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*A neuro(psychological) approach for emotion recognition deficits in depression.**

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Un enfoque neuro(psicológico) de los déficits de reconocimiento de emociones en la depresión.

Uma abordagem neuro(psicológica) dos défices de reconhecimento de emoções na depressão.

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ABSTRACT

Recognition of emotional facial expressions is an important skill for social functioning. Deficits in this ability have been linked to poor social adjustment and symptomatic relapse in patients with Major Depression Disorder (MDD). This paper reviews current neuropsychological studies of emotion recognition deficits in depression, highlighting its neuropsychological correlates, and provides an overview of commonly used measures to assess emotion recognition with MDD patients. We support an integrative neuropsychological approach which accounts for the interaction between emotion and cognition for emotion and cognition, to understand the emotion recognition deficits in depression and discuss its implication for clinical practice and research.

Keywords: Emotion; Recognition; Facial expressions; Depression; Integrative neuropsychology.

RESUMEN

El reconocimiento de expresiones faciales emocionales es una habilidad importante para el funcionamiento social. Los déficits en esta capacidad se han relacionado con una mala adaptación social y la recaída sintomática en pacientes con Trastorno Depresivo Mayor (TDM). Este artículo revisa los estudios neuropsicológicos actuales sobre los déficits de reconocimiento de emociones en la depresión, destacando sus correlatos neuropsicológicos, y proporciona una visión general de las medidas utilizadas con frecuencia en el reconocimiento de emociones para evaluar a los pacientes con TDM. Apoyamos un enfoque neuropsicológico integrador de la emoción y la cognición, para comprender los déficits de reconocimiento de emociones en la depresión y discutimos sus implicaciones para la práctica clínica y la investigación.

Palabras clave: Emoción; Reconocimiento; Expresiones faciales; Depresión, Neuropsicología integradora.

RESUMO

O reconhecimento de expressões faciais de emoções é uma habilidade importante para o funcionamento social. Pacientes com depressão parecem apresentar déficits nesta aptidão, o que contribui para um pobre ajustamento social e recorrente relapso sintomatológico. Neste artigo revisam-se os estudos atuais de cariz neuropsicológico sobre os déficits de reconhecimento de emoções na depressão, destacamos os correlatos neuropsicológicos e apresentamos uma panorâmica das medidas e instrumentos usados com frequência nos estudos de reconhecimento de emoções com pacientes com TDM. Apoiamos uma abordagem neuropsicológica integrativa da emoção e cognição, para compreender os déficits de reconhecimento de emoções na depressão e discutimos suas implicações para a prática clínica e investigação.

Palavras-chave: Emoção; Reconhecimento; Expressões faciais; Depressão; Neuropsicologia integrativa.

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Emotion recognition is an important skill for social functioning. It allows us to provide a feedback accordingly to others in social interactions, using a variety of channels, including facial expressions (Matsumoto, Keltner, Shiota, O'sullivan & Frank, 2008), voice (Bachorowski & Owren, 2008), and touch (Bolognini, Rossetti, Convento, & Vallar, 2013). However, in some psychiatric disorders such as Major Depression, the ability to accurately decode emotional cues from faces appears to be impaired. These deficits carry important implications for clinical practice, considering that they may account for poor social adjustment, persistence of symptoms, recurring symptomatic relapse and suicidal behavior (Phillips, Drevets, Rauch, Lane, 2003b; Stuhmann, Suslow & Dannlowski, 2011).

Whereas for disorders such as Schizophrenia and Autism Spectrum, the research on emotion recognition deficits is relatively well established, for depression, however, this is still an ongoing debate. Nevertheless, the need to unravel and mitigate risk factors with updated research and alternative interventions remains mandatory. Especially considering that the projections suggest that Depression will become the second leading debilitating health problem in the world by 2020 (WHO, 2001). Thus, the increasing comprehension of vulnerability factors, biological markers, and maintenance factors might contribute significantly for approaching interventions more efficiently (Stuhmann et al., 2011).

