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A NEUROPSYCHOLOGICAL APPROACH OF EMOTION RECOGNITION DEFICITS IN DEPRESSIVE PATIENTS

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RESUMEN

El reconocimiento de expresiones faciales de las emociones es una habilidad importante para el funcionamiento social. Sin embargo, pacientes con depresión parecen tener déficits en esta capacidad, que pueden contribuir al malo ajustamnto social y las recaídas. En este artículo se revisan los estudios de naturaleza neuropsicológicos y neurofisiológicos actuales sobre los déficits de reconocimiento de emociones en la depresión; se presenta un resumen de las medidas y los instrumentos utilizados con frecuencia en estudios de reconocimiento de emociones en pacientes con trastorno depresivo mayor. Apoyamos un enfoque neuropsicológico integrador de la emoción y la cognición, para entender los déficits en el reconocimiento de emociones y analizar sus implicaciones para la práctica clínica y la dimensión de la investigación aplicada.

Palabras clave: emoción; reconocimiento; expresiones faciales; depresión; Neuropsicología integral.

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APROXIMACIÓN NEUROPSICOLÓGICA DEL DÉFICIT DE RECONOCIMIENTO DE EMOCIONES EN PACIENTES CON DEPRESIÓN

ABSTRACT

Emotion recognition of facial expressions is an important skill for social functioning. Studies have been reporting emotion recognition deficts in Depression which may contribute to poor social adjustment and symptomatic relapse. In this paper we review current neuropsychological studies of emotion recognition deficits in Depression higlignting its neuropsychological correlates and provide an overview of frequently used measures in the studies of emotion recognition with MDD patients. We support an integrative neuropsychological approach for emotion and cognition, to understand the emotion recognition deficits in depression and the need to discuss its implication for clinical practice and research.

Keywords: emotion recognition; facial expressions; depression; integrative neuropsychology.

RESUMO

O reconhecimento de expressões faciais de emoções é uma habilidade importante para o funcionamento social. Contudo, pacientes com depressão parecem apresentar deficits nesta aptidão, o que pode contribuir para o pobre ajustamnto social e recaídas. Neste artigo revisam-se os estudos atuais de cariz neuropsicológico sobre os deficits de reconhecimento de emoções na Depressão, destacamos os correlatos neuropsicológicos e apresentamos uma panorâmica das medidas e instrumentos usados com freqüência nos estudos de reconhecimento de emoções com pacientes com transtorno depressivo maior. Apoiamos uma abordagem neuropsicológica integradora da emoção e da cognição, para entender os deficits no reconhecimento de emoções e discutimos suas implicações para a prática clínica e na dimensão de pesquisa aplicada.

Palavras-chave: emoção; reconhecimento; expressões faciais; depressão; neuropsicologia integrativa.

Emotion recognition is an important skill for social functioning. It allows us to provide a adjusted feedback accordingly to what others transmit to us in social interactions, using facial expressions (Matsumoto, Keltner, Shiota, O'sullivan y Frank, 2008), voice (Bachorowski y Owren, 2008), and touch (Bolognini, Rossetti, Convento y Vallar, 2013). In some psychiatric disorders such as Major Depression however, the ability to accurately decode emotional cues from the face seems to be impaired. This has important implications for clinical practice, considering that these deficits may contribute more to poor social adjustment, persistence of symptoms, recurrent relapse and suicide than other difficulties (Phillips, Drevets, Rauch, Lane, 2003b; Stuhrmann, Suslow y Dannlowski, 2011).

While for schizophrenia and other problems like Autism Spectrum Disorder, the research on emotion recognition deficits is well established, for depression however, the debate is still open. Nevertheless, the need to unravel and mitigate risk factors with updated research and alternative interventions remains mandatory, especially considering the projections for Depression becoming the second most debilitating health problem in the world by 2020 (WHO, 2001). Depression is currently one of the most debilitating psychiatric disorders in the world. By 2020 will be the second most debilitating pathology according WHO projections. Thus, the increasing comprehension of vulnerability factors, biological markers and maintenance factors could contribute greatly for approaching interventions more effectively, clinical and preventively (Stuhrmann *et al*, 2011).

The aim of this paper was to review the main neuropsychological studies of emotion recognition deficits (recognition of facial expressions) in Depression. The methodology adopted for this review consisted of a web search for articles and studies published until November 2015 in online research databases: EBSCO, Science Direct, Psychology Annual Review and Springer. We also considered important bibliographical sources hand searched, such as books series devoted specifically to the subject of emotion recognition and neuropsychology.

