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Interaction of emotion and cognitive control along the psychosis continuum: A critical review

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ABSTRACT

To better understand how emotion impacts cognitive control is important as both influence adaptive behavior in complex real-life situations. Performance changes in emotion and cognitive control as well as in their interaction are often described in psychotic patients as well as in non-clinical participants who experience psychosis-like symptoms. These changes are linked to low motivation and limited social interaction. However, it is unclear whether these changes are driven by emotion, cognitive control, or an interaction of both. This review provides an overview of neuroimaging evidence on the potential interaction of emotion and cognitive control along the psychosis continuum. The literature confirms that over-sensitivity towards negative and lowered sensitivity towards positive emotional stimuli in tasks exploring emotion-cognitive control interaction are associated with the severity of positive and negative symptoms in psychosis. Changes in the dynamic interplay between emotion and context-sensitive cognitive control, mediated by arousal, motivation, and reward processing may underlie poor interpersonal communication and real-life skills in psychosis. In addition, structural and functional changes in subcortical and cortical associative brain regions (e.g., thalamus, basal ganglia, and angular gyrus) may contribute to alterations in emotion and cognitive control interaction along the psychosis continuum. There is limited evidence on how antipsychotic medication and age at illness-onset affect this interaction.

1. Introduction

Next to positive (e.g., hallucinations, delusions) and negative (e.g., poverty of speech, apathy) symptoms, impairments of emotion processing, cognitive control, and their interaction are key features of psychosis (Becerril and Barch, 2011; Benes, 2010; Dichter et al., 2010; Minzenberg et al., 2009; Ruocco et al., 2014). Emotion and cognitive control processes conjointly contribute to socially appropriate and goal-directed behavior (Drevets and Raichle, 1998; Gray et al., 2002; Pessoa, 2008). Changes in how emotion impacts cognitive control have been linked to reduced motivation and poor daily-life functioning in psychotic patients (PP, Anticevic et al., 2012; Becerril and Barch, 2011; Bertocci et al., 2012; Rey et al., 2014). Neural changes observed in psychosis, such as hypoactivation of the cognitive control system (e.g., lateral prefrontal cortex [PFC], anterior cingulate cortex) or hyperactivation of the emotion processing system (e.g., amygdala, orbitofrontal cortex, hippocampus, insula), have been associated with the impaired ability to use emotional cues to guide cognitive control processes required for efficient social interactions (Anticevic et al., 2012; Becerril and Barch, 2011; Dichter et al., 2010). However, it is still unclear if such impairment arises from inefficient cognitive control, affected emotion processing, or from dysfunctional neural processing in brain regions that engage in converging and transferring sensory information to emotion processing and cognitive control systems (e.g., thalamus, basal ganglia, angular gyrus; Eack et al., 2016; Ettinger et al., 2014, 2015; Pauly et al., 2008, 2010). Behavioral and neuroimaging studies have shown that these changes also extend to clinically high-risk psychosis (CHR-P) individuals and non-clinical individuals with psychosis-like experiences (Table 1, Fig. 1), suggesting that the respective impairments manifest early on and potentially deteriorate following illness onset (Addington et al., 2008; Modinos et al., 2015; Mohanty et al., 2005, 2008; Nenadic et al., 2015; Pauly et al., 2010; van't Wout et al., 2004).

The current review has two main goals: (i) to identify and discuss

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Table 1
Glossary of terms.

S. No	Term	Definition
1	Affect	An abstract concept that represents the experience of an emotion. Valence, arousal, and motivation are three dimensions of affect.
2	Cognitive control	An adaptive mechanism that uses attentional processes (e.g., sustained attention, selective attention, inhibition, updating and maintenance of contextual information) to select relevant responses and to inhibit inappropriate responses while maintaining contextual information to guide goal-directed behavior (Botvinick et al., 2001; Niendam et al., 2012).
3	Clinically high-risk psychosis	Sub-clinical individuals experiencing attenuated (positive) psychotic symptoms (e.g., hallucinations, delusions) for at least 3 months that are clinically relevant but below the required threshold for a DSM-IV axis I psychotic disorder diagnosis. In this prodromal phase, individuals display decline in daily functioning for at least 12 months; however, they understand and have insight into their symptoms (for details see Woods et al., 2010; McGlashan et al., 2010; Addington et al., 2011). Depending upon the level of distress caused, these individuals may or may not receive antipsychotic medication.
4	Emotion-cognitive control interaction	The term emotion-cognitive control interaction refers to the dynamic reciprocal communication of emotion processing and cognitive control networks either directly or via intermediary associative brain regions (Eack et al., 2016; Ettinger et al., 2014, 2015; Pauly et al., 2008, 2010). This interaction is often influenced by internal factors such as motivation, arousal and self-regulation.
5	Hyperarousability	A personality trait where external or internal stress/stressful events cause a strong increase in arousal making an individual over-sensitive towards environmental stimuli. This state of hyperarousal or over-sensitivity may cause anomalous perceptual experiences such as hallucinations in highly vulnerable individuals (Clamor et al., 2014).
6	Psychotic patients	Patients suffering from schizophrenia and related disorders such as schizoaffective disorder and bipolar disorder with psychosis.
7	Psychosis	Transdiagnostic range of conditions symbolized by severe distortions in thoughts and emotions. Positive symptoms include false beliefs (delusions) and anomalous perceptual experiences (hallucinations), whereas negative symptoms include depression, apathy, and anhedonia.
8	Psychosis-prone individuals	Non-clinical healthy individuals with psychotic-like experiences (e.g., hallucinations). These symptoms are not clinically relevant and therefore have no negative effect on daily life functioning and nor cause distress to the individual (Allen et al., 2006). As the symptoms cause no distress, these individuals are not under antipsychotic medication. These individuals usually score high on questionnaires measuring predisposition to symptoms of psychosis (e.g., Launay-Slade Hallucinations Scale).

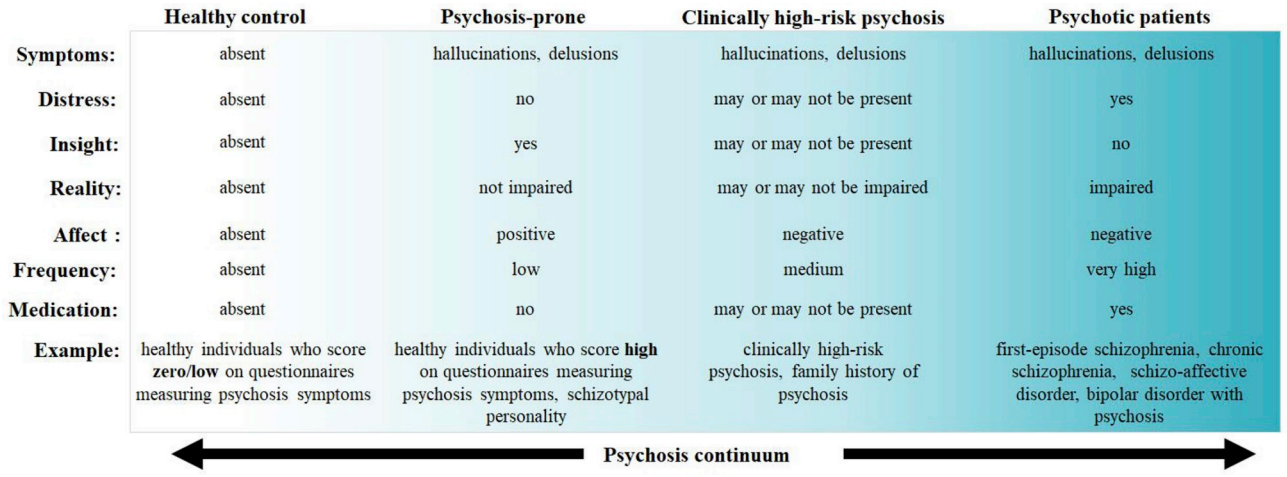

Fig. 1. The psychosis continuum. This continuum implies that psychotic patients and non-clinical populations experience the same symptoms of psychosis, however, the quality (affect content, frequency, effect of symptoms on daily-life functioning) is different. Definitions of the groups depicted on the continuum are provided in Table 1.

the factors affecting the interaction of emotion and cognitive control, and (ii) to distinguish whether altered emotion processing is a defining feature of psychosis independent of cognitive control or altered cognitive control results in impaired emotion processing. A systematic review of the functional magnetic resonance imaging (fMRI) literature on the interaction of emotion and cognitive control along the psychosis continuum (Fig. 1), published until January 2019, was conducted using the PubMed database (https://www.ncbi.nlm.nih.gov/pubmed). In line with the definition of cognitive control used in Table 1, the review focuses on experimental studies employing typically used "cognitive control" tasks (e.g., Stroop, Flanker or Simon task) as well as an n-back working memory task, a continuous performance task, an oddball paradigm or Go/No-Go and emotion regulation tasks where attentional processes involved in cognitive control are used to examine the impact of emotion on cognitive control. Advanced search terms included the keywords ("schizophrenia" or "psychosis" or "bipolar disorder" or "schizotypy" or "schizotypal personality disorder" or "at-risk psychosis" or "high-risk psychosis" or "psychosis-prone") AND ("functional magnetic resonance imaging" or "fMRI" or "functional neuroimaging") AND ("cognitive control" or "cognitive conflict" or "conflict resolution" or "executive control" or "attentional control" or "executive attention" or "executive functioning" or "attention" or "Stroop" or "Flanker" or "Simon") AND ("affect" or "emotion perception" or "emotion dysregulation" or "emotion regulation" or "emotional conflict" or "emotional control" or "emotion"). Titles and abstracts of eligible studies were first screened for these keywords before the full text was analyzed (see Table 2). A total of 26 studies met the above search criteria and were included in the current review.

To address our first aim, the identified studies were assessed in terms of how emotion was implemented in the respective task settings to identify a set of discernible categories influencing emotion-cognitive control interaction. These categories included valence-specificity, antipsychotic medication, age at illness onset, and illness chronicity. Each of these categories have shown to influence both cognitive control and emotion processing in psychosis (Eack et al., 2016; Pinkham et al., 2007; Pino et al., 2014; Rajji et al., 2009). The review then discusses the

Table 2
Summary of fMRI studies examining the interaction of emotion and cognitive control in psychosis.