Considering the existence of well-established recent systematic reviews conducted on the topic (see. Bourke, Douglas, & Porter, 2010; Castanho, Rocca, Heuvel, Van Den, Caetano, & Lafer, 2009; Delle-Vigne, Wang, Kornreich, Verbanck, & Campanella, 2014; Kohler, Hoffman, Eastman, Healey, & Moberg, 2011; Stuhmann et al., 2011) the purpose of the current literature review is to provide a broader descriptive and integrative argument for emotion recognition deficits in depression. In this sense, we reviewed current neuropsychological studies of

recognition of emotional facial expressions in Depression available by November 2015 through the following databases: EBSCO, Science Direct, Psychology Annual Review and Springer. Additional bibliographical sources identified by hand-searches, such as books series devoted specifically to the subject of emotion recognition and neuropsychology were also considered.

We revisit the framework of facial expression and emotion research, summarize the key aspects of the studies with depressed patients and highlight its neuropsychological correlates and provide an overview of measures employed in these studies. We support an integrative neuropsychological approach which accounts for the interaction between emotion and cognition, to understand the emotion recognition deficits in depression and further discuss its implications for clinical practice and research.

1. Emotion recognition and facial expressions

Almost a century ago, Darwin (1872/1965) pointed the conceptual importance of emotions, highlighting its evolutionary and adaptive meaning for species (Darwin, 1965; Ekman, 1973, 1998). Later, in cross-cultural studies, Ekman found evidence for constants of six basic emotions, namely happiness, sadness, anger, disgust, surprise and fear (Ekman, Friesen, Wallace., & Ancoli, 1980; Ekman, 1998, 1999b; Matsumoto et al., 2008).

Understating facial expressions of emotion serves a social utility, as it allows people to make sense of the affective information conveyed in interpersonal interactions and provide a feedback accordingly (Fischer & Manstead, 2008; Matsumoto et al., 2008; Niedenthal & Brauer, 2012). An efficient emotion processing tends to be associated with better levels of health functioning and social performance (Hoffman, 2008; Niedenthal & Brauer, 2012). On the contrary, difficulties in this ability are often linked to the development and maintenance

of psychopathology and other health problems (Delle-Vigne, Wang, Kornreich, Verbanck & Campanella, 2014; Diefenbach et al., 2008; Gross, 2008).

In fact, there is a growing body of research reporting deficits on emotion recognition of facial expressions, in many clinical subgroups, such as schizophrenia (Kohler, Walke, Martin, Healey & Moberg, 2010), Autism Spectrum Disorders (Harms, Martin & Wallace, 2010), neurodegenerative disorders (Kumfor, Sapey-Triomphe, Leyton, Burrell, Hodges, Piguet, 2014) brain lesions (Adolphs, Damasio, Tranel, Cooper & Damasio, 2010) and depressive disorders (Kohler, Hoffman, Eastman, Healey & Moberg, 2010).

2. Emotion research in neuropsychology

Might be surprising to point, that some pioneers in neuroscience had initially a keen interest in understanding how emotion operates in the brain (LeDoux, 2000; LeDoux & Phelps, 2009; Rick & Loewestein, 2008). However, some argue that emotion research was a victim of the cognitive revolution that shifted the interest towards the emerging cognitive psychology hot topics, which were more easily, but mistakenly, understood in terms of computer processing metaphors (LeDoux, 2000; Niedenthal & Brauer, 2012; Phelps, 2006). Besides, brain lesion studies apparently had resolved the problem of the nature of emotions, with the appealing concept of the “limbic system” (LeDoux, 2000; LeDoux & Phelps, 2008). However, it is now known that the limbic system is not limited to, and neither sufficiently explanatory of emotions matters.

The ancestral debate over the suitability of emotion as a topic for cognitive neuroscience research, was perhaps what caused more harm to emotion research. The main question focused then on whether emotion precedes consciousness or the cognitive processing comes before the emotional processing (Panksepp, 2008; Phelps,

2006). Contrary to emotion researchers, cognitive scientists figured out a way to study mental processes without having to worry about the consciousness problem (LeDoux, 2006; LeDoux & Phelps, 2008). It was possible to study brain processing of external stimuli without first resolving how the conscious perceptual experiences come about. Even though, it is widely recognized that “cognitive processes occur unconsciously, with only the products reaching awareness, and then only sometimes” (Kihlstrom 1987 cited in LeDoux, 2006).

