Facial emotion recognition in neurological disorders: a narrative review

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Summary. Facial emotion recognition refers to a person's interpretation of facial features of another to identify a particular emotional state. It is essential in human evolution and encompasses distinct neural networks. Facial emotion recognition is altered in most neurodegenerative diseases, but literature just focus on single neurological pathologies or limited comparison with psychiatric pathologies. It is unknown if a common pattern of affection through pathologies exists or if facial emotion recognition changes according to the underlying pathology. This review discusses its development in healthy population, synthesizes facial emotion recognition studies regarding most common neurological diseases, as well as most relevant findings in neuroimaging and current treatments. Facial emotion recognition, especially negative emotions, is altered in all described neurodegenerative diseases and could constitutes an early marker of cognitive deterioration.

Key words. Emotions. Facial expression. Facial recognition. Nervous system diseases. Therapeutic treatment.

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Introduction

Emotions are one of the most basic psychophysiological reactions existing in the human being. Its study is relatively recent; it was in 1971 when psychologist Ekman established six basic emotions of the human species: anger, disgust, fear, happiness, sadness and surprise [1]. This classification is relatively universal and it is present from very early age in all cultures [2]. Therefore, knowing how to recognize and to interpret them is a key aspect of human evolution.

Emotions are mainly perceived through facial expressions. Facial emotion recognition (FER) allows us to understand, discriminate and respond to a large number of stimuli, as well as being key in interpersonal relations and in the prediction of prosocial behavior [3].

Brain damage or neuronal degeneration can cause specific deficits in FER leading not only to a decrease in the ability to socially relate, but a loss of effective communication with the rest and a clear neurological deterioration. In some disorders, a FER deficit may indicate an alteration in cognitive areas such as perception, attention and memory [4], or be an early warning sign for neurological diagnosis [5]. Therefore, the study of FER is essential in neurological pathologies, in order to elucidate what mechanisms underlay each of them and to be able to diagnose and treat these deficits in a timely manner.

Until now, literature on FER focuses only in one pathology like Alzheimer's or Parkinson's disease, or in limited comparisons of neurological disorders and psychiatric disorders, being schizophrenia the most common. No studies synthesizing impairments in FER along major neurological pathologies have been found, and therefore, it is not known whether there is a common pattern of affection through different pathologies or if perception of emotions changes according to the underlying pathology.

In the present study, the main aim is to describe the characteristics of FER in the most important neurological diseases, as well as to expose FER processes in healthy population, to present the most relevant findings in neuroimaging and to define the most relevant psychological, neurological and pharmacological treatments in this field.

This narrative review includes the most recent research on FER area regarding the most relevant neurological diseases. For the selection of these pathologies, the classification proposed by World Health Organization [6] has been followed, and it has been selected those manuscripts that were related to FER in a minimum of three articles in indexed journals.

It is intended to summarize the current knowledge that exists in this extensive field, providing a rigorous perspective about the onset, development and alteration of FER, as well as current treatments and to provide future research lines. Therefore, a review of 'state-of-the-art' has been considered to be the best approach since the broad field and of-

fers contrasted information on the different aspects included in these areas. Providing a global view of the current state-of-the-art also offers the possibility of establishing connections and conclusions between pathologies that would not be possible with the study of a single disease. In addition, this article synthesizes several systematic reviews and meta-analysis, so a narrative review could be the best way to summarize all the results found.

In order to provide a greater scientific rigor for this manuscript, the following methodology has been conducted. A search of literature has been done in the scientific web search engines PubMed and PsychInfo. Search terms were the name of each pathology or topic (for example, gender) combined with the Boolean operator 'AND' and 'facial emotion recognition*'. In addition, it has been performed a manual search on Google Scholar for further studies not yet identified.

The inclusion criteria for manuscripts were the following: temporal criteria (less than twenty years of publication date from now) and relevance on literature (journals rank from quartile 1 and quartile 2 in most cases). Systematic reviews and meta-analysis of each topic have been prioritized but, in most cases, research studies have been included, given the lack of review data on certain topics.

Facial emotion recognition in healthy population

Development of facial emotion recognition

There are two complementary pathways in FER: the first pathway involves perception of facial features and the second pathway decodes the specific emotion they express [7].

Bruce and Young [8] presented a functional 3-step cognitive model to explain perception of facial features: structural coding (to analyze the perceived stimulus in order to extract invariant properties of a face), complex visual processing (analysis of metric relationships between face elements together with other aspects like age or gender), and face representation construction. This model is still valid, having been validated by several authors [9].