Study	Paradigm (stimuli)	Emotion		Patient Information					Patient group as compared to control group	Instructions and performance
		Valence (sub-type)	Task-relevance	Number	Age [patient (SD), HC(SD)]	Illness duration (SD)	Medication (typical/atypical antipsychotics)	Neuropsychological assessment		
Schizophrenia										
(Pauly et al., 2008)	2-back WM task (letters)	Negative (disgust/ unpleasant), neutral smells	Irrelevant, non-target	12 Adolescent-onset SZ; 12 HC	17.5 (0.55); 17.5 (1.76)	1.4[a]	Yes (atypical)	PANSS, GAF, CPT-IP, LN span, TMT, MWT-B, verbal fluency, Sniffin' sticks	• Interaction of WM and negative emotion: ↑ activity in EP regions (OFC, mFC) ↓ activity in WM regions (dlPFC, ACC)	I: 2-back task instructions, respond to target accurately and quickly; emotion manipulation through olfactory simulation every 5 s P: decreased RT for emotional trials in both groups but no significant main effect of group or interaction of group and emotion
(Dichter et al., 2010)	Forced-choice visual oddball task (circles, squares, aversive and neutral pictures)	Negative (aversive), neutral pictures	Irrelevant, non-target	12 SZ/ schizoaffective disorder, 13 HC	29.4 (10.2); 31.6 (10.7)	No info	Yes (atypical and other)	SANS, SAPS, IQ test, NART	• Inhibition of aversive stimuli: ↓ activity in executive and limbic regions (dlPFC, ACC) • Inhibition of aversive stimuli: ↑ activity in ventral fronto-limbic areas for aversive stimuli • Target stimuli: ↑ activity in executive and limbic regions (dlPFC, ACC)	I: left index finger response to circles and right to all others P: slower response and poor accuracy in SZ but no significant main effect of group
(Becerril and Barch, 2011)	N-back task with neutral, happy and fearful faces	Positive (happy), negative (fearful) and neutral faces	Irrelevant, target dimension	38 SZ; 32 HC	36.66 (9.12); 36.19 (10.86)	17.4 (11.2)	Yes (atypical)	SANS, SAPS	• Negative > positive and neutral: ↑ Amygdala, hippocampus, middle frontal (dlPFC) activity • No effect of medication.	I: n-back task instructions P: *RT:* Neutral > Negative (significant): SZ Positive > Neutral: SZ Neutral = Negative: HC Positive = Neutral: HC *Accuracy:* Negative > Neutral: SZ (significant) Neutral > positive: SZ Negative > neutral: HC Neutral > positive: HC
(Diaz et al., 2011)	Short term memory task: encode, maintain (while looking at negative and neutral distractor pictures) and recognize through forced choice probe	Negative (no info) and neutral pictures	Irrelevant, Non-target	11 SZ; 17 HC	32.57 (12.7); 24.01 (3.89)	No info	Yes (mixed psychotropics)	PANSS	• No effect of negative (vs. neutral) distractors in SZ; ↓ activity in pre-frontal and amygdala regions in SZ	I: encode and retrieve P: no significant interaction of group and trial type/emotion Same performance for negative and neutral trials in SZ
(Anticevic et al., 2011)	Visual WM task (encode, delay fixation, distractor, delay fixation, retrieve)	Negative (threat: on the basis of picture) and neutral pictures	Irrelevant	28 SZ; 24 HC	36.39 (9.54); 37.18 (7.59)	No info	Yes (no info)	SAPS, SANS	• Negative: ↓ activity in dlPFC, vlPFC, amygdala as compared to neutral and HC	I: encode and retrieve P: significant effect of diagnosis × distraction however, no difference in interference between negative and neutral for SZ
				28 SZ; 24 HC		No info	Yes (no info)	SANS, SAPS		

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Table 2 (*continued*)

Study	Paradigm (stimuli)	Emotion		Patient Information					Patient group as compared to control group	Instructions and performance
		Valence (sub-type)	Task-relevance	Number	Age [patient (SD), HC(SD)]	Illness duration (SD)	Medication (typical/atypical antipsychotics)	Neuropsychological assessment		
(Anticevic et al., 2012)	Simple perceptual decision task (fixation➔ emotionally aversive/ neutral distractor➔ flanked by blue and green circle➔fixation)	Negative (threat) and neutral pictures	Irrelevant; Shown before and during the trial, non-target		36.79 (7.72); 36.73 (8.85)				• Negative > neutral: negative connectivity between amygdala and prefrontal cortex in SZ	I: indicate the location of blue circle P: SZ were slower for negative distraction but no significant interaction of group × distraction effect
(Vercammen et al., 2012)	Verbal emotional go/ no-go task (words)	Positive (no info), negative (no info) and neutral words	Relevant (attend) and irrelevant (inhibit) blocks	20 SZ; 23 HC	34.4 (7.8); 33.3 (7.1)	No info	Yes (atypical)	PANSS, SQLS, WAIS-III	• Negative inhibition: No activity in dorsal prefrontal-parietal network for SZ • Positive inhibition: ↑ activity in middle frontal cortex	I: inhibit or attend instructions before the block P: SZ were significantly slower and made more errors in emotional trials (harder to inhibit negative than positive distractors)
(Tully et al., 2014)	Multi-source interference task with negative and neutral pictures in the background (MSIT-Emotion)	Negative (sad) and neutral pictures	Irrelevant, non-target	23 SZ; 24 HC	39.3 (9.60); 35.54 (12.23)	No info	Yes (both typical and atypical)	PANSS; SAS-SR	• Negative incongruent: ↓ activity in dorsal LPFC in SZ	I: identity of number different from the other two by pressing a button (task is a combination of Flanker and Simon) P: no significant main effect of group; SZ responded significantly slower and made more errors during negative as compared to neutral trials
(Kim et al., 2015)	Verbal memory retrieval (encode, delay, retrieve, words)	Negative (mixed-threat) and neutral words	Irrelevant, target dimension	15 SZ; 15 HC	28.4 (8.0); 29.7 (5.3)	6.8 (4.5)	Yes (psychotropic, no info)	PANSS	• ↓ activity in OFG and insula for negative words	I: Memorize and retrieve P: Performance decreased with negative emotional words more for SZ than HC; no significant group difference on accuracy during retrieval trials
(Eack et al., 2016)	n-back task (letters, random flanking of emotional faces to create emotional interference)	Positive (happy), negative (fear) and neutral faces	Irrelevant, non-target, present in some trials	20 SZ; 20 HC	27.80 (6.61); 26.50 (5.82)	4.85 (3.18)	Yes (psychotropic, no info)	BPRS, GAS	• Positive > negative emotion stimuli: ↑ activity in bilateral striatum, OFG, vmPFC	I: n-back task instructions P: no significant effect of group or condition or emotion or group-condition interaction.
(Mukherjee et al., 2016)	Modified version of the emotional face assessment paradigm (face match)	Negative (mixed: anger, fear) and neutral faces	Irrelevant, target	22 SZ, 29 HC	45.82 (7.55); 46.38 (7.27)	No info	No info	Social functioning, deficit syndrome severity, SES	• Emotional distractors: ↓ connectivity between amygdala and mPFC; ↓ activity in dACC	I: face match P: accuracy decrease for emotional trials in SZ as compared to both neutral and HC
(Comte et al., 2017)	Variable attention and congruency task (VAAT, visual/emotional pictures)	Positive (joy) and negative (mixed: fear, disgust, anger) pictures	Relevant, target	26 SZ; 33 HC	32.31(8.87);	No info	Yes (no info)	PANSS	• ↓ amygdala and ACC activity for bottom up processes; ↑ connectivity between vACC and dACC and vACC and dlPFC • Negative correlation between dlPFC and antipsychotic dosage	I: Determine the emotional content of center face or background picture P: SZ significantly slower but no main effect of valence
(Park et al., 2018)	Visual Simon task	Positive (mixed) and negative (mixed) pictures	Relevant, target	17 SZ; 20 HC	27.2 (7.3); 26.1 (5.1)	No info	Yes (typical)	PANSS	• Negative emotion: ↓ amygdala activity and ↓ connectivity with dlPFC • Controlled for medication effects	I: right or left button in response to positive or negative emotion P: significant slower RT for

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Table 2 (*continued*)

Study	Paradigm (stimuli)	Emotion		Patient Information					Patient group as compared to control group	Instructions and performance
		Valence (sub-type)	Task-relevance	Number	Age [patient (SD), HC(SD)]	Illness duration (SD)	Medication (typical/atypical antipsychotics)	Neuropsychological assessment		
										both negative and positive trials in SZ
Bipolar disorder										
(Pavuluri et al., 2008)	Pediatric color word matching task (words)	Positive (happy, excitement), negative (sad, depressed) and neutral words	Target, irrelevant	10 PBD; 10 HC	15.0 (2.36); 16.2 (1.32);	4 months before testing	No	WASI; YMRS; CDRS-R; WRAT-3, Reading (SS)	• Negative: ↑ activity in rACC and left amygdala, ↓ activity in vlPFC, dlPFC • Positive: ↓ posterior ACC, insula, vlPFC, OFC; ↑ activity in amygdala	I: match color of the word with one of two colored circles below P: overall PBD slower and less accurate than HC but no significant main effect of group; significant effect of valence. RT: Negative > Neutral > Positive
(Passarotti et al., 2011)	2-back task (faces)	Positive (happy), negative (angry) and neutral faces	Target, relevant	17 PBD; 13 HC	14.29 (2.05); 14.38 (3.57)	No info	Yes (medication free 7 days before baseline scanning, second generation antipsychotics)	YMRS; WASI-FSIQ; CDRS-R;	• Negative: ↓ activity in vlPFC, caudate • Positive: ↑ activity in amygdala, vlPFC, r-dlPFC, MTG, MFG	I: 2-back: match both face and emotion P: No significant difference between BD and HC for RT; more errors in BD than HC in emotional conditions; overall PBD slower and less accurate than HC PBD➔ RT: Negative > positive > neutral Accuracy: positive > negative ≈ neutral HC➔ RT: negative > neutral > positive Accuracy: positive > negative ≈ neutral
(Bertocci et al., 2012)	2-back task (letters, flanked by emotional face distractors)	Positive (happy), negative (fear) and neutral faces	Non-target, irrelevant	18 BD; 16 HC	31.94 (8.54); 29.74 (8.22)	14.00 (6.63)	Yes (mixed psychotropic)	HAMD-25; YMRS; NART; STAI	• ↓ activity in ACC and ↑ activity in striatum (putamen)	I: n-back related P: RT was slower for face distractor trials, no significant group difference
(Townsend et al., 2013)	Emotion regulation task (pictures, passive viewing and emotion downregulation)	Negative (no info) and neutral pictures	Target, relevant	30 BD; 26 HC	37.9 (12.6); 35.5 (12.4)	20.7 (13.6)	Yes (mixed psychotropic)	YMRS; HDRS	Emotion down-regulation: • ↓ activation in bilateral vlPFC, bilateral ACC and posterior CC, medial frontal gyrus and bilateral dlPFC • Amygdala: ↓ activity for both groups for negative downregulation compared to observe condition	I: two conditions ➔ passive view, decrease emotion P: NA
(Favre et al., 2013)	Modified version of the word-face emotional Stroop task (emotional words embedded on emotional faces)	Positive (happy), negative (fear) and neutral faces and words	Relevant, target	16 BD; 16 HC	40.47 (11.8); 40 (12.5)	13.9 (6.8)	Yes (mixed psychotropic)	MADRS; YMRS; STAI	• Incongruent > congruent: ↓ activity in bilateral inferior and left superior frontal gyri, right insula, right fusiform gyrus and bilateral occipital gyri	I: identify emotional expression of faces and ignore emotional words embedded on them; word and face could be congruent (both positive affect, "joy" embedded on happy face) or incongruent (one positive