These problems that undermined emotion research for decades emerged in part from dichotomist views of cognition and emotion. Contrary to this extreme polarization, current research emerging from the revived interest of neuroscientists in emotions, shows that such distinction is unrealistic, and there is substantial evidence that both systems interact in many levels (Adolphs, Baron-Cohen & Tranel, 2002; LeDoux & Phelps, 2008; Panksepp, 2009; Phelps, 2006; Wager et al., 2008).

The current literature presents a general framework that demonstrates that through the influence of structures, such as the amygdala, in forebrain areas, emotions can affect attention and perception processes. These are the first stages of stimulus processing and encoding, facilitating awareness of emotionally salient stimuli in situations where attentional resources are limited (Kensinger & Schacter, 2008; LeDoux & Phelps, 2008; Phelps, 2006).

This framework suggests that when it comes to recognition of emotional facial expressions, it is both a perceptual and an emotional process, and each system can interfere with one another. Thus, the understanding of human cognition requires the consideration of human emotions and vice versa. Both areas could greatly benefit from an integrative and interactive approach (LeDoux, 2000; LeDoux & Phelps, 2008; Phelps, 2006; Panksepp, 2008).

3. Deficits of emotion recognition of facial expressions in depression

Studies on emotion recognition with MDD patients report a mood-congruent bias towards emotional faces. It seems that depressed patients are more sensitive to negative expressions and prone to rate emotions more negatively or to attribute emotional valence to neutral expressions (Bourke et al., 2010; Burt, Frigerio, Perrett & Young, 2006; Stuhmann et al., 2011). Few studies have suggested that this bias seems to persist even after recovering from symptoms contributing to social withdrawal, rejection feelings, and persistence of depressive symptoms, representing a risk factor for future depressive episodes (Joormann & Gotlib, 2007).

Auerbach, Stewart, Stanton, Mueller, and Pizzagalli (2015) reported that these deficits are also seen in depressed female adolescents, who demonstrated a dual emotion-processing bias towards sad and happy facial expressions. Lopez-Duran, Kuhlman, George, Kovacs (2013) examined perceptual sensitivity to sad cues in facial expressions among children at familial risk for depression and low-risk peers. They found that high-risk boys, but not girls, displayed enhanced perceptual sensitivity to sad facial expressions when compared to their low-risk peers. The authors speculated that these biases might be present in boys even before the onset of major depression, therefore, might be a mechanism of risk among male offspring of depressed parents.

One worrisome linkage that cannot be ignored in depression is suicidality. Some studies connected deficits in facial expression recognition to vulnerability factors for suicidal attempts. These studies found that suicide attempters make more errors in identification of emotional expressions (Maniglio, Gusciglio, Lofrese, Belvederi, Murri, Tamburello, & Innamorati, 2014; Richard-Devantoy, Guillaume, Olié, Courtet & Jollant, 2013). Hence, cognitive social skills training may be a target for prevention.

3.1. Neural basis of emotional recognition: can anyone tell where?

The processing of emotional stimulus such as facial expressions involves physiological arousal, appraisal, subjective experience, expression, and goal-directed behavior. Nonetheless, at present, there is no general accepted theoretical framework for human emotions (Clow & Ortony, 2008; Niedenthal & Brauer, 2012; Phillips et al., 2003a).

Some previous studies supported the hemispheric localization for emotional processing (Posamentier & Abdi, 2003) while other correlated deficits in facial emotion recognition to various somatosensory brain lesions including anterior supramarginal gyrus, insula and left frontal operculum (Adolphs et al., 2000; Adolphs et al., 1996). These results support the hypothesis that emotion recognition and emotional expression use the same system or at least partially uses the same routes (Niedenthal & Brauer, 2012).

Most neuroimaging studies suggest that critical structures for identification and generation of emotional facial expressions are subcortical and prefrontal, including but not limited to: amygdala, putamen, ventral striatum, globus pallidus, dorsomedial nuclei thalamus, hippocampus and hippocampal gyrus (Craig, 2008; Flaherty-Craig et al., 2002; Lawrence et al., 2004, Phelps, 2006; Wager, et al., 2008). The basal ganglia, insula, occipital-temporal lobes and orbitofrontal cortex are also part of this complex (Delle-Vigne et al., 2014).