We first revisit the framework of facial expression and emotion research, second we summarize the main aspects of the studies with depressed patients and highlight its neuropsychological correlates and finally we provide an overview of the frequent

measures used in the studies of emotion recognition with MDD patients. We support an integrative neuropsychological approach of emotion and cognition, to understand the emotion recognition deficits in depression and further discuss its implications for clinical practice and research.

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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 the species (Ekman, 1973, 1998; Darwin, 1965; Cuve, 2014; Tooby y Comsides, 2008; Rick y Loewestein, 2008). Later, in cross-cultural studies, Ekman found evidence for constants in six basic emotions, namely happiness, sadness, anger, disgust, surprise and fear (Ekman, Friesen, Wallace., y Ancoli, 1980; Ekman, 1998, 1999b Matsumoto *et al,* 2008).

Understating facial expressions of emotion serves a social utility, it allows people to understand affective information in interpersonal interactions and provide a feedback according to it (Niedenthal y Brauer, 2012; Matsumoto *et al,* 2008; Fischer y Manstead, 2008). An efficient emotion recognition tend to be associated with better levels of health functioning and social performance, while difficulties in this ability tend to be linked with the development and maintenance of psychopathology (Delle-Vigne, Wang, Kornreich, Verbanck y Campanella, 2014; Gross, 2008; Hoffman, 2008; Diefenbach *et al,* 2008; Niedenthal y Brauer, 2012).

In fact there a significant research reporting the presence of emotion recognition deficits in many clinical subgroups, such as schizophrenia (Kohler, Walke, Martin, Healey y Moberg, 2010), Asperger (Harms, Martin y Wallace, 2010), neurodegenerative disorders (Kumfor, Sapey-Triomphe, Leyton, Burrell, Hodges, Piguet, 2014) brain lesions (Adolphs, Damasio, Tranel, Cooper y Damasio, 2010) and depressive disorders (Kohler, Hoffman, Eastman, Healey y Moberg, 2010).

2. Emotion research in neuropsychology

Might be surprising to point that some pioneers in neuroscience had initially a strong interest in understanding how emotion works in the brain (LeDoux, 2000; LeDoux y Phelps, 2009; Rick y 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, that were more easily (but mistakenly) understood in terms of computer processing operations (LeDoux, 2000, p.156; Phelps, 2006; Niedenthal y Brauer, 2012).

In addition, the brain lesion studies seemed to have simplified the problem of the nature of emotions, with the appealing concept of the "limbic system" (LeDoux, 2000; LeDoux y Phelps, 2008). In fact, we now know that the limbic system is not limited to, neither fully explanatory for emotions matters.

The ancestral debate over the suitability of the scientific study of emotion was perhaps what caused more problems for emotion research, whether emotion precedes consciousness or the cognitive processing comes before the emotional processing (Phelps, 2006; Panksepp, 2008). 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 y Phelps, 2008). It was possible to study brain processing of external stimuli without first resolving how the conscious perceptual experiences came about. Even though it is widely recognized that "cognitive processes occur unconsciously, with only the products reaching awareness, and then only sometimes" (Kihlstrom 1987 cit. in LeDoux, 2006).

These problems that undermined emotion research emerged in part from dichotomist views of cognition and emotion. Contrary to this extreme dualism, research shows that such separation is unrealistic, and there is a strong evidence that both systems interacts in many aspects (Phelps, 2006; Panksepp, 2009; LeDoux y Phelps, 2008; Young *et al*, 1994; Adolphs, Baron-Cohen y Tranel, 2002; Wager *et al,* 2008).

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

This general data suggest that when it comes to recognition of emotional facial expressions we are considering both a perceptual (cognitive task) and an emotional

processing, and both can interfere in one another. The understanding of human cognition requires the consideration of human emotions, and vice versa, and has it is advocated by some researchers that both areas would benefit greatly from an integrative and interactive approach to the functioning of emotional and cognitive circuits (LeDoux, 2000; Phelps, 2006; LeDoux y Phelps, 2008; Panksepp, 2008).

3. Deficits of emotion recognition of facial expressions in depression in

The studies of emotion recognition with MDD patients report a mood congruent bias in recognizing facial expressions. It seems that depressed patients are more sensitive to negative expressions and prone to rate expressions more negatively, and to attribute emotional valence to neutral expressions (Stuhrmann *et al,* 2011; Burt, Frigerio, Perrett y Young, 2006; Bourke *et al,* 2010). Few studies suggest that this bias seem to persist even after recovering of symptoms and for that reason may contribute to social withdrawal, rejection feelings, persistence of depressive symptoms and be a risk factor for future depressive episodes (Joormann y Gotlib, 2007).