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Table 2 (*continued*)

Study	Paradigm (stimuli)	Emotion		Patient Information					Patient group as compared to control group	Instructions and performance
		Valence (sub-type)	Task-relevance	Number	Age [patient (SD), HC(SD)]	Illness duration (SD)	Medication (typical/atypical antipsychotics)	Neuropsychological assessment		
									• Negative > positive incongruent: ↑ bilateral hippocampus, parahippocampal gyri and the left middle temporal gyrus activity.	and other negative affect, "joy" embedded on an angry face) P: overall significant larger RT in BD, but similar accuracy, no significant group × valence or group × Stroop effect Significant effect of emotional valence: negative incongruent trials generated slower RT and more errors than positive incongruent trials in both groups
(Brotman et al., 2014)	Constrained and unconstrained processing of emotional faces	Positive (happy), negative (angry and fear) and neutral faces	Target, relevant	36 PBD; 26 BD; 57 PHC; 62 HC	14.77(2.55); 41.70(10.30); 14.30 (2.57); 34.24 (9.54)	4.03[a]; 20.26[a]	Yes (mixed psychotropic)	WASI; SIGH-SAD; YMRS; CDRS	• (PBD > BD) > HC explicit and implicit conditions: ↑ activity in Amygdala • Explicit and implicit conditions: ↓ in IFG (only positive), r-ACC (negative and positive), putamen (explicit, positive) •Passive view negative: ↑ r-ACC, putamen	I: passive viewing, implicit attention: "How wide is the nose?", two explicit attention: "how hostile is the face?", "how afraid are you?". P: PBD rated neutral faces as more hostile; overall patients responded significantly slower
(Rey et al., 2014)	Modified version of the word-face emotional Stroop task (emotional words embedded on emotional faces) with either low (same response button) or high attentional demands (response button depended upon stimulus gender)	Positive (joy) and Negative (fear) faces and words	Relevant, target	11 BD; 12 HC	42.6 (11.4); 41.3 (12)	21.8 (9.9)	Yes (psychotropic)	YMRS; MADRS-S	• ↓ activity in cognitive control network e.g., rACC, MFG	I: identify emotional expression of faces and ignore emotional words embedded on them; word and face could be congruent (both positive affect, "joy" embedded on happy face) or incongruent (one positive and other negative affect, "joy" embedded on an angry face) P: RT was significantly longer for BD for both congruent and incongruent trials; no effect of valence
(Favre et al., 2015)	Modified version of the word-face emotional Stroop task (emotional words embedded on emotional faces)	Positive (joy) and negative (fear) faces and words	Relevant, target	14 BD; 13 HC	44.07 (9.63); 44.08 (10.85)	16.08 (11.10)	Yes (psychotropic)	MADRS; YMRS	• ↓ activity in dlPFC for conflict monitoring • ↑ activity in amygdala during emotional conflict	I: identify emotional expression of faces and ignore emotional words embedded on them; word and face could be congruent (both positive affect, "joy" embedded on happy face) or incongruent (one positive and other negative affect, "joy" embedded on an angry face) P: BD were slower but the difference was not significant

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Table 2 (*continued*)

Study	Paradigm (stimuli)	Emotion		Patient Information					Patient group as compared to control group	Instructions and performance
		Valence (sub-type)	Task-relevance	Number	Age [patient (SD), HC(SD)]	Illness duration (SD)	Medication (typical/atypical antipsychotics)	Neuropsychological assessment		
(Corbalan et al., 2015)	Emotion regulation paradigm (view or downregulate, pictures);	Negative (no info) and neutral pictures	Relevant, target	19 BD; 17 HC	41.0 (12.5); 41.4 (13.3)	No info	Yes (no info)	HAMD-29; YMRS; SIGH-SAD; MADRS; CGI; STAI-S	• Negative downregulation: ↑ amygdala activity • Negative passive view: ↑ vlPFC • Controlled for medication effects	I: passive view, downregulate emotion P: not applicable
(Mukherjee et al., 2016)	Modified version of the emotional face assessment paradigm (face match)	Negative (anger, fear) and neutral faces	Irrelevant, target	15 BDP, 29 HC	42.93 (6.46); 46.38 (7.27)	No info	No info	Social functioning, deficit syndrome severity, SES	• No difference between BD and HC	I: face match P: no significant difference
CHR psychosis or non-clinical psychosis-prone individuals										
(Mohanty et al., 2005)	Emotional Stroop task (words)	Negative (no info), Positive (no info), neutral words	Irrelevant; Emotional target word	17 high PP; 17 HC	19.1 (1.9); 20.5 (3.9)	NA	No	CSPP, PSWQ	•Negative vs Neutral: - ↑ activity in amygdala, hippocampus; right dlPFC, basal ganglia, cerebellum - ↓ left dlPFC, ITG, STG, MOG • No info about Positive vs. Neutral	I: identify the color of the word, ignore the meaning P: Stroop interference of High PP was more than HC but not significantly different
(Modinos et al., 2010)	Passive viewing of pictures (viewing neutral, viewing negative, and reappraising negative)	Negative, neutral pictures	Relevant, target	17 high PP; 17 low PP	High PP = 19.8 (1.8); Low PP = 21 (2.8)	NA	No	CAPE	High PP: • ↓ prefrontal-amygdala coupling • ↑ prefrontal activity during reappraisal, amygdala response did not decrease during reappraisal • ↓ cognitive control of emotion	I: View: view the photo and experience the emotion naturally Attend: continue viewing Reinterpret: reappraise the content of negative picture so that it no longer elicited negative response Rate the negative affect after few seconds Relax: relax P: both groups reported successful reduction of experienced negative emotion
(Pauly et al., 2010)	2-back WM task (words);	Negative (disgust), neutral smells	Non-target, irrelevant	12 CHR psychosis; 12 HC	24.22 (4.61); 24.46 (4.67)	No info	Yes (mixed medication:, some received antipsychotics)	PANSS, HDRS, GAF, MWT-B, LN-span, CPT-IP, TMT, PERT, Sniffin' Sticks	Interaction of emotion and WM: • ↓ activity in STG, ITG, caudate nucleus, posterior insula, supramarginal gyrus • ↑ activity in thalamus, cerebellum, posterior ITG	I: n-back WM task related P: no difference between groups

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Table 2 (*continued*)

Study	Paradigm (stimuli)	Emotion		Patient Information			Medication (typical/atypical antipsychotics)	Neuropsychological assessment	Patient group as compared to control group	Instructions and performance
		Valence (sub-type)	Task-relevance	Number	Age [patient (SD), HC(SD)]	Illness duration (SD)				
(van der Velde et al., 2015)	Emotional regulation task (pictures)	Negative and neutral pictures	Target, relevant	15 CHR psychosis, 16 HC	23.1 (4.4); 22.1 (3.6)	No info	Yes (mixed medication: antipsychotics, antidepressants)	PANSS	• Negative appraisal: ↓ vlPFC	I: attend neutral, attend negative and reappraise P: no significant difference

Abbreviations: SZ = Schizophrenia, BD = Bipolar disorder, HC = healthy controls, PP = psychosis proneness, CHR = clinically high risk, I = Instructions, P = Performance, EST = emotional Stroop task, EP = emotion processing, WM = working memory, RT = reaction time, dlPFC = dorsolateral prefrontal cortex, rACC = rostral anterior cingulate cortex, OFC = orbitofrontal cortex, mFC = middle frontal cortex, STG = superior temporal gyrus, ITG = inferior temporal gyrus, vmPFC = ventral medial prefrontal cortex, rs-fc = resting state functional connectivity, tb-fcMRI = task based functional connectivity, NA: not applicable. PANSS = positive and negative syndrome scale, SANS = scale for the assessment of the negative symptoms, SAPS = scale for the assessment of the positive symptoms, SCID – DSM = Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, SQLS = schizophrenia quality of life scale, MSCEIT = Mayer–Salovey–Caruso Emotional Intelligence Test, BPRS = brief psychiatric rating scale, VAAT = variable attention and congruency task; MADRS = Montgomery and Asberg Depression Rating Scale, YMRS = Young Mania Rating Scale, CAPE = Community Assessment of Psychic Experiences Questionnaire; WRAT = Wide Range Achievement Test – Third Edition (Reading Subtest), KYMRS = Kiddie Young Mania Rating Scale, CDRS-R = Child Depression Rating Scale-Revised, PBD = Pediatric Bipolar Disorder, WASI IQ = Wechsler Abbreviated Scale of Intelligence Intelligent Quotient, SIGH-SAD = Structured Interview Guide for the Hamilton Depression Rating Scale; DIGS = the Diagnostic Interview for Genetic Studies; SAS-SR = social adjustment scale – self-report; CPZ = chlorpromazine; GAS = global assessment scale; PERT = Penn Emotion Recognition Test; TMT = Trail Making Test; CPT-IP = computerized identical pairs version of the Continuous Performance Test; LN span = working memory-letter–number span; MWT-B = Mehrfachwahl-Wortschatz-Intelligenztest-Version B; GAF = Global Scale of Functioning; HDRS = Hamilton Rating Scale for Depression; CSPP = Chapman scales for psychosis proneness, PSWQ = penn state worry questionnaire, NART = National adult reading test, SAS = Social adjustment scale, SES = Socio-economic status, HAMD = Hamilton depression rating scale, STAI = State-trait anxiety inventory, CGI = Clinical global impression, PERT = penn emotion recognition test

[a] Illness duration was calculated by subtracting mean illness onset from mean age; ↑increased; ↓decreased.

mediating role of affect in the interaction of emotion and cognitive control in psychosis. Lastly, we address the likely neuroanatomical causes underlying significant changes in the interaction of emotion and cognitive control in psychosis based on the reviewed literature.