Phillips et al. (2003a) summarize that the ventral areas are responsible for the identification of the valence of the emotions while the dorsal are responsible for the forced regulation of affective states and the subsequent behavior. Dal Monte et al. (2013) added that prefrontal cortex is mentioned to be central for regulating emotional responses while the left temporal cortex and the left inferior frontalis is necessary for the lexical semantic of the faces.

In sum, these studies suggest less support for the asymmetrical theory of emotions, evidencing a fronto-temporo-limbic circuitry for processing emotions (Atkinson & Adolphs, 2011; Dal Monte et al., 2013; Flaherty-Craig et al., 2002; Wager et al., 2008). Also, this data provides evidence for a more integrative approach to understanding facial emotion recognition and further integration of emotional and cognition aspects.

a) The emotional circuitry for processing facial expressions in depression

Research suggest that dysfunctions on left and right, frontal and posterior areas in the brain are linked to specific symptoms present in depression (Shenal, Harrison & Demaree, 2003). In a meta-analysis of studies that investigated neural correlations associated with processing emotional facial expressions in MDD patients, Bourke, et al. (2010) found that negativity bias was present, although the general data did not suggest specific deficits. Suslow, Kugel, Rufer, Redlich, Dohm, Grotegerd & Dannlowski (2015) showed that alexithymia acts as a modulator for automatic responses from facial expressions. Thus, alexithymic patients display less neural responses to facial expressions.

Lawrence et al. (2004) demonstrated that MDD patients display more activation in putamen even for moderate expressions of sadness, and low activation in response to happy facial expression compared to controls. This result supports the hypothesis of over-facilitation for negative facial expressions reported by behavioral studies.

Phillips et al., (2003) discussing the deficits in emotional processing and its implications for major psychiatric disorders suggested that the pattern of abnormalities in face processing are linked to specific symptoms and specific neuropsychological abnormalities. This review highlighted the cognitive deficits associated with depression specifically the executive function and the negativity bias. The authors documented structural and neuroanatomical functional abnormalities in regions

that are important for emotion, specifically a reciprocal influence of the ventral system, the dorsolateral prefrontal cortex (DLPFC) and probably the cingulate anterior pregenual gyrus and the ventrolateral prefrontal cortex (Phillips et al., 2003b) (See Table 1). Auerbach et al. (2015) also reported correlated DLPFC abnormalities for emotion recognition processing in depressed adolescents. These structures are known to be responsible for regulation of affective states, forced regulation of emotion and induction to a sad mood.

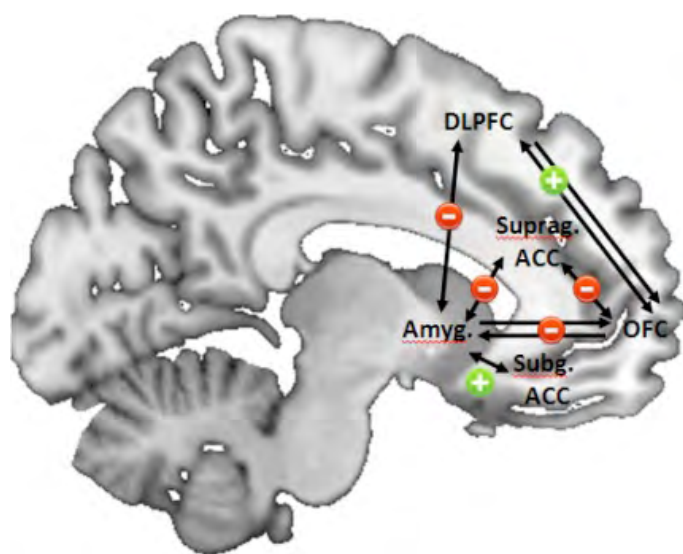


Figure 1. Schematic illustration of main results reported in fMRI connectivity studies on aberrant emotional face processing in Major depressive disorder (MDD) patients.