Once the facial features have been recognized, we can perceive and discriminate the emotions. But how and when does this process take place? It has been studied how babies, when they are exposed to a face, associate the most salient stimuli (mouth or eyes) with the vocal expression they hear [10]. It seems that, while information from the lower part of the face is useful in recognition of happiness, disgust and surprise, information from the eyes helps in fear, anger and sadness [11]. Then, together with other social or environmental characteristics, emotion learning is brought upon, remaining stable until adulthood [12].

In general, it is considered that babies, at 4-6 months of age, can discriminate between happiness and fear, showing preference for the latter [13]. In fact, happiness recognition is one of the first to mature: at 5 years of age, children have a similar accuracy and speed level to adults [12].

About the rest of basic emotions, a fine-grained mapping of FER development was established by Rodger et al [12]. His group characterized three distinct groupings: expressions that remain stable from early childhood (happiness and fear), expressions with more gradual improvement between 5-12 years (sadness, surprise) and those that show a steep improvement during early adolescence to adulthood (disgust, neutral and anger). However, there is discrepancy in literature regarding this classification. Some authors, consider that sadness, for example, is recognized much earlier [14]; even though these contrasts may be due to methodological differences in studies [15].

Finally, this process improves through adolescence (11-15 years), where performance seems to approach adult's [16], although some studies claim that FER will continue improving until adult age [17].

Gender differences in facial emotion recognition

An important controversy exists over whether there are gender differences in FER. In general, females are considered to have a greater advantage over males for several reasons: differences in brain maturation, different neural activation patterns, by cognitive factors such as more empathy or attention to facial features [15] or to modulation by hormonal factors on FER structures [18].

Notwithstanding, other reviews have not found any gender differences in FER, and argue that female advantage could be explained by the use of different methodologies in studies, by the participant's own gender bias or by the intensity of presented items [19]. Because to date literature is mixed and inconsistent, more research is needed in this area.

Ageing in facial emotion recognition

As we age, difficulties gradually emerge in FER. These changes usually begin at the age of 40-50, with a clear tendency towards progressive worsening with

age [20]. Specifically, studies indicate that older adults, compared to the younger ones, have more problems in identifying negative emotions like sadness, anger and, to a lesser degree, fear [3]. The recognition of disgust, joy and surprise seems to be preserved and stable over time [21].

In their review, Ebner et al [3] exposed the reasons that could explain this progressive deterioration:

- *Cognitive changes:* both by a decline in divided attention, in memory, in executive function or in processing speed. Even at a motivational level, older adults identify positive emotions better than young people.
- *Progressive neuronal degeneration in areas responsible for FER:* especially, frontal and temporal regions.
- *Visual pattern of facial scan:* the elderly focus their eyes on mouth area (a focused-gaze strategy), while young adults repeatedly look at several areas (exploratory-gaze strategy), which is more informative for FER.

Neuronal basis on facial emotion recognition

Recognizing facial emotions requires of the proper functioning of several brain structures, shaping an interactive network with distributed activity in time and space [22].

One of the classic models that explains this network procedure is by Haxby et al [9]. His group explained that, in FER, a double autonomous processing exists: on one hand, invariant features of face (facial structure) would be processed in a 'central system' located in occipito-temporal regions of the extraestriatal visual cortex, namely fusiform gyrus. On the other hand, as it would be necessary to interpret other aspects, other neural regions would be recruited: specifically, the changing aspects (such as gaze, identity or lips movement) would be processed in the superior temporal sulcus and amygdala [23].

There is a huge discussion on whether this dual processing actually follows independent pathways or occurs simultaneously. Some studies support the idea that invariant and changeable properties activate independent neuronal circuits [24]. Contrary, other authors claim that not only the fusiform gyrus is also involved in the analysis of changeable properties [25], but that, in general, both circuits interact, being influenced by each other [26].

Some research in neurological pathologies such as prosopagnosia seems to support the 'independency hypothesis' [27], as well as different electrophysiological evidences [28]. In this line, Luo et al [28], analyzed different event-related potentials and magnetoencephalic recordings of subjects before a FER task, developed an electrophysiological perceptual model of three stages. His team discovered that in FER there is a first processing stage, which is fast, automatic and coarse, and serves to distinguish the fear faces from the rest of the emotions. This processing stage relies on the activation of the amygdala and the orbitofrontal cortex. During the second stage, it discriminates between emotional and neutral facial stimuli. On the third, emotional facial expressions are differentiated as positive or negative. Processing in the last two stages is sensitive to attentional resources while processing in the first stage remains relatively independent. Further studies are on the same line to this model [29], although more research is required in this area.

Regarding the neural correlates of recognizing different emotions, Fusar-Poli et al [30], synthesized 105 fMRI studies comparing neuronal activation of patients exposed to faces with different emotions with respect to neutral faces, creating a functional atlas. Their conclusions are summarized in the table.