2. Experimental paradigms testing the interaction of emotion and cognitive control

The interaction of emotion and cognitive control has been studied in multiple task settings. Tasks may engage conflict resolution and monitoring, selective and sustained attention, inhibition, working memory, emotion regulation, and require constant maintenance of the task context (Anticevic et al., 2012; Anticevic et al., 2011; Becerril and Barch, 2011; Diaz et al., 2011; Dichter et al., 2010; Mukherjee et al., 2016; Tully et al., 2014; Vercammen et al., 2012). Distinct manipulations of emotion are introduced at different time points into these task settings. Examples include tasks in which emotional information precedes a trial, probing conflict to challenge reorienting of attention to resolve a conflict (Anticevic et al., 2011; Diaz et al., 2011). In other tasks, emotional information is presented throughout a trial to challenge the sustaining of attention and maintaining of contextual information (Anticevic et al., 2012; Besnier et al., 2011; Dichter et al., 2010). Overall, emotion has been manipulated in cognitive control tasks in three ways (Fig. 2):

(i) Prior passive cueing, i.e., an emotional cue is passively/implicitly presented, prior to a cognitively challenging task, without requiring an explicit behavioral response. In this case, emotion is not part of the target dimension established by the task instructions and, thereby, can be considered task-irrelevant.

Although presenting emotional distractors prior to task-relevant stimuli only transiently captures attention, it disrupts the continuous maintenance of contextual information that is necessary to execute task-appropriate behavior during cognitive control tasks. This has immediate consequences for task performance, reflected in slower reaction times and higher error rates. Compared to healthy controls, PP tend to show larger interference effects when emotional distractors precede a cognitively challenging trial (such as the retrieval of information in a short-term memory task or the conflict resolution in a Stroop task) (Anticevic et al., 2011; Diaz et al., 2011). On the other hand, performance of CHR-P individuals was not significantly different from healthy controls in these tasks (Pauly et al., 2010).

(ii) Continued passive exposure, i.e., an emotional cue is passively presented in the background whilst performing an unrelated task. Here, emotion may or may not be part of the target dimension but is task-irrelevant. In this setting, task-irrelevant emotion draws attention away from task-relevant cognitive processes, resulting in deteriorated task performance. Examples include typically used conflict processing tasks such as the emotional Stroop or Flanker tasks, in which participants have to identify the ink color of emotional words while ignoring the stimulus meaning (Ben-Haim et al., 2016). Here, emotion is part of the target dimension although it remains task-irrelevant (Besnier et al., 2011). Healthy participants respond faster to negative emotion (as compared to neutral) when it is part of the target dimension that results in facilitation of conflict processing during typically used cognitive control tasks (Kanske and Kotz, 2011a, 2011b, 2011c). However, studies using cognitive control tasks such as n-back working memory task have yielded mixed results (Kessel et al., 2016; Luo et al., 2014). Compared to healthy controls, PP take longer to identify the color of negative than neutral words, indicating greater task-interference due to a heightened attentional bias towards task-irrelevant emotional information (Besnier et al., 2011). The background presentation of emotional distractors in a

modified version of the Simon task is another variant of this setting. The Simon task requires participants to respond to the location of a digit on a screen or entails a forced choice task in which participants judge the color of geometric figures embedded in the distractor (Anticevic et al., 2012; Dichter et al., 2010). Here, emotion is not part of the target dimension, and it is task-irrelevant. PP take longer to react to emotional stimuli in visual and verbal n-back working memory tasks (Dichter et al., 2010; Pauly et al., 2008). Impaired task performance in an emotional Stroop task extends to CHR-P and non-clinical individuals who experience psychotic-like symptoms in some studies (Yaffe and Walder, 2016; van Strien and van Kampen, 2009; Besnier et al., 2009), whereas other studies report no significant differences between these populations and healthy controls (Mohanty et al., 2005; van't Wout et al., 2004).

(iii) Explicit selective attention, i.e., an emotional cue is the target dimension and therefore task-relevant. Conflict or task-interference arises with competing attentional demands associated with two different emotional stimuli. These stimuli can be of the same (e.g., both visual; Comte et al., 2017) or different modalities (e.g., auditory vs. visual; Zinchenko et al., 2017). PP struggle to consciously and selectively monitor emotional stimuli that are of primary relevance for a task but incongruent (Comte et al., 2017; Park et al., 2018). They also experience difficulty (reflected in slower reaction times and low accuracy) in judging the emotion conveyed by a face embedded in a broader emotional background serving as an emotional distractor (Comte et al., 2017). Similarly, they showed impaired task performance (i.e., slower reaction times) in a modified version of the Simon task that uses emotional face stimuli, in which participants have to press a right-hand response button for a positive stimulus and a left-hand button for a negative emotional stimulus (Park et al., 2018). We did not find any studies with CHR-P individuals or non-clinical individuals with psychotic-like symptoms (Table 2). In addition, emotion regulation tasks are also included in this category, as they require (explicit) attentional control of emotion processing regions to efficiently regulate the emotional experience (Brotman et al., 2014; Corbalan et al., 2015; Modinos et al., 2010; Townsend et al., 2013; van der Velde et al., 2015). Taken together, emotion seems to disrupt cognitive control processes in behavioral tasks and this disruption is exaggerated in PP (Table 2).

3. Key factors contributing to the interaction of emotion and cognitive control

Several key factors (e.g., emotional valence, medication effects, age at illness onset, illness chronicity) influencing the interaction of emotion and cognitive control can be differentiated on the basis of the current literature review. These factors are separately discussed in the ensuing sections.

3.1. Emotional valence

Although both positive and negative emotions attract attention, they evoke distinct behavioral responses (Fredrickson, 1998, 2001; Fredrickson and Branigan, 2005). Positive emotion is associated with the broadening of attentional scope, relaxation and well-being, whereas negative emotion is linked to attention constriction and decreased attentional flexibility (Eysenck et al., 2007; Fredrickson and Branigan, 2005; Johnson et al., 2010). This suggests that they may also differentially influence task performance when emotion interacts with cognitive control (Fredrickson, 1998, 2001; Fredrickson and Branigan, 2005). Earlier studies testing healthy individuals in cognitive control tasks have shown that valence-specific influence on cognitive control depends on the context and task-relevance of the emotional stimulus (Berger et al., 2017; Hart et al., 2010; Kanske and Kotz, 2011b, 2011c, 2011d; Kessel et al., 2016).

Most studies examining the impact of negative emotion on cognitive control in PP have reported impaired task performance (i.e., slower reaction time; Bertocci et al., 2012; Brotman et al., 2014; Comte et al.,

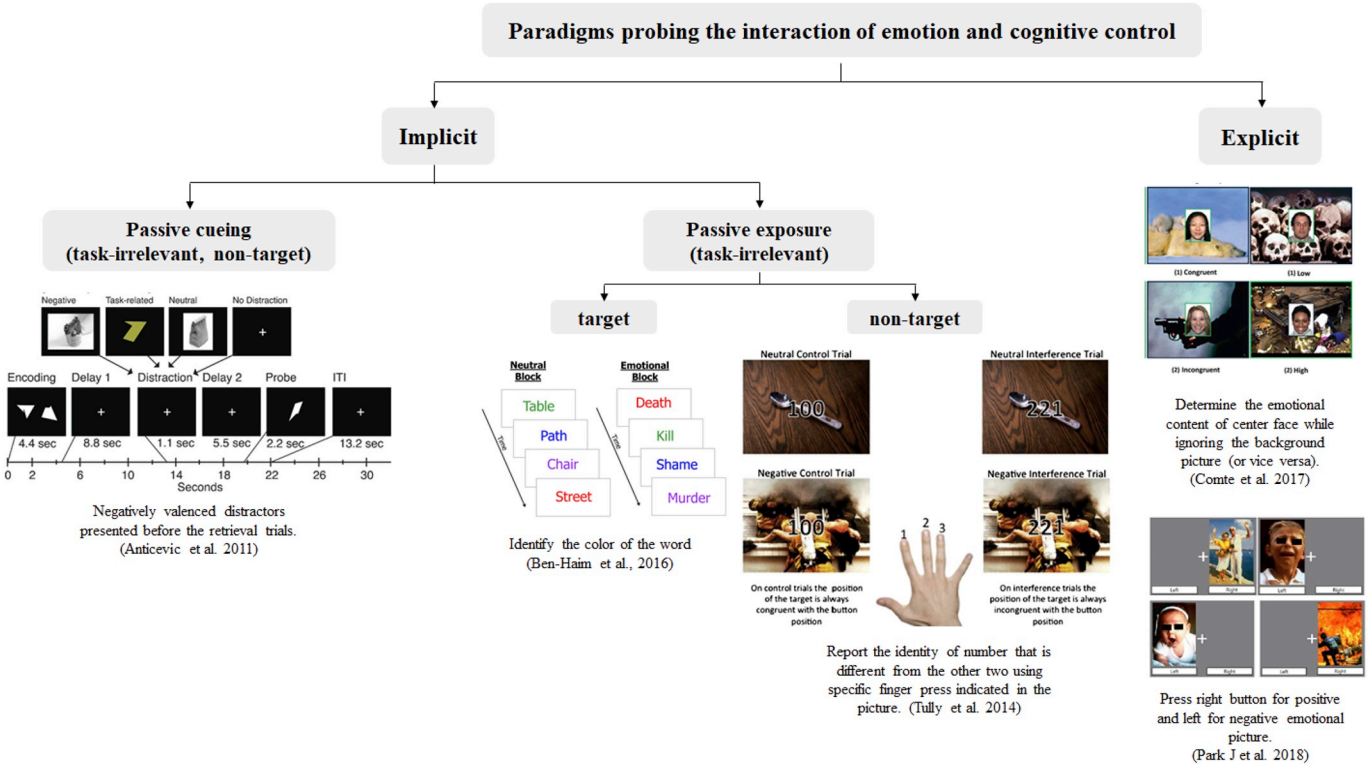

Fig. 2. Paradigms testing the impact of emotion on cognitive control.

2017; Corbalan et al., 2015; Dichter et al., 2010; Favre et al., 2015; Kim et al., 2015; Mukherjee et al., 2016; Park et al., 2018; Pauly et al., 2008; Pavuluri et al., 2008; Rey et al., 2014), whereas others have shown an enhanced effect of negative emotion (i.e., faster reaction time) compared to neutral stimuli (Becerril and Barch, 2011), or even null effects (Anticevic et al., 2011; Diaz et al., 2011; Eack et al., 2016; Kim et al., 2015). Impaired task performance in cognitive control tasks with negative emotional stimuli in PP could result from an enhanced attentional bias towards stimuli representing threat, fear, and paranoia. This potentially relates to specific illness symptoms (Kapur, 2003; Kinderman et al., 2003). For instance, as hallucinations often elicit negative affect, PP are likely to be more sensitive to negative cues, which may constitute more potent distractors than neutral stimuli (Kapur, 2003; Kapur et al., 2005). Similarly, as negative emotions exert a lasting effect on attention (Strauss et al., 2008), they might continue to influence cognitive processes even when the negative stimulus is absent (Anticevic et al., 2011; Diaz et al., 2011). Thereby, PP may assign salience to neutral stimuli (Kapur, 2003). As negative and neutral stimuli are presented in close temporal succession in cognitive control tasks, performance will not differ for both stimulus types. An interaction of negative emotion and cognitive control is associated with reduced neural activity in the cognitive control network and increased activity in emotion processing in PP (Bertocci et al., 2012; Brotman et al., 2014; Comte et al., 2017; Corbalan et al., 2015; Dichter et al., 2010; Favre et al., 2015; Kim et al., 2015; Mukherjee et al., 2016; Pauly et al., 2008; Pavuluri et al., 2008; Rey et al., 2014). Behavioral results also extend this effect to CHR-P and non-clinical individuals with psychotic-like experiences (Mohanty et al., 2005; Besnier et al., 2009; Yaffe and Walder, 2016; van Strien and van Kampen, 2009). Reduced lateral PFC and increased amygdala activity has been observed in CHR-P individuals in an emotional Stroop task (Mohanty et al., 2005). Similarly, decreased functional connectivity between the PFC and amygdala was reported in an emotion regulation task in healthy individuals who are highly prone to psychotic symptoms (Modinos et al., 2010). These neuroimaging data may suggest failure of attentional control of emotional processing regions when negative emotional stimuli are used in cognitive control tasks in psychosis.

On the other hand, only a small number of studies investigated the impact of positive emotion on cognitive control and the results are inconsistent/inconclusive (Table 1). Studies that examined the influence of both positive and negative emotions on cognitive control either showed a selective attentional bias towards negatively valenced stimuli (Becerril and Barch, 2011; Brotman et al., 2014; Favre et al., 2013; Pavuluri et al., 2008; Vercammen et al., 2012) or no difference between negative and positive stimuli (Bertocci et al., 2012; Comte et al., 2017; Eack et al., 2016; Park et al., 2018; Rey et al., 2014). A failure to observe a positive attentional bias may be associated with the inability to process reward-related cues due to increased levels of striatal dopamine in schizophrenia (Juckel et al., 2006). This is supported by altered basal ganglia activity in n-back tasks with positive words in PP (Eack et al., 2016; Juckel et al., 2006). In summary, PP seem to be over-sensitive to negative emotion and under-sensitive to positive emotional stimuli, which may be related to the severity of positive and negative symptoms, respectively.

3.2. Medication effects

Although antipsychotic medication has been reported to affect both cognitive control and emotion processing separately, its impact on tasks probing the interaction between emotion and cognitive control remains unclear. Studies investigating the effects of antipsychotics on emotion and cognitive control systems produced mixed findings (Cohen and Servan-Schreiber, 1992; Del-Ben et al., 2005; Harmer et al., 2006; Meltzer and McGurk, 1999; Pinkham et al., 2007; Sharma, 1999; Weinberger et al., 1988; Weiss et al., 2002; Weiss et al., 2003). Effects of conventional antipsychotics acting on dopamine receptors (e.g., haloperidol) and atypical antipsychotics acting on both dopamine and serotonin receptors (e.g., risperidone) are distinguishable in schizophrenia and bipolar disorder with psychosis (Gaebel and Wölwer, 1992; Williams et al., 2003). While typical antipsychotic medication tends to show no favorable effect on emotion processing, atypical antipsychotics can lead to improvement in emotion perception and cognitive control that remain significant even after controlling for the effects of positive symptoms (Kee et al., 1998; Williams et al., 2003). However, other studies could not replicate such beneficial effects of atypical antipsychotics on emotion processing and cognitive control, even though symptoms had been remitted (Herbener et al., 2005). Atypical antipsychotics have also been shown to alter the function of the amygdala and prefrontal cortex in the context of tasks tapping into emotion-cognitive control interaction (Cohen and Servan-Schreiber, 1992; Del-Ben et al., 2005; Harmer et al., 2006; Meltzer and McGurk, 1999; Pinkham et al., 2007; Sharma, 1999; Takahashi et al., 2005; Weinberger et al., 1988; Weiss et al., 2002; Weiss et al., 2003). However, again results are mixed. Some studies show a reduction whereas others show no effect of atypical antipsychotics on amygdala activity (Del-Ben et al., 2005; Harmer et al., 2006; Takahashi et al., 2005).

Likewise, several other pharmacological agents (e.g., selective serotonin reuptake inhibitors, β-adrenergic blockers; dopamine D2 receptor antagonists; anxiolytics, benzodiazepines; agonists and antagonists at the $GABA_A$ receptor) were also shown to modulate the amygdala and prefrontal activation in schizophrenia (Del-Ben et al., 2005; Harmer et al., 2006; Lewis and Gonzalez-Burgos, 2006; Paulus et al., 2005; Takahashi et al., 2005; van Stegeren et al., 2007). However, the results have not been replicated (Pinkham et al., 2007). Although the pharmacological treatment of schizophrenia involves atypical or typical antipsychotics or a combination of both, the treatment of bipolar disorder tends to rely on a combination of different types of psychotropic medications such as antipsychotics, anticonvulsants, antidepressants, anxiolytics, and mood stabilizers (Table 2). As these drugs have differential effects on distinct neurotransmitter systems and brain regions, the effects of medication on behavior or neural systems in the context of emotion-cognitive control interaction remain unclear. Similarly, the studies included in the current literature review do not allow disentangling the effects of distinct antipsychotics on the interaction of emotion on cognitive control in psychosis as the evidence is mixed. However, there is some indication that antipsychotics may normalize but perhaps not completely eradicate psychosis-related changes in emotion-cognitive control interaction. This tentative conclusion is supported by studies with non-clinical individuals with psychotic-like experiences. Alterations in emotion processing, cognitive control, and their interaction have been observed in CHR-P and non-clinical individuals with psychotic-like experiences (Bourque et al., 2017; Modinos et al., 2010; Mohanty et al., 2005, 2008; Pauly et al., 2010; van't Wout et al., 2004).

As alterations of emotion-cognitive control interaction are seen in both CHR-P and non-medicated psychosis-prone individuals, preceding the clinical diagnosis of psychosis and administration of antipsychotics, it is unlikely that the observed changes are entirely caused by medication. Future research should focus on longitudinal investigations of the psychosis continuum to explore whether and how medication affects the interactions between emotion and cognitive control in PP.

3.3. Age at illness-onset

Recent studies have suggested that the functional impact of psychosis is associated with age at illness-onset (Gogtay et al., 2011; Girard and Simard, 2008; Howard et al., 2000; Mason et al., 2013). Although both early- and late-onset psychosis share the same core psychopathophysiology, symptoms tend to be more severe in early-onset psychosis (Ju et al., 2019; Vahia et al., 2010). Accordingly, early-onset psychosis requires a higher daily dosage of antipsychotic medication (Vahia et al., 2010). Early onset of psychosis refers to experiencing psychotic

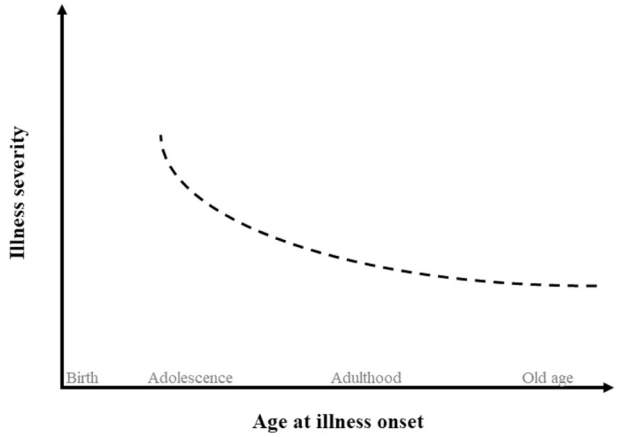

Fig. 3. Illness chronicity and age at illness onset. Psychotic symptoms (and prognosis) are most severe if the first-episode of psychosis occurs in early adolescence.

symptoms either before (childhood-onset psychosis) or during the critical phase of adolescence, which is characterized by major morphological and functional changes in the brain and represent increased vulnerability to different types of psychopathology (Gogtay et al., 2011). This is particularly relevant for emotion-cognitive control interaction as structural and functional maturation of brain regions implicated in executive function and cognitive control (e.g., lateral PFC, inferior parietal lobe) occurs at the end of adolescence (Biffin et al., 2009; Frangou, 2010; Gogtay et al., 2011). This suggests that if the onset of psychosis occurs before or during adolescence, it will likely disrupt the normal maturation process in these brain regions, affecting associated cognitive functions such as executive and cognitive control (Biffin et al., 2009; Feinberg, 1982; Frangou, 2010; Gogtay et al., 2004; Gogtay et al., 2011; Vidal et al., 2006). On the other hand, studies show inconsistent findings with respect to emotion processing. While some studies reported reduced gray matter (GM) volume in insular cortex, hippocampus, and amygdala in CHR-P individuals compared to healthy controls (Velakoulis et al., 2006), others reported no significant differences (Gogtay et al., 2011). Overall, a reciprocal relationship is observed between age at illness-onset and severity of illness (Fig. 3).

We did not identify any effect of age at illness-onset on emotion-cognitive control interaction in the current literature (Table 2). However, extrapolating the discussed findings to the issue of how emotion and cognitive control interact, we would expect distinct changes in the dorsal cognitive control network, which, in turn, should have consequences for emotion-cognitive control interaction. Future longitudinal studies are required to specify how emotion and cognitive control mechanisms interact along the developmental trajectories of age at illness onset.