Auerbach, Stewart, Stanton, Mueller, y 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 and found that high-risk boys, but not girls, displayed enhanced perceptual sensitivity to sadness when compared to their low-risk peers. The study suggests that these biases may be present in boys even before onset of major depression and may be a mechanism of risk among male offspring of depressed parents.

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

3.1. Neural correlates of emotional recognition deficits in depression: can anyone tell where?

a) Neural basis of facial emotion recognition

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

Some studies supported the hemispheric localization for emotional processing (Posamentier y Abdi, 2003) while other authors correlated deficits in facial emotion recognition with vary somatosensory lesions including sumpramarginal anterior gyrus, insula and left frontal operculu (Adolphs *et al,* 2000; Adolphs *et al,* 1996). The results from these studies support the hypothesis that emotion recognition system are the same or in part use same rotes of emotional expression (Niedenthal y Brauer, 2012).

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

Philips *et al* (2003a) summarizes 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) findings added that prefrontal cortex is important to regulate emotional responses while the left temporal cortex and the left inferior frontal is important for the lexical semantic of the faces.

I sum, recent studies suggest less support to the asymmetrical theory for emotions, evidencing a fronto-temporo-limbic circuitry for processing emotions (Dal Monte *et al*, 2013; Atkinson y Adolphs, 2011; Wager *et al*¸2008; Flaherty-Craig *et al,* 2002). Once more, this data supports the idea of a more integrative approach for understanding

facial emotion recognition, and farther integration of emotional aspects in the study of cognition and vice versa.

b) **The emotional circuitry for processing facial expressions in Depression**

Research suggest that dysfunctions of left and right, frontal and posterior areas in the brain are linked with specific symptoms present in depression (Shenal, Harrison y Demaree, 2003). In a meta-analyze 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 suggested specific deficits. Suslow, Kugel, Rufer, Redlich, Dohm, Grotegerd y 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 presents more activation in putamen even for moderate sadness expressions, and low activation in response to happy facial expression compared to controls. This supports the over facilitation to negative facial expressions reported by behavioral studies.

Phillips *et al,* (2004) discussing the deficit's in emotional processing and its implications for major psychiatric disorders suggests that the pattern of abnormalities in face processing are linked to specific symptoms and also specific neuropsychological abnormalities. This review highlights the cognitive deficits associated with depression specifically the executive function and the negativity bias. Evidences of structural abnormalities and neuroanatomical functions in regions that are important for emotion, specifically a reciprocal influence of the ventral system, the dorsum neural and probably the cingulate anterior pregenual gyrus and the ventral lateral prefrontal cortex (Philips *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.

Table 1. Structural and Functional Abnormalities in the Ventral and Dorsal Neural Systems Important for Emotion Processing that May Be Associated with Symptom of Major Depression

Identification of Emotional Significance	Production of Affective States and Behavior	Effortful Regulation of Affective States	Symptoms
Structural: Reduced volume within 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 in studies monitoring neural activity in MDD patients, concluding that amygdala, insula, parahipocampal gyrus and orbitofrontal

cortex are working abnormally. Stuhrmaman, Suslow y Dannlowski (2011) summarized schematically the results of fMRI studies as presented in **Fig. 2.**

This bias is seen echoed also in 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 y Wang, 2012).