Despite the fact that this meta-analysis did not include surprise, later studies with fMRI state exposed that the activated areas are right amygdala and right thalamus, right postcentral gyrus and left posterior insula [31].

Cognitive factors in facial emotion recognition

Cognitive factors that can also influence in FER should be considered. Attention, for example, is an essential cognitive resource in FER. From an evolutionary perspective, it has allowed us to detect emotional facial information that may signal potential threats in the environment. Studies show that an attention failure causes a deficit in accuracy and speed of FER in pathologies such as schizophrenia [32], in attention deficit disorder [33] or in autism spectrum disorders [34], although this deficit can be improved with guided instructions to focus attention [35].

Executive function is a cognitive domain related to complex problem-solving, retrieval abilities, organizational strategies and concept formation, processes that are likely to be involved when making judgments about emotional expressions. A dysexecutive deficit has been related to a poor performance in FER in neurologic pathologies like Parkinson's disease [36] or in psychiatric as schizophrenia [37].

Working memory can influence FER as well, as it continuously updates, stores and retents facial features generated from personal knowledge and also

Table. Differential emotional processing compared with neutral faces.

	Left areas	Bilateral	Right areas
Happiness	Fusiform gyrus	Amygdala	Anterior cingulate cortex
Fear	Fusiform gyrus	Amygdala	Medial frontal gyrus
Sadness	Lingual gyrus		Amygdala
Angry	Insula		Inferior occipital gyrus
Disgust	Fusiform gyrus	Insula	Thalamus

helps in the manipulation of complex and numerous social cues [38] .

As it has been exposed, although there is little literature at the present moment, there are certain cognitive factors clearly related to FER functioning, but more research is needed in this area and future studies should consider it to be of the same importance as neuronal structures or electrophysiological records.

Neurodegenerative disorders

The neurodegenerative disorders currently represent one of the principal causes of mortality and chronic disability worldwide [6]. In most of them, there are alterations in FER, which can be an early warning sign of the development of the disorder or aggravating other deficits when the pathology has already manifested. Knowing these alterations can not only help us in early detection, but also understand their impact and the implications for people and their environment. Therefore, on the following section, the most relevant neurological pathologies and their relationship to FER are exposed.

Mild cognitive impairment

Mild cognitive impairment refers to an intermediate stage between normal ageing and dementia, where there some cognitive functions are impaired but still allow for reasonable independent living.

Overall, recent systematic reviews and meta-analyses indicate that subtle deficits in recognition of negative emotions such as anger, sadness and fear already appear in mild cognitive impairment [39]. On the contrary, recognition of disgust, happiness and surprise remain stable [40]. These deficits are observed in both amnesic and non-amnesic mild cognitive impairment [5], although there are authors that only report deficits in the amnesic subtype [41].

Alzheimer's dementia

In several cases, mild cognitive impairment progresses to Alzheimer's dementia, in which a progression in FER alterations is found [42].

Sadness has been the emotion in which alterations have been mostly reported [43], what can be considered early marker of deterioration in this disorder. Deficits in surprise recognition and disgust have also been found [44].

More controversy exists in happiness recognition: some authors find alterations [45], while others assert that it remains relatively stable, even in advanced stages of the disease [46]. These differences in literature could be explained by the different paradigms used, the size of the sample or the disease stages of the subjects analysed.

This process seems to respond to a progressive degeneration of the neuronal circuit responsible for FER; frontotemporal regions [39]. In particular, Sapey-Triomphe et al [47], through fMRI studies, concluded that fear recognition was negatively correlated with the amygdalar volume, disgust with the volume of the pallidum and happiness with the volume of the fusiform gyrus. Other authors argue that, more than a self-alteration in FER, these patients fail in these tasks due to alterations in attention and processing speed, in long-term memory, in visuospatial dysfunctions or social and behavioural difficulties [48], or because of mood [43].

Finally, it is important to emphasize the importance of cognitive reserve in this pathology due to its high influence in the beginning and progression of the disease. Cognitive reserve refers to 'the extent to which an individual uses neural networks or cognitive paradigms efficiently and flexibly rather than anatomic differences'; and includes a higher intellectual quotient, education, occupational attainment or participation in leisure activities [49]. It is considered that, the higher cognitive reserve the greater connectivity between areas, the higher the volume in neuronal regions resulting in a later onset of the disease [50].

Parkinson's disease

Given the special difficulty of patients with Parkinson's disease in expressing both spontaneous and faked facial emotions, the study of FER in this pathology is of particular relevance.

Studies have produced some inconsistent results, although a closer inspection seems to point to a decay in the ability [51], more specifically towards negative emotions like anger, sadness or fear [52]. Recently, alterations in positive emotions recognition in advanced stages have also been reported [53].