Double arrows represent results from functional connectivity approaches, whereas the normal arrows present results derived from effective connectivity analyses. Plus and minus characters indicate increased and decreased connectivity between brain regions in MDD. ACC = anterior cingulate cortex; Amyg = amygdala; DLPFC = dorsolateral prefrontal cortex; OFC = orbitofrontal cortex; suprag = supragenual; subg = subgenual. Reprinted with the authors permission: Stuhmann et al. (2011, p. 2).

Table 1. Structural and functional abnormalities in the ventral and dorsal neural systems important for emotion processing that might be associated with Major Depression

Identification of Emotional Significance	Production of Affective States and Behavior	Effortful Regulation of Affective States	Symptoms
Structural Reduced volume of the amygdala and ventral striatum. Functional Increased activity within the amygdala, anterior insula, ventral striatum, and thalamus to emotional stimuli and during a major depressive episode.	Structural Reduced volume within the subgenual anterior cingulate gyrus. Functional Increased activity in the subgenual anterior cingulate gyrus and the ventrolateral prefrontal cortex during a major depressive episode ¹ .	Structural Reduced volume within prefrontal cortical regions and hippocampus. Functional Decreased activity within dorsomedial and dorsolateral prefrontal cortices during a major depressive episode ¹ .	A restricted emotional range, but with a bias towards the perception of negative rather than positive emotions, resulting in depressed mood and anhedonia.

1. This pattern of activity reverses after recovery from a depressive episode, with increased activity within dorsomedial and dorsolateral prefrontal cortices, and decreases within the subgenual cingulate gyrus, hippocampus, thalamus, ventral striatum, and insula. Adapted from Philips et al. (2003b, p. 523).

The mood-congruent bias is also reported on studies monitoring neural activity in MDD patients, concluding that amygdala, insula, parahippocampal gyrus and orbitofrontal cortex are working abnormally. Stuhmaman, Suslow & Dannlowski (2011) summarized schematically the results of fMRI studies as presented in **figure 1**.

Consistently, the bias is seen echoed on cognitive event-related potentials studies. The waves associated with cognitive processing and attention exhibit larger amplitudes and latencies in MDD patients, during emotion recognition tasks. Especially the P1 (facilitation of processing for negative stimulus) P2 (orientation to salient stimuli), N2 (attention) N170 (perceptual processes for faces) and P300 (memory) (Delle-Vigne et al., 2014; He, Chai, Chen, Zhang, Xu, Zhu & Wang, 2012).

Klempman, Sequeira, Canetti, Lalovic, Ernst, French-Mullen *et al.* (2009) studied the GABAergic expression of subjects who committed suicide. Their findings suggest an overexpression of the BA46 gene in suicidal and depressive subjects. These genes are known to be involved in GABAergic transmission associated with

depression and relates to processing facial expressions. Thus, it was proposed that deficits in recognizing emotional facial expression may represent a biological marker of suicidality in Depression (Fu, Williams, Cleare, Scott, Mitterschiffthaler, Walsh & Murray, 2007; Klempman et al., 2009).

b) Emotion Recognition vs. Emotion regulation: integrating perspectives

Emotional recognition and emotional regulation tend to be discussed as discrete processes, but both interact. Understanding this interaction is crucial to understand the role of emotion recognition deficits in clinical problems such as depression.

The affective theory suggests that in normal conditions, an emotional stimulus is processed semi-automatically in three stages. First, occurs the identification of the emotional significance of an environmental stimulus (e.g. facial expression); second is the production of an affective state and emotional behavior induced by the stimulus; and

third, the regulation of the affective state and emotional response. This process is useful to generate contextually appropriate, complex affective states, emotional experiences (feelings), and behaviors (Diefenbach et al., 2008; Clore & Ortony, 2008; Gross, 2008; Philips et al., 2003a; Stuhmann et al., 2011). The problem emerges when the recognition or regulation pattern is biased or working abnormally, inducing inadequate emotional responses. Thus, when stimuli are consistently misjudged it generates a loop of negative emotional responses, such as those seen in depressive disorders. Philips (2003a) proposed such a model (see Fig. 2) to explain these three stages schematically, linking emotion regulation and emotion recognition.