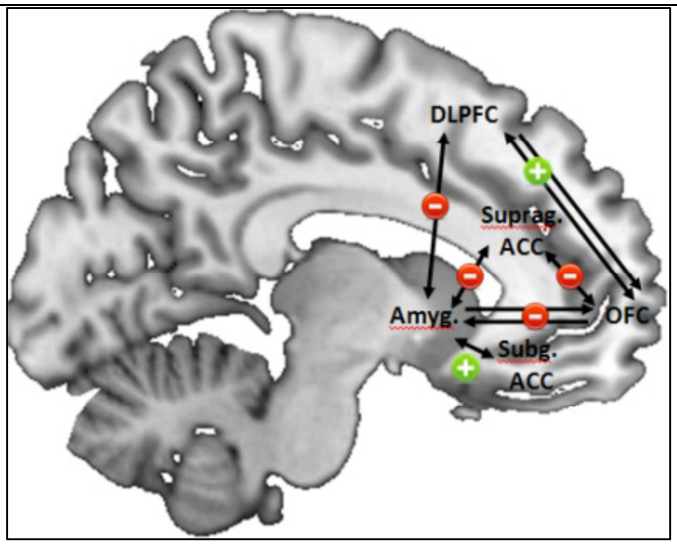

Figure 1. Schematic illustration of main results reported in fMRI connectivity studies on aberrant emotional face processing in Major depressive disorder (MDD) patients. Double arrows represent results derived by functional connectivity approaches, whereas the normal arrows present the result derived by 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 permission from. Stuhrmann, A., Suslow, T., y Dannlowski, U. (2011, *p.* 2)

Klempan, Sequeira, Canetti, Lalovic, Ernst, ffrench-Mullen *et al* (2009) studied GABAergic expression of subjects who committed suicide. Their findings suggests an overexpression of the BA46 gene in suicidal and depressive subjects. These genes are involved in GABAergic transmission associated with depression, and relates to processing facial expressions. Thus, the authors suggest that deficits in facial expression recognition in depression may represent a biological marker of suicidality (Klempan *et al,* 2009; Fu, Williams, Cleare, Scott, Mitterschiffthaler, Walsh y Murray, 2007).

c) Emotion Recognition vs Emotion regulation: integrating inside perspectives

Emotional recognition and emotional regulation tend to be discussed as discrete processes, but in practice, 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, emotional stimulus are processed semi automatically in three phases. 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 behavior. This is useful to generate contextually appropriate, complex affective states, emotional experiences (feelings), and behaviors (Philips *et al,* 2003a; Stuhrmann *et al,* 2011; Diefenbach *et al,* 2008; Clore y Ortony, 2008; Gross, 2008). The problem appears when the recognition pattern of the stimuli is biased it induces inadequate emotional responses. Thus, when stimuli's are consistently misjudged it generates a loop of negative emotional responses, such as those seen in depressive disorders. Philips (2003a) proposed a

model (**see Fig.1**) to explain schematically these three stages, linking emotion regulation and recognition.

This has important implications for psychiatric disorders like depression, especially because it may represent one of its maintenance factors and a risk factor for relapse (Stuhrmann *et al,* 2011; Philips *et al,* 2003a,b). While the current clinical focus in assessment and treatment tend to rely only in emotional regulation, they should be viewed as reciprocal, and should both be considered as targets in research and clinical practice.

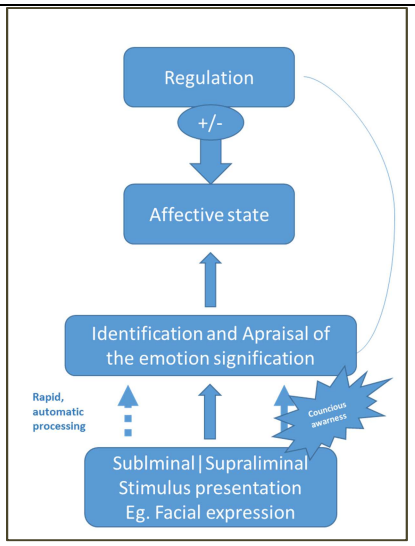

Fig 1. **Stages of emotion processing**. After stimulus presentation (subliminal or supraliminal) the central emotion perception and processing stages are: (1) the identification and appraisal of stimulus significance, taking place with or without conscious awareness; (2) the generation of an affective state, expression of emotion and behavioral response; and (3) up or down regulation (circles with positive/negative signs) of the affective state and identification process. Modified from Stuhrmann *et al,*

2011 y Philips *et al,* 2003a (reprinted with permission)

3.2. The neuropsychological assessment of emotional recognition deficits in MDD

Almost all major neuropsychology textbooks and major taxonomies models for neuropsychological assessment focus mainly in neurocognitive functions. For example, the most cited neuropsychological batteries such as Wechsler Scales, Luria Nebraska Neuropsychological assessment battery, Benton Test, Bender test, Trail making Tests, Rey verbal auditory test, Rey and Osterrieth figures are mainly cognitive (Tupper, 1999; Groth-Marnat, 2009; Flaherty-Craig *et al,* 2002; Mitrushina, Boone, Razani, y D'Elia, 2005; Harrison, 2002; Maia y Leite, 2009).

