study did not include Positive Urgency in analyses

Table 3.

Summary of Structural MRI Studies

Study	Sample Size by Group	Quantitative Method	Findings with High Negative Urgency	Findings with High Positive Urgency
Hoptman et al., 2014 (21)	SCZ: 33 HC: 31	Cortical Thickness	**SCZ**: ↓ frontal pole, ↓ medial OFC **HC**: Null	**SCZ**: ↓ frontal pole, ↓ rACC **HC**: Null
Wang et al., 2017 (109)	Obese: 31 Normal Weight: 49	Gray Matter Volume	**Obese**: Null **Normal Weight**: ↓ AI	Null
Ruiz de Lara et al., 2018 (110)	HC: 25 GD: 25	Gray Matter Volume	**HC**: Null **GD**: ↓ VLPFC	*N/A*
Muhlert & Lawrence, 2015 (70)	HC: 152	Gray Matter Volume	↓ DMPFC ↓ temporal pole ↓ ventral striatum	*N/A*
Moreno-Lopez et al., 2012 (139)	HC: 38 CD: 38	Gray Matter Volume	**HC**: ↓ SFG **CD**: ↑ SFG	Null
Albein-Urios et al., 2013 (140)	HC: 34 CD + PD: 32 CD: 44	Gray Matter Volume	HC: Null **CD + PD**: ↑ rolandic operculum **CD**: ↓ rolandic operculum **Whole group**: ↓ temporal pole	Null

AI, anterior insula; CD, cocaine dependent; DMPFC, dorsomedial prefrontal cortex; GD, gambling disorder; HC, healthy control; OFC, orbitofrontal cortex; PD, personality disorder (cluster B); rACC, rostral anterior cingulate cortex; SCZ, schizophrenia; SFG, superior frontal gyrus; VLPFC, ventrolateral prefrontal cortex.

↑ Represents a positive correlation with Urgency

↓ Represents a negative correlation with Urgency

N/A – Study did not include Positive Urgency in analyses

Table 4.

Summary of PET and MRS Studies

Study	Sample Size by Group	Imaging Method	Findings with High Negative Urgency	Findings with High Positive Urgency
Boy et al., 2011 (80)	HC: 12 (Cohort 1) HC: 13 (Cohort 2)	MRS: GABA-edited MEGA-PRESS spectra	↓ GABA in DLPFC[a]	*N/A*
Mick et al., 2017 (65)	HC: 19 GD:15	PET: {11C]Ro15–4513	**HC**: Null **GD**: ↑ GABA-A receptor availability in amygdala, hippocampus and ventral striatum	*N/A*
Clark et al., 2012 (69)	HC: 9 PG: 9	PET: [11C]-raclopride	**HC**: Null **PG**: ↓ D2/D3 receptor binding potential in striatum	**HC**: Null **PG**: ↓ D2/D3 receptor binding potential in striatum

D2/D3, dopamine receptor D2/D3; DLPFC, dorsolateral prefrontal cortex; GABA, gamma-amino butyric acid; GD, gambling disorder; HC, healthy control; MEGA-PRESS, MEshcher-GArwood Point RESolved Spectroscopy; MRS, magnetic resonance spectroscopy; PET, positron emission tomography; PG, pathological gambling.

[a] Finding from cohort 1 replicated in cohort 2

↑ Represents a positive correlation with Urgency

↓ Represents a negative correlation with Urgency

N/A – Study did not include Positive Urgency in analyses