This hypothesis has important implications for psychiatric disorders like depression, especially because it may represent one of its maintenance factors and a risk mechanism (Philips et al., 2003a, b; Stuhmann et al., 2011). Nonetheless, the current clinical focus on assessment and intervention tend to rely heavily on emotional regulation compared to emotion recognition. However, they should be viewed as reciprocal, and should both be considered as targets for research and clinical interventions.

3.2. The neuropsychological assessment of emotional recognition deficits in MDD

Almost all the primary neuropsychology textbooks and major taxonomies models for neuropsychological assessment focus mainly on neurocognitive functions. For example, the most cited neuropsychological batteries (Flaherty-Craig et al., 2002; Groth-Marnat, 2009; Harrison, 2002; Maia & Leite, 2009; Mitrushina, Boone, Razani, & D'Elia, 2005; Tupper, 1999).

The assessment of cognitive functions is in fact widely comprehensive but, when it comes to emotions, it tends to be one sighted and unidirectional. Emotion matters are reduced to personality aspects, and affective regulation, assessed trough inventories (Bergquist & Maleg, 2002;

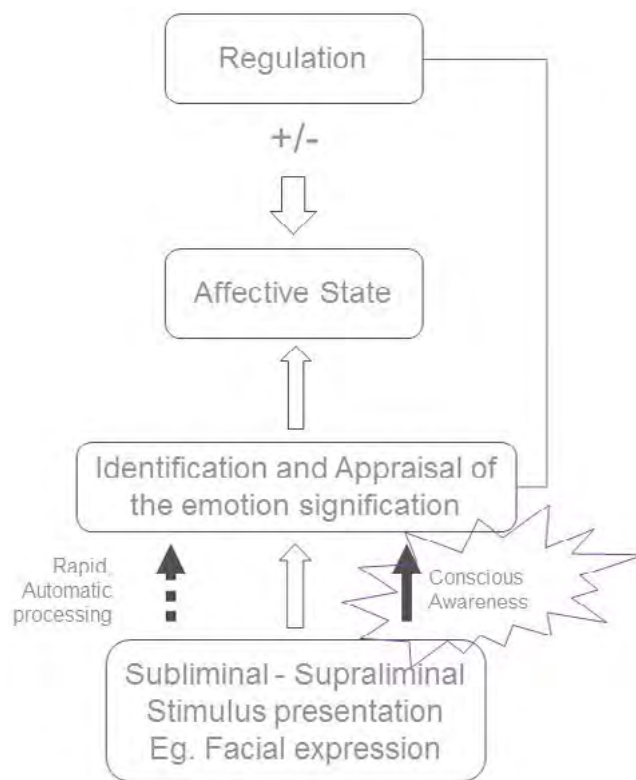


Figura 2. Stages of emotion processing

After subliminal or supraliminal presentation of a stimuli, the central emotion perception and processing stages follow as: (1) the identification of stimulus significance, with or without awareness; (2) the generation of an affective state, which is manifested in emotion expression and behavioral response; and (3) up or down regulation paths of the affective state and identification process. Modified from Stuhmann et al. (2011) (reprinted with permission).

Flaherty-Craig et al., 2002; Groth-Marnat, 2009). This shows that cognitive functions are widely considered, not solely in research but also in clinical practice. Emotional processing, on the other hand, is mostly, not to say exclusively, addressed in research.

One of the complications related to the assessment of emotion recognition in studies with MDD patients is that

they tend to use different experimental paradigms, making it difficult to draw parsimonious conclusions (Bourke et al., 2010; Lawrence et al., 2004; Mercer & Becerra, 2013; Stuhmann et al., 2011). However, most of these measures can be grouped into three categories: psychiatric (clinical), neuropsychological, neurophysiological/neuroimaging techniques and the facial expression recognition tasks/experiment as organized in **Table 2**.