The assessment of cognitive functions is in fact widely comprehensive but, when it comes to emotions, the sight tends to be unidirectional, reducing emotion matters to personality aspects and affective regulation generally assessed trough MMPI alike inventories as seen in such taxonomies (Groth-Marnat, 2009, Bergquist y Maleg, 2002; Flaherty-Craig *et al,* 2002). This shows that cognitive functions are widely considered not solely in research but also in clinical practice, while emotional processing compared to emotion regulation is been mostly, to not say solely, approached 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 paradigms, different tasks, making it difficult to draw parsimonious conclusions (Bourke, 2010; Lawrence *et al,* 2004; Mercer y Becerra, 2013; Stuhrmann *et al,* 2011). However, we can group the majority of those measures into three categories: psychiatric (clinical), neuropsychological, neurophysiological/neuroimaging techniques and the facial expression recognition task or experiment as organized in **Table 2**.

The psychiatric measures such as BDI e HDS are mainly focused on assessing symptoms and severity of Depression (Bourke, 2010; Suslow *et al*, 2015; Maniglio *et al*, 2014; Loi, Vaidya y Paradiso, 2013**).** The neuropsychological measures range from IQ tests to more specific neuropsychological tests such as attentional, visuo-perceptive tests, verbal and memory tests (Martino, Strejilevich, Fassi, Marengo, y 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 fMRI and Event Related Potentials (ERP) (Adolphs *et al* 2000; Delle-Vigne *et al,* 2014).

In relation to emotion recognition tasks, the variety of experimental paradigms can be categorized in: (a) static vs dynamic stimuli's (b) identification vs differentiation (c) manual vs computadorized as we put in **Table 2**. The most cited tasks in neuropsychological studies are adapted from Ekman and Friesen (1975). They use a set of widely validated facial stimulus of seven universal basic emotions mimicked by male and female models (Kohler *et al,* 2011; *Young*, Perrett, Calder, Sprengelmeyer y Ekman, 2002; Matsumoto *et al,* 2008). The static stimuli consists on static pictures of facial expressions while dynamic stimuli's presents short videos. While in normative populations the static stimuli's seem to work well, there is a recent debate on their ecological validity for research with clinical populations. Some studies suggest that dynamic stimuli's contain relevant information that enhance attention in neurologically fragile populations (Richoz *et al*, 2015; Loi *et al,* 2013; Trautmann, Thorsten, Fehr y 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) ready to be programmed to run experiments with clear procedures and norms for scoring (Young *et al,* 2002; Kohler *et al,* 2011). It allows running experiments for categorization or differentiation of emotions.

While some tasks can be applied manually, others can also use computadorized programmed stimulus (such as FEEST), stroop affective forms and dot priming probs. One problem seen in the studies using a computadorized experiments is a wide variance in the length of stimulus presentation, varying from few milliseconds to a couple of seconds (Kohler *et al,* 2011).

Although less reported in the studies reviewed in this paper, Flaherty *et al,* (2002) mention other measures developed especially for emotional perception and expression, respectively the Chimeric Face Task from Levy and colleagues (1983), the Florida Affect Battery for neurological patients with emotional processing disorders and the

“reading the mind in the eyes” task designed by Baron-Cohen and colleagues (2001) at Cambridge to study the influence of gaze patterns.

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) The Core Depression Scale,
	General symptomatology/functionality	Mini-Mental State Examination DSM structured interviews (APA) MINI Neuropsychiatric Interview (Shenan *et al,* 1998)
Neuropsychological	Cognitive	Benton Facial Recognition Test Executive functions: Semantic and Phonological Fluency (Benton *et al*., 1983); WAIS
	Attention	WAIS: Attention: Forward Digit (Wechsler) Attention with a letter-cancellation task D2test Affective priming dot Stroop tasks
	Visuo-perceptive	Trail Making Test part B (Reitan, 1958). Visual Object and Space Perception Battery (VOSP)

		Rey Osterieth (Rey)
	Memory	Rey verbal memory.
Neurophysiological and Neuroimaging	Electrophysiological Imaging techniques – localization	Event related potentials fMRI – Functional Magnetic Resonance PET
Emotion recognition Task	Static	Ekman e Friesen (1978) FFEST (Young *et al,* 2002) Ekman 60 faces Pictures of facial Affect - POFA (Ekman e Friesen, 1978) Reading the mind and the eyes (Baron Cohen *et al,* 2001)
	Dynamic	FACS dynamic generated
	Manual	Ekman e Friesen (1978) FFEST (Young *et al,* 2002) Ekman 60 faces POFA (Ekman) Reading the mind and the eyes (Baron Cohen *et al,* 2001)