The impairment in FER seems to have a positive correlation with a decrease in the ability to express facial emotion, indicating an 'emotional mirror neural mechanism', suggestive of a shared system with common pathways [54]. The impairment has also been linked to a diminished visual form perception, even though its influence would only be partial [55] and to a greater severity of symptoms. The deficit also seems independent from working memory or executive dysfunction [51] or from psychiatric status, such as depression, anxiety or apathy [56].

These findings clearly point to a critical role of the dopaminergic nigrostriatals pathways and the basal ganglia-thalamocortical circuits, specifically in the connections of basal ganglia to the orbitofrontal cortex and the anterior cingulate cortex [53].

Therefore, it is logical to consider the possibility of improving these deficits with the active taking of dopaminergic medication. According to the literature, patients with Parkinson's disease (with active taking of medication and without taking), show deficits on FER in contrast to controls, both for recognizing emotions in photographs and for the computerized ones [57]. However, it is true that, in case of no medication taking, a more pronounced deficit exist and specifically regarding the recognition of negative facial emotions. Differences have been found in recognition of disgust, anger or fear [58] regarding controls.

The effect of deep brain stimulation of the subthalamic nucleus has also been studied. There are discrepancies in literature: some authors such as Aiello et al [59] state that, in a situation of dopaminergic therapy, the continuous stimulation produces brain micro-lesions that impair FER, especially the disgust. Mondillon et al [60] agree that deep subthalamic stimulation alters the recognition of disgust but improves if dopaminergic therapy is added and concludes that the combined application of both treatments would benefit FER. On the contrary, authors such as Albuquerque et al [61] consider that the combined treatment does not affect FER.

It should be noted that these FER alterations seem to be associated to apathy and to manifest before the appearance of cognitive symptoms [62], so it could be considered as an early marker of cognitive degeneration in some cases, having an important role for detection and treatment of deficits.

Huntington's disease

Huntington's disease is determined by an autosomal dominant mutation of the huntingtin gene; therefore, its diagnosis can be known long before it is manifested. Thus, there is a 'pre-stage' and the actual Huntington's disease stage once the cognitive and motor symptoms are present.

Interestingly, the scientific literature confirms that there are already failures in the recognition of negative emotions in presymptomatic stages [63], especially in disgust in several sensorial modalities [64]. These early deficits would be secondary to the reduction of grey matter in structures like the bilateral insula [65] and the basal ganglia, mostly in the striatum [66]. As neurodegeneration progresses, failures in FER are aggravated, especially in the recognition of anger and fear [63]. On the other hand, similar to Parkinson's disease, it seems that recognition and facial expression failures are related, suggesting common brain structures such as striatum [66].

Unlike other neurodegenerative disorders, when contextual cues (such as body position) are added to FER stimulus, Huntington's disease patients seem to obtain similar results to controls [67]. Since their visual scanning pattern is similar to controls, it appears that this would be more related to the use of additional superior cognitive processes [68].

Finally, social cognition impairments seem to be an early marker to have in consideration related to the onset and progression in Huntington's disease [63].

Frontotemporal dementia

Frontotemporal dementia appears due to progressive atrophy of fronto-temporal regions, producing dramatic changes in several areas. Depending on the severity, three subtypes can be considered: behavioral (major alteration in behavior), semantic dementia, and progressive non-fluent aphasia if it affects language.

Studies have focused mainly on the behavioral variant and its comparison with Alzheimer's disease, given its similar neural basis. Studies show that FER is more severely impaired in the behavioral variant than in Alzheimer's disease patients and healthy controls [69], in negative emotions, but also in positive emotions when there is more impairment in frontal regions [70].

When frontotemporal dementia and Alzheimer's disease are compared, frontotemporal dementia

has been found to present more deterioration in the regulation of behavior and executive functions, as well as a greater alteration in theory of mind [71]. Since these areas appear to be modulators of FER, it would explain the greater severity of these alterations with respect to Alzheimer's disease.

However, does recognition of negative emotions deteriorate evenly in different subtypes? This is the question that Kumfor et al [72] wanted to find a response to. His team studied the neural correlates of each subtype, and established that there was a greater alteration in the orbitomesial frontal regions in the behavioral variant; in the anterior temporal lobes in semantic dementia; and in the left insula, upper temporal gyrus and lower frontal regions in progressive non-fluent aphasia. Future research will help clarifying each altered region and its FER impaired correlate.

Finally, Kumfor et al note that all variants present alterations in FER, but point out that there is a primary emotion processing impairment in semantic dementia, whereas in the other two subtypes there may be an attentional deficit that influencing the results. In contrast, Oliver et al [73] reject this conclusion as they were not able to find any improvement in FER