The psychiatric measures include Beck Depression Inventory (BDI) and Hamilton Depression Schedule (HDS) and are mainly focused on assessing symptoms and severity of depression (Bourke et al., 2010; Loi, Vaidya & Paradiso, 2013; Maniglio et al., 2014; Suslow et al., 2015). The neuropsychological measures range from IQ tests to more specific neuropsychological tests such as attentional, visuoperceptive tests, verbal and memory tests (Martino, Strejilevich, Fassi, Marengo, & Igoa, 2011), stroop and dot priming tasks (Delle-Vigne et al., 2014). The neurophysiological and neuroimaging techniques aiming to determine the areas involved with the abnormal functioning of emotion perception, and are mainly Functional Magnetic Resonance Imaging (fMRI) and Event-Related Potentials (ERP) (Adolphs et al., 2000; Delle-Vigne et al., 2014).

Concerning emotion recognition tasks, the variety of experimental paradigms can be categorized into (a) static vs. dynamic stimuli's and (b) identification vs. differentiation as we put in **Table 2**. The most commonly cited tasks are adapted from Ekman and Friesen (1975) which consists of a set of a widely validated facial stimulus of six universal basic emotions mimicked by male and female models (Kohler et al., 2011; Matsumoto et al., 2008; Young, Perrett, Calder, Sprengelmeyer & Ekman, 2002). The static stimuli consist of static pictures of facial expressions while dynamic stimuli's presents short videos. Whereas for normative samples, the static stimuli seem to work well enough, there is a growing debate over their ecological validity for research with clinical subgroups. Some studies suggest that dynamic stimuli's contain relevant information that enhances attention in

neurologically fragile populations (Loi et al., 2013; Richoz et al., 2015; Trautmann, Thorsten, Fehr & Hermann, 2009).

The Facial Emotion Expression Stimuli Test (FEEST) contain a series of these tests based on pictures used in research by Ekman and Friesen (1987). It is a pack ready to be programmed to run a variety of experiments, including categorization, differentiation, and morphing of emotions, with precise procedures and norms for scoring (Young et al., 2002; Kohler et al., 2011).

One advantage of these tasks is that they can be computerized and implemented with rigorous consistency. However, the significant variability in the length of stimulus presentation, ranging from few milliseconds to a couple of seconds seen in such studies, is a subject of discussion (Kohler et al., 2011).

4. Final considerations

The literature concerning emotion recognition deficits in depression and its underlying mechanisms are still unclear (Bourke et al., 2010; Rocca & Heuvel, 2009). The available data supports a negative mood-congruent bias in depressed patients. Depressed patients tend to rate emotional facial expressions more negatively and as less intense, compared to controls, especially for sadness. Correspondingly, they tend to attribute an emotional valence to neutral expressions (Bourke et al., 2010; Kohler et al., 2011; Maniglio et al., 2014; Phelps, 2006; Stuhmann et al., 2011).

These biases are also reported in neurophysiological and neuroimaging studies that reveal structural and functional abnormalities related to emotional regulation and processing (Phillips et al., 2003a,b; Stuhmann et al., 2011). However, there is a debate about if the abnormalities in face processing in MDD patients represent a state or trait, and whether it can represent a vulnerability marker in depression as some proposed. In fact, some authors did not find any association of deficits in identification and differentiation of facial expressions in depression, and neither psychophysiological brain waves

Table 2. Instruments and measures used in studies of facial expression recognition in depression.

Measure	Areas	Instruments
Clinical (Psychiatric)	Depression	Beck Depression Inventory (Beck, 1996). Hamilton Depression Scale (Hamilton, 1960).
	General symptomatology/ functionality	Mini-Mental State Examination (Cockrell & Folstein, 2002). DSM structured interviews MINI Neuropsychiatric Interview (Shenan et al., 1998)
	Cognitive	Benton Facial Recognition Test (Benton, 1990) Executive functions: Semantic and Phonological Fluency (Benton et al., 1983); WAIS
Neuropsychological	Attention	WAIS: Attention: Forward Digit; Attention with a letter-cancellation task D2test Affective priming dot Emotional Stroop Task (William et al., 1996) Dot priming-probe (Halkiopoulos, 1981)
	Visuoperceptive	Trail Making Test (Reitan, 1958). Visual Object and Space Perception Battery (VOSP) Rey Osterieth
	Memory	Rey verbal memory.
Neurophysiological and Neuroimaging	Electrophysiological	Event-related potentials
	Imaging techniques	fMRI – Functional Magnetic Resonance PET
Emotion Recognition Task	Static	Pictures of Facial Affect - POFA (Ekman e Friesen, 1978) FEEST (Young et al., 2002) Ekman 60 faces Reading the mind and the eyes (Baron Cohen et al., 2001)
	Dynamic	FACS dynamic generated
	Identification (labeling)	Ekman 60 faces (Ekman e Friesen, 1978) POFA (Ekman) Reading the mind and the eyes (Baron-Cohen et al., 2001)
	Differentiation	Ekman 60 faces
	Identification and differentiation	Borrod et al. (1977) FEEST (Young et al., 2002)