Computadorized	FFEST (Young *et al,* 2002)
	Ekman 60 faces (Ekman e Friesen, 1978)
	Reading the mind and the eyes (Baron-Cohen *et al,* 2001)
	FACS dynamic generated
	Emotional Stroop Task (William *et al, 1996)*
	Dot priming-probe (Halkiopoulos, 1981)
Identification (labeling)	Ekman 60 faces (Ekman e Friesen, 1978)
	POFA (Ekman)
	The Florida Affect Battery (Flaherty-Craig *et al,* 2002)
	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 intend to provide an overall (and not conclusive) view of the types of instruments used in studies of emotion recognition with MDD patients. We intentionally excluded not human stimuli's there is a singificative reserach suggesting that they do not represent ecological valid stimuli's, and included the measures that seem to produce more consistent results in research. We also organized the table with aim to provide a representative sample of measures in terms of paradigms and not to represent the number in which these instruments were actually cited in the studies.

4. Final considerations

The literature concerning emotion recognition impairments in depression and the mechanisms involved are not still clear and the clinical implications are not widely discussed (Rocca y Heuvel, 2009; Bourke *et al,* 2010). The available data supports a negative mood-congruent bias associated with the emotion recognition in depressed patients. Depressed patients tend to rate emotional facial expressions more negatively and less intense compared to controls, especially for sadness. In addition, they tend to attribute an emotional valence to neutral expressions (Maniglio *et al*, 2014; Phelps, 2006; Sthutmaman, 2011; Bourke *et al*, 2010; Kholer *et al,* 2011).

These biases are also reported in neurophysiological studies (Stuhrmann *et al,* 2011; Philips *et al,* 2003b) and neuroimaging studies reveal structural and functional abnormalities related to emotional regulation and processing (Phillips *et al, 2003a;* Stuhrmann *et al*¸2011). However, there is a debate if the abnormalities in face processing in MDD patients represents a state or trait, and whether it can represent a vulnerability marker in depression. In fact, some authors did not found any association of deficits in identification and differentiation of facial expressions in depression, and neither neuroelectrophysiological waves alterations, suggesting more generalized deficits instead of the negative bias reported in the literature (Kohler *et al,* 2011; Radke, Schäfer, Müller, y Bruijn, 2013; Martino *et al,* 2011).

We assume that these conflicting results are a consequence of different methodological approaches including heterogeneity of groups of patients and measures (Delle-Vigne *et al,* 2014) Nevertheless, it is also important to consider that if these emotion perception problems might be contributing to relapse and suicidality, thus, these findings might have some practical implications on prevention (Maniglio *et al,* 2014). Some authors even proposed that deficits in facial expression are biological markers in depression (Fu *et al,* 2007; Kempleman, 2009) and cognitive social skills training may be a target for prevention (Richard-Devantoy *et al,* 2013).

Further research is needed specially controlling the methodological issues that tend to add noise to the conclusions drawn by these studies. Heterogeneity of subjects, paradigms, experiment design and issues related to medication need to be controlled in order to produce more clear results that may lead to more parsimonious theories (Castanho *et al*, 2009; Sthurmaman *et al,* 2011).

These accounts also the need to approach in experimental designs the interactions between emotional processing and cognitive functions, such as memory, attention and perception. Although many studies agree that the deficits in processing emotional expressions are not well explained by cognitive deficits, it is also shown that some cognitive processing aspects seems to be automatic for emotional and social stimulus (Phelps, 2006; Stuhrmann *et al,* 2011).

The debate on the static vs dynamic paradigms also need to be carefully treated, while normal adults seems to not have problems in decoding static emotional facial expressions, psychiatric populations reveal some difficulties that some authors suggest that are minimized when using dynamic stimulus (Richoz *et al,* 2015; Longmore y Tree, 2013). This is especially important considering that in real life situations, people deal with dynamic stimulus, so there is a risk that the literature may be overestimating deficits that in real life are minimized by the use of compensatory cues.

Altogether, these data inform us about the need 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 in a depth understanding of depression.

One aspect that may constitute a limitation of this review is the fact that we included some studies with mixed group of patients with Bipolar and schizophrenic patients, other studies did not controlled comorbidity, the effect of the severity of MDD and the effect of medication. Also, our goal present an integrative argument, rather than presenting a systemtic review of studies, our discussions need to be considered with some relativity.

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