3.4. Illness chronicity

Progressive decline in behavioral performance in tasks exploring emotion-cognitive control interaction has been observed along the psychosis continuum, from non-clinical individuals with psychotic-like experiences to patients (Table 2). Whereas no significant behavioral differences were observed (e.g., ratings of emotional stimuli, affect downregulation, reaction time, errors) between healthy controls and non-clinical individuals with psychotic-like experiences (Modinos et al., 2010; Mohanty et al., 2005; Pauly et al., 2010; van der Velde et al., 2015), task performance was found to be significantly reduced in PP compared to healthy controls (Anticevic et al., 2011; Anticevic et al., 2012; Becerril and Barch, 2011; Comte et al., 2017; Diaz et al., 2011; Dichter et al., 2010; Mukherjee et al., 2016; Pauly et al., 2008; Tully et al., 2014; Vercammen et al., 2012). This is in line with several cross-sectional studies that compared CHR-P, first-episode psychosis, and chronic schizophrenia patients, where progressive deterioration in cognitive functions such as attention and executive function was observed post illness-onset in patients compared to healthy controls (Fusar-Poli et al., 2012; Mesholam-Gately et al., 2009; Pino et al., 2014). Similarly, alterations in emotion processing were observed along the psychosis continuum from CHR-P individuals (Addington et al., 2008; Amminger et al., 2012; Dickson et al., 2014; Kohler et al., 2014; Roddy et al., 2012; van Rijn et al., 2011; Wolwer et al., 2012) to chronic schizophrenia (Hooker et al., 2011; Pinheiro et al., 2013; Pinheiro et al., 2014; Pinkham et al., 2011; Thaler et al., 2013) in several cross-sectional studies. However, longitudinal studies revealed that alterations in cognitive function were not progressive after illness onset (Keefe et al., 2006; Reichenberg et al., 2010), which may be a consequence of medication.

Progressive neurodegeneration leads to changes in gray matter and cortical thickness in regions relevant for the interaction of emotion and cognitive control in CHR-P, first-episode psychosis, and chronic schizophrenia patients (Feinberg, 1982; Gogtay et al., 2004; Vidal et al., 2006). These regions include prefrontal cortices, hippocampus, amygdala, basal ganglia, and thalamus (Pino et al., 2014). However, the interpretation of progressive functional and structural neurodegeneration with increased illness severity is limited by inconsistent results within PP. This may be due to symptoms' heterogeneity and medication effects. Most studies investigating schizophrenia and bipolar disorder with psychosis were conducted with patients on antipsychotic medication. As antipsychotics have been shown to profoundly impact both brain structure and function, it is difficult to determine whether progressive neurodegeneration is exclusively due to illness (Cahn et al., 2002; Cohen and Servan-Schreiber, 1992; Del-Ben et al., 2005; Harmer et al., 2006; Meltzer and McGurk, 1999; Pinkham et al., 2007; Pino et al., 2014; Sharma, 1999; Takahashi et al., 2005; Weinberger et al., 1988; Weiss et al., 2002; Weiss et al., 2003). However, recent studies reported structural (cortical thickness) and functional (connectivity in fronto-parietal and cingulate-opercular networks) changes also in non-clinical schizotypy (DeRosse et al., 2015; Nenadic et al., 2015; Wiebels et al., 2016 and first-episode unmedicated schizophrenia patients (Lin et al., 2019; Wang et al., 2019). This suggests that neurodegenerative processes occur at an early stage of illness and manifest irrespective of medication. These findings support the notion of progressive deterioration of the neural circuits underlying the interaction between emotion and cognitive control along the psychosis continuum.

4. Affect, emotion-cognitive control interaction and psychosis

Understanding the influence of emotion on cognitive control is crucial as emotion affects how cognitive control is regulated and the adaptive interaction between them leads to appropriate behavior in real-life situations (Pessoa, 2008). Likewise, the role of arousal, valence, self-regulation, motivation, and reward processing on emotion-cognitive control interaction should also be considered as these variables (see Table 1) may mediate the dynamic reciprocal interactions between the dorsal cognitive control and the ventral emotion processing systems in psychosis (Barch, 2005, 2008; Barch et al., 2008; Clamor et al., 2014; Gard et al., 2009; Mueller, 2011; Pessoa, 2008; Roseman and Smith, 2001; Roseman, 2008; Vanlessen et al., 2015, 2016).

The existing evidence suggests that an optimal arousal level is required for adaptive behavior (Clamor et al., 2014). Both hyperarousal and hypoarousal alter the way one perceives and interacts with environmental stimuli (Clamor et al., 2014; Freeman et al., 2002). Altered states of arousal can contribute to perceptual anomalies such as hearing of thoughts as voices or exacerbate paranoid or threat belief (Clamor et al., 2014). Increased levels of arousal can have a negative impact on emotion regulation (e.g., oversensitivity or increased responsiveness towards emotional stimuli) and, consequently, affect their interactions

with cognitive control mechanisms. Accordingly, high arousal levels may trigger psychotic symptoms in PP (Docherty et al., 2009; Myin-Germeys et al., 2003), CHR-P (Palmier-Claus et al., 2012; Trotman et al., 2014) and non-clinical individuals with psychotic-like experiences (Clamor et al., 2014). Exposure to stress leading to increased arousal was found to exacerbate psychotic symptoms in schizophrenia patients and in CHR-P individuals (Dinzeo et al., 2004, 2008; Docherty et al., 2009; Myin-Germeys et al., 2003; Palmier-Claus et al., 2012; Trotman et al., 2014), whereas hyperarousability was positively correlated to higher levels of psychotic-like experiences in non-clinical individuals (Clamor et al., 2014). Similarly, the ability to self-regulate goal-directed behavior amidst conflict and errors, referred to as self-regulation or effortful control, has been shown to influence both emotion processing and cognitive control (Hofmann et al., 2012; Rothbart and Ahadi, 1994; Kanske and Kotz, 2012). Studies have shown negative correlations between positive and negative symptoms and self-regulation measures in schizophrenia (Santosh et al., 2015). However, the reviewed literature did not take into account the effect of measures such as self-regulation on emotion-cognitive control interaction in psychosis (Table 2). As self-regulation is negatively affected by an increase in cognitive load, threat and reduced motivation (Hofmann et al., 2012; Li et al., 2018; Ward and Mann, 2000) and as psychosis is associated with amotivation and increased sensitivity towards negative emotional stimuli, we would expect self-regulatory abilities to influence the impact of emotion on cognitive control in psychosis. These studies indicate that personality traits such as hyperarousal (see Table 1) and diminished self-regulation may specifically affect the interaction of emotion perception and attention and suggest that these factors may represent an index of psychosis vulnerability.

As mentioned in Section 3.1, PP are not only oversensitive to negative emotional stimuli but they also assign salience to neutral stimuli, which may contribute to the formation of positive symptoms such as hallucinations or delusions. On the contrary, under-sensitivity to positive emotional stimuli has been associated with alterations in motivation and reward processing, which may result in negative symptoms such as social withdrawal, apathy, and anhedonia (Abbas et al., 2019; Barch, 2005, 2008; Barch et al., 2008; Carra et al., 2019; Gard et al., 2009; Kapur, 2003). Positive and negative symptoms tend to influence each other in a causative manner, where hallucinations can cause social withdrawal and lead to other negative symptoms and vice versa (Carra et al., 2019; Fowler et al., 2012; Messinger et al., 2011; Millan et al., 2014; Wickham et al., 2014).

Altered neurochemical mechanisms associated with the dopamine neurotransmitter system have been shown to influence cognitive control and emotion processing in psychosis and an imbalance in dopamine is associated with psychosis pathology (Breiter et al., 2001; Juckel et al., 2006; Kapur, 2004; Kapur et al., 2005; Ott and Nieder, 2019; McCutcheon et al., 2019). Dopamine is primarily associated with detecting motivational salience in the environment and using it to form associations in a goal-directed fashion (Berridge and Robinson, 1998; Braver and Cohen, 2000; Schultz, 2002). Under normal physiological circumstances, healthy individuals show a balance between dopamine release in the mid-brain regions and the ability to understand context and to assign salience to environmental stimuli (Kapur, 2004; Kapur et al., 2005). In psychosis, however, genetic or environmental predispositions lead to a dysregulation of dopamine release/firing (Kapur, 2004; Schulze et al., 2019). Dopamine dysregulation has been associated with the formation of delusions ("a way to advocate false beliefs") and abnormal perceptual experiences such as hallucinations ("anomalous salience of internal representations of percepts, memories", Kapur, 2004). As dopaminergic systems (e.g., nucleus accumbens and ventral tegmental area) are extensively connected to both PFC and amygdala, alterations in dopaminergic transmission not only affect the ability of the amygdala to assign salience to a stimulus but also the PFC's ability to guide goal-directed behavior (Braver and Cohen, 2000; Ott and Nieder, 2019; Rosenfeld et al., 2011). In the healthy population, increased dopamine firing is associated with successful cognitive control by the PFC, with activation of the striatum by positive affect (mood, motivation, and reward processes) and increased amygdala activation by negative stimuli (Ashby and Isen, 1999; Kienast et al., 2008; Kumakura et al., 2007; Ott and Nieder, 2019). However, antipsychotic medication blocks the effect of dopamine and, consequently, dampens the salience of preoccupying symptoms in psychosis. This may result in hyperactivity of the amygdala due to overprocessing of negative stimuli, in PFC hypoactivity due to failed attentional control, and altered basal ganglia activity resulting in low motivation and inadequate processing of reward/positive stimuli (Berridge and Robinson, 1998; Juckel et al., 2006; McCutcheon et al., 2019). As there is a long prodromal phase prior to the onset of clinically relevant psychotic symptoms (McGlashan et al., 2010), it is likely that dopamine dysregulation also characterizes the prodrome phase manifested as preclinical psychotic symptoms. This is supported by studies reporting salience misattribution in CHR-P individuals (Addington et al., 2008; Amminger et al., 2012; Dickson et al., 2014; Kohler et al., 2014; Roddy et al., 2012; van Rijn et al., 2011). As dopamine seems to be involved in all key processes underpinning the interaction of emotion and cognitive control at the brain level (PFC, amygdala and striatum), it is likely that alterations in the mesocorticolimbic dopamine pathways play a critical role in the dysfunctional emotion-cognitive control interaction in psychosis (Mueller, 2011; Xu et al., 2019). However, this hypothesis warrants further investigation.

5. Neural underpinnings of emotion-cognitive control interaction in psychosis

In line with the second aim of this review, this section focuses on the neurofunctional changes associated with the cognitive control and emotion processing systems in psychosis. Alterations in the dorsal cognitive control system and in the ventral emotion processing system have been repeatedly associated with alterations of emotion-cognitive control interaction in psychosis (Table 2; Alustiza et al., 2017; Lin et al., 2018; Minzenberg et al., 2009; Modinos et al., 2010; Modinos et al., 2015; Mohanty et al., 2005; Ruocco et al., 2014). However, it is unclear if such changes manifest in (i) the emotion processing neural network independent of cognitive control, (ii) the cognitive control system, or (iii) some intermediary neural circuitry leading to disruptions in emotion-cognitive control interaction along the psychosis continuum (Fig. 4).

5.1. Impaired emotion processing?

Alterations in emotion processing have long been recognized as a core feature of psychosis pathology. They include disturbances in emotion regulation, perception, recognition, and salience attribution in all sensory modalities (Hoekert et al., 2007; Kohler et al., 2010; Kring and Elis, 2013; Taylor et al., 2012; Thaler et al., 2013; Wynn et al., 2013; Wynn et al., 2008). Relative to healthy controls, both reduced (Comte et al., 2017; Diaz et al., 2011; Kim et al., 2015; Park et al., 2018; Townsend et al., 2013) and increased (Bertocci et al., 2012; Brotman et al., 2014; Eack et al., 2016; Favre et al., 2015; Passarotti et al., 2011; Pauly et al., 2008; Pavuluri et al., 2008; Corbalan et al., 2015) activity in emotion processing regions such as the amygdala, orbitofrontal cortex, insula, thalamus, and hippocampus have been observed in PP. The discrepant findings may be attributed to either task demands (such as the type of cognitive control tasks, implicit or explicit processing of emotional stimuli during these tasks) or individual differences (such as symptom severity or differences in personality traits, including anxiety and arousal).

When emotional stimuli are processed implicitly in cognitive control tasks, participants detect and perceive the emotion but are required to ignore/disengage from them the cognitive control task. As PP are (over-) sensitive to (negative) emotional stimuli as a function of

symptom severity, the detection and perception of these stimuli would result in either intact or increased neural activity in the amygdala. In this case, the cognitive control system would be required to exert greater "attentional control" to allow the participant to disengage from the emotional stimuli in order to deliver appropriate task performance. This is supported by neuroimaging studies, in which emotional stimuli were presented implicitly in cognitive control tasks in PP (Pauly et al., 2008; Dichter et al., 2010; Pavuluri et al., 2008; Favre et al., 2015). However, a few other studies reported decreased activity in the amygdala and in other emotion processing brain regions (Anticevic et al., 2011; Diaz et al., 2011; Kim et al., 2015). Decreased amygdala activity in PP may be a result of reduced motivation and increased negative symptoms. Indeed, PP presented higher negative symptom scores compared to positive symptoms in these studies (Anticevic et al., 2011; Diaz et al., 2011; Kim et al., 2015). This suggests that the ability to detect and perceive emotional stimuli at an early processing stage during the implicit presentation of emotional stimuli in emotion-cognitive control tasks depends on the severity of positive and negative symptoms in psychosis.

In the case of CHR-P individuals, the implicit presentation of emotional stimuli during a working memory task elicited increased activity in the frontal operculum (lateral PFC) in these participants as compared to healthy controls, in addition to increased activity in the thalamus and decreased activity in the basal ganglia and insula (Pauly et al., 2010). While an increase in PFC activity can be ascribed to facilitated performance, altered activity in the basal ganglia, insula, and thalamus can be ascribed to compensatory activity to maintain task performance in CHR-P individuals. Like PP, these individuals also scored high on negative symptoms. Non-clinical individuals with psychotic-like experiences also showed increased activity in the amygdala and basal ganglia, as well as decreased activity in the PFC in an emotional Stroop task (Mohanty et al., 2005). Although task performance was not significantly different from healthy controls, it significantly correlated with "anxiety apprehension and sensitivity" (Mohanty et al., 2005). This indicates that although they may be sensitive to emotional stimuli, they recruit additional brain regions to maintain task performance.

Reduced activation in emotion processing regions (e.g., amygdala, hippocampus, insula) was observed in PP when emotion was task-relevant and required explicit attention during cognitive control tasks (Comte et al., 2017; Park et al., 2018; Townsend et al., 2013). For example, during the variable attention and congruency task, emotional pictures of same or opposite valence were overlaid with each other and participants had to determine the emotion of the target or the background picture (Comte et al., 2017; see explicit tasks in Fig. 2). Similarly, during a modified version of the Simon task, positive and negative emotional pictures were serially presented on the left or right side of the screen and participants were asked to indicate the emotion of the picture via a button press (Park et al., 2018; see explicit tasks in Fig. 2). In these tasks, attention is either divided between two competing emotional pictures of opposite valence at the same time point or switched between these emotions continuously during the course of the task, which may overburden the emotion processing system resulting in reduced activation. Task performance (reaction time) was also significantly impaired in PP as compared to healthy individuals in these tasks (Comte et al., 2017; Park et al., 2018). However, reduced activity in emotion processing regions during the explicit presentation of emotional stimuli was not replicated (Brotman et al., 2014; Corbalan et al., 2015; Favre et al., 2015). We did not observe any such pattern in studies with non-clinical populations (Table 2).

Overall, changes in emotion processing regions can be ascribed to either (i) over-sensitivity towards negative emotional stimuli or (ii) severity of positive symptoms. However, most of the studies discussed above reported decreased activity or altered connectivity of the PFC with the amygdala and associated these changes with impaired task performance in psychosis. An open and relevant question therefore is whether impaired emotion processing occurs independent of cognitive control in psychosis.

5.2. Dysfunctional context-sensitive attentional control?

Successful conflict resolution requires the efficient use of attentional control on emotion regulation, attribution of salience to relevant stimuli, and inhibition of distracting emotional cues to maintain task performance. This is reflected in increased activation in the frontal-cingulate-parietal (cognitive control) network comprising the anterior cingulate cortex, lateral PFC and inferior parietal gyrus and intact activation in emotion processing regions such as the amygdala (Alustiza et al., 2017; Egner et al., 2008; Minzenberg et al., 2009). In PP, hypoactivation in the cognitive control network is observed regardless of the task-relevance of emotional stimuli and is often paired with altered activity in emotion processing regions such as the amygdala, hippocampus, and insula (Table 2). Similarly, CHR-P individuals and non-

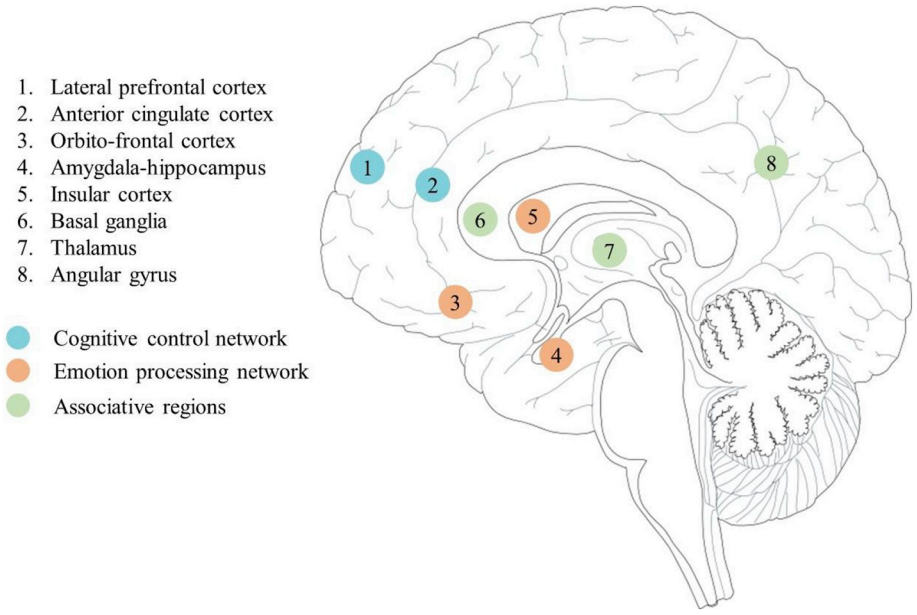

Fig. 4. Cortical and subcortical brain regions involved with emotion-cognitive control interactions (adapted from Kotz et al., 2018).

clinical individuals with psychotic-like symptoms show hyperactive amygdala and hypoactive prefrontal regions (Modinos et al., 2010; Mohanty et al., 2005; Pauly et al., 2010). Further, decreased connectivity between the cognitive control and the emotion processing networks has been observed along the psychosis continuum (Park et al., 2018; Modinos et al., 2010). These findings suggest that alterations in attentional control (hypoactive PFC) result in emotion dysregulation (hyperactive amygdala) in emotion-cognitive control tasks. As both emotion regulation and orienting/inhibiting attention require an intact prefrontal attentional control system, alterations in the emotion processing network engaged in tasks probing emotion-cognitive control interaction, may not be independent of cognitive control dysfunction in PP.

Another aspect of attentional control in cognitive control tasks is the ability to comprehend and actively maintain instructions required to execute a task (Cohen and Servan-Schreiber, 1992). Studies with PP have consistently documented difficulties in interpreting lexical ambiguities due to an inability to understand the context of a situation in language tasks (Baez et al., 2013; Cohen and Servan-Schreiber, 1992; Salzinger, 1971). PP also show difficulties in constructing and maintaining internal representations of context during information-processing and attention-related tasks (Park et al., 2011; Rizzo et al., 1996; Schenkel et al., 2005; Servan-Schreiber et al., 1996; Uhlhaas et al., 2006; Waters et al., 2004). The concept of *"internal representation of context"* refers to the process of maintaining either task-instructions (e.g., during Flanker/Stroop tasks, in which participants have to respond to one aspect and ignore other aspects of the stimulus) or a particular stimulus (e.g., during n-back tasks, in which participants need to remember and update each stimulus so as to match it with the -nth preceding stimulus), or processing stimulus sequences (e.g., during language-related tasks, in which one needs to interpret the meaning by processing a sequence of words) (Cohen and Servan-Schreiber, 1992). A failure to understand context leads to the selection of inappropriate responses in the respective task. Alternatively, PP may understand the context but either fail to maintain it over longer periods of time or fail to integrate the contextual information in the presence of emotional cues/distractors. Several frontal brain regions such as the medial PFC, ventro-medial PFC, inferior frontal, precentral gyrus, and orbito-frontal cortex, associated with difficulties in understanding, maintaining, and using an internal representation of context in emotion and non-emotion tasks in PP, are also a part of an interaction between emotion-cognitive control (Hoenig and Scheef, 2009; Kircher et al., 2007; Rapp et al., 2004; Smith et al., 2004). Impairments in contextual processing are subtler in psychosis-prone non-clinical individuals. Task interference by emotional stimuli in emotional Stroop or n-back working memory tasks in CHR-P and psychosis-prone non-clinical individuals is higher than in healthy controls, even though not significantly different. These individuals often show an enhanced processing effort by recruiting additional brain regions (e.g., thalamus, basal ganglia, angular gyrus) in order to maintain task performance (Ettinger et al., 2014; Mohanty et al., 2005; Pauly et al., 2010).

Furthermore, neurochemical changes involving the PFC also support a dysfunctional attentional control hypothesis and the impact of emotion on cognitive control. As mentioned in Section 4, alterations in midbrain dopamine projections to several cortical and subcortical regions have been repeatedly associated with psychotic symptoms (McCutcheon et al., 2019). In addition, increased dopamine levels in the PFC and anterior cingulate cortex are thought to promote successful cognitive control and improve cognitive flexibility (Ashby and Isen, 1999; Braver and Cohen, 2000; Ott and Nieder, 2019). This suggests that a dopaminergic imbalance in the PFC could, in turn, down-regulate attentional control. With respect to emotion-cognitive control interaction, altered prefrontal activity could therefore impair context-sensitive attentional processing and the ability to assess the emotional significance of a stimulus.

More recent meta-analyses of fMRI studies, investigating various aspects of cognitive control (e.g., attentional control, inhibition, sustained and selective attention, working memory), also showed reduced activation in this network in PP irrespective of the presence of emotional stimuli (Table 2; Alustiza et al., 2017; Mueller, 2011). Reductions in cortical thickness in the PFC and cingulate cortex have also been observed in CHR-P (Gisselgard et al., 2018). The observation of functional and structural changes, particularly in prefrontal regions, indicates high replicability of cognitive control impairments in psychosis. Overall, the presence of impairment in the cognitive control network in tasks with or without emotional stimuli along the psychosis continuum indicates that dysfunctional context-sensitive attentional control may be inherent to psychosis pathology. Changes in cognitive control may reflect an increased vulnerability to psychosis and could thus serve as neuroimaging biomarkers of psychosis vulnerability (Falkenberg et al., 2015; Gisselgard et al., 2018).

5.3. Alterations in the functioning of intermediary associative regions

When emotion interacts with cognitive control, altered patterns of activation and connectivity within cognitive control and emotion processing networks have been observed in intermediary cortical and subcortical brain regions, such as the thalamus, basal ganglia, and angular gyrus. These regions are implicated in the integration of converging information in both clinical and non-clinical individuals with psychotic-like experiences (Bertocci et al., 2012; Brotman et al., 2014; Pauly et al., 2008; Takahashi et al., 2004). In the following paragraphs, we will discuss the role of these regions with respect to emotion-cognitive control interaction along the psychosis continuum.

The thalamus acts as a filter and integrates information from different sensory systems and relays it to higher cortical regions involved in emotional and cognitive processes (Pauly et al., 2008; Takahashi et al., 2004). Activity in this region increases with increased cognitive load, which may indicate greater effort and efficiency required to orchestrate increased amounts of sensory information (Pauly et al., 2008). Disruption in thalamic activity may result in the discoordination of the transmission of information between emotion processing and cognitive control regions. Neuroimaging studies have shown not only reduced functional activity but also decreased thalamic volume in schizophrenia patients (Kemether et al., 2003; Pinault, 2011). Structural and functional changes in the thalamus have been associated with attention and emotion impairments but also with positive symptoms in schizophrenia and bipolar disorder (Caetano et al., 2001; Chen et al., 2019; Gilbert et al., 2001; John et al., 2018; Gong et al., 2019; Pergola et al., 2015; Yamamoto et al., 2018). These findings suggest that the ability of the thalamus to process sensory input may be reduced and lead to impaired perceptual processing particularly in situations of increased arousal, ultimately resulting in anomalous perceptual experiences such as hallucinations in schizophrenia (Behrendt, 2006; Pinault, 2011). Whereas reduced thalamus activity was observed in schizophrenia when emotion interacts with cognitive control, increased bilateral thalamus activity was observed in CHR-P individuals during an n-back working memory task with emotional stimuli (Pauly et al., 2008, 2010). Similarly, a positive correlation was observed between thalamus activity and psychosis-prone personality factors in healthy individuals (Ettinger et al., 2013). The increase in thalamus activity in the prodromal phase of psychosis, which stands in contrast to the reduced activity in schizophrenia, points towards a compensatory role of thalamus to maintain task performance (Ettinger et al., 2014, 2015). These data support the concept of a psychosis continuum (Diederen et al., 2012; Ettinger et al., 2014, 2015).

The basal ganglia have close associations with sensorimotor, associative/cognitive, and limbic systems, and alterations in this region could be associated with psychiatric disorders such as psychosis (Macpherson and Hikida, 2019). This region has been implicated in tasks involving broadening the scope of thoughts and actions leading to positive experiences and exploratory behavior on the one hand, and

developing strategies to deal with threat during aversive situations on the other hand (Fredrickson, 2001; Wager et al., 2003). Aberrant activity in sub-parts of the basal ganglia (e.g., striatum, putamen, caudate nucleus) has been reported in psychosis in tasks testing emotion-cognitive control interaction (Bertocci et al., 2012; Brotman et al., 2014; Mohanty et al., 2008; Pauly et al., 2010). Relative to healthy controls, decreased neural activity during negative and increased neural activity in positive emotion-cognitive control tasks were reported in the striatum and PFC in an emotional n-back task in schizophrenia (Eack et al., 2016). Diminished striatal function in schizophrenia was proposed to reflect fronto-limbic disengagement so as to process fearful stimuli in moderation to maintain working memory task performance (Eack et al., 2016). On the other hand, increased functionality of the striatum and PFC during positive emotion-cognitive control interaction correlated with longer reaction times, suggesting effortful regulation of positive emotion (Eack et al., 2016). Reduced basal ganglia activity was also observed in CHR-P individuals whereas increased activity was seen in healthy individuals with high psychosis proneness in negative emotion-cognitive control interaction (Pauly et al., 2010; Mohanty et al., 2005). As task performance was not significantly different from healthy controls, alteration in basal ganglia activity can be attributed to compensatory emotion regulation to maintain task performance.

Further, cortical regions such as the angular gyrus, precuneus, and posterior cingulate have shown structural abnormalities, in addition to aberrant activity in emotion-cognitive control tasks in psychosis (Nierenberg et al., 2005; Niznikiewicz et al., 2000; Pauly et al., 2008; Thompson et al., 2001). Due to their location at the junction of temporal, parietal, and occipital cortex, these regions play a significant role in the integration of incoming information and channeling this information to frontal-cingulate regions (Pauly et al., 2008). Reduced activity in these regions in negative emotion-cognitive control tasks may also be ascribed to compensatory emotion regulation to maintain task performance. We did not find any studies that looked into these brain regions in non-clinical individuals with psychotic-like experiences in emotion-cognitive control tasks. Altered communication between these intermediary and associative brain regions may result in disruption of emotion-cognitive control coupling. As most studies focused on lateral PFC, anterior cingulate cortex and amygdala, the role of these intermediary regions in emotion-cognitive control interactions along the psychosis continuum warrants further investigation.

6. Summary and future directions

Based on the reviewed studies, factors affecting the interaction between emotion and cognitive control in psychosis include the valence of emotional stimuli, severity of positive and negative symptoms, and individual differences (e.g., arousal). The early stages of psychosis experience may not be distinguishable from healthy individuals; however, at later stages (CHR-P, first-episode schizophrenia, or chronic schizophrenia) a significant decline in emotion-cognitive control interaction is observed regarding neural activation and task performance (Table 2). This decline can be ascribed to an altered interplay between emotion processing and context-sensitive attentional control mediated by intermediary associative regions (thalamus, basal ganglia, and angular gyrus). As a true course of this decline along the postulated psychosis continuum may be obscured by differences in methodological aspects, heterogeneity and severity of symptoms, individual differences in personality traits and medication, future efforts to understand the emotion-cognitive control interaction in psychosis should focus on longitudinal follow-up studies, taking into account these variables and using the same tasks and paradigms.

Further, impairments reflecting the impact of emotion on cognitive control have been described in many other neurological disorders and mental illnesses such as major depressive disorder, autism, borderline personality disorder, anxiety disorder, post-traumatic stress disorder, obsessive-compulsive disorder, and even Alzheimer's disease (Liao et al., 2012; Loveland et al., 2008; Mayberg, 1997; Minzenberg et al., 2007; Trzepacz et al., 2013). Similarities and differences in these impairments may be associated with subtle differences in illness-specific symptoms or concerns. Comparable to PP, patients suffering from major depression, post-traumatic stress disorder, borderline personality disorder, and anxiety disorders show a negative attentional bias in emotion-cognitive control tasks (aan het Rot et al., 2012; Minzenberg et al., 2007; Nica and Links, 2009; Santangelo et al., 2014). However, this bias is associated with specific illness-related negative emotional stimuli and not with general negative emotions (Joyal et al., 2019). For example, stimuli related to war and abuse caused greater performance impairment (slower responses) in post-traumatic stress disorder, whereas major depressive disorder patients were more affected by stimuli showcasing sadness and discouragement in an emotional Stroop task (Joyal et al., 2019). Similarly, illness-specific abnormalities were observed in neural activations of emotion processing and cognitive control systems in these disorders (Schulze et al., 2019). Borderline personality disorder and post-traumatic stress disorder patients showed hyperactivity in amygdala-hippocampus regions as compared to major depressive disorder during the processing of negative emotional stimuli (Schulze et al., 2019). This is similar to PP with increased positive symptoms (e.g., hallucinations, Pauly et al., 2008; Dichter et al., 2010; Pavuluri et al., 2008; Favre et al., 2015), who showed hyperactive amygdala as compared to PP with increased negative symptoms (e.g., apathy, anhedonia, Anticevic et al., 2011; Diaz et al., 2011; Kim et al., 2015). Diagnosis-specific differences were also observed in lateral PFC regions where both patients with borderline personality disorder and post-traumatic personality disorder showed hyperactive ventrolateral PFC, reflecting enhanced regulation of negatively affected emotion. However, reductions in dorsolateral PFC specifically in borderline personality disorder may be due to increased impulsivity and difficulties in attentional control of emotion (Joyal et al., 2019; Schulze et al., 2019). Taken together, while these studies highlight the significance of diagnosis-specific effects on the attentional control of emotion, relevant questions such as whether and to what extent different types of negative emotions recruit distinct neural systems in different disorders remain an open issue for future investigations. Future research should therefore dissociate the dysfunctions of emotion-cognitive control interaction considering differences in illness-specific etiology.

7. Conclusion

The current review discussed and integrated fMRI evidence examining the influence of emotion on cognitive control along the psychosis continuum to identify alterations in emotion-cognitive control interaction and its connection to psychopathology. Specific processes and corresponding neuroanatomical correlates such as diminishing context-sensitive attentional control of prefrontal regions on the emotion processing network, and structural and functional alterations in subcortical and cortical associative brain regions have been identified as underlying changes affecting emotion-cognitive control interaction. However, open questions concerning the valence-specificity of these brain regions and the relationship between them remain to be further specified. A comprehensive understanding of the neural mechanisms underlying emotion-cognitive control interaction across the psychosis continuum is critical to achieve finer insights into the psychopathology of the illness. This would further help develop therapies and tools focusing on improving attentional control and ultimately improving socio-cognitive functioning of people with psychosis in their everyday life.

Declaration of competing interest

Authors report no conflict of interest.

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