Note: This table does not intend to be an exhaustive listing as it provides only an overview of the types of instruments used in studies of emotion recognition with MDD patients. On the emotion recognition tasks, we intentionally excluded non-human stimuli's such as avatars due to their less ecological validity. We also organized the table with the aim to provide a representative sample of measures regarding paradigms, so this table does not account for the actual frequency in which these instruments were used in the reviewed studies.

alterations, suggesting a more generalized deficit instead (Kohler et al., 2011; Martino et al., 2011; Radke, Schäfer, Müller, & Bruijn, 2013).

These conflicting results can be presumed to be a consequence of diverse methodological approaches and heterogeneity of the samples in the respective studies (Delle-Vigne et al., 2014). Nevertheless, it is important to consider that if the emotion perception problems are contributing to symptomatic relapse and suicidality, thus, these findings might have some practical implications for prevention and intervention (Maniglio et al., 2014). Some authors even proposed deficits in recognition of facial expression to be biological markers of depression (Fu et al., 2007; Kempleman, 2009) and recommend cognitive social skills training as a target for prevention and intervention (Richard-Devantoy et al., 2013).

Future research is needed aiming to control the methodological issues that tend to add noise to the conclusions drawn by these studies and to test possible interventions. Methodologically, studies need to control for heterogeneity of patients in the sample including medication status, paradigms and experimental design (Castanho et al., 2009; Stuhmann et al., 2011). Experimental designs in future research should also take into account the interactions between emotional processing and cognitive functions, such as memory, attention, and perception. Although many agree that the deficits in emotional processing are not well explained by cognitive deficits, it was also shown that some cognitive processing aspects seem to be automatic for emotional and social stimulus (Phelps, 2006; Stuhmann et al., 2011).


The debate over the use of static vs. dynamic paradigms is another pertinent question to clarify. While normal adults seem not to have problems in decoding static emotional facial expressions, psychiatric samples reveal some difficulties which some authors suggest that are minimized when using dynamic stimulus (Longmore & Tree, 2013; Richoz et al., 2015). This is especially important considering that in real world situations, people

deal with the dynamic stimulus. Therefore, there is a risk that the literature might be overestimating deficits that in real-life are attenuated by using compensatory cues.

On the other hand, if the evidence of the reported deficits holds true against all the current methodological limitations, might be of use to test intervention programs directed to ameliorate the mood-congruent bias. This might mean pairing conventional treatments with alternative interventions targeting training of emotion recognition abilities. Such examples can be seen from trials with individuals with Schizophrenia (See: Statucka & Waldera, 2013) and Autism (See: Southal & Campbel, 2015) which appears to be a promising avenue for future intervention research.

Furthermore, it is advocated for the inclusion of neuropsychological assessment in contexts of tertiary public attention including for cases of depression (Buller, 2008) which should include emotion recognition tasks to allow practitioners to build a complete profile of the patients and signal the need for intervention.

Altogether, these data inform us about the necessity of an integrative neuropsychological view of emotional and cognitive processing in depression. Both cognitive and emotion research can benefit from this integration, and a better comprehension of emotional facilitation and regulation and its effects on cognitive functions might be a step forward towards the growing understanding of depression.

One aspect that may constitute a limitation of this review is the fact that we included some studies with mixed groups of patient conditions (e.g. bipolar and schizophrenia), other studies did not control comorbidity, the effect of the severity of MDD and the effect of medication. Therefore, our conclusions should be considered with precaution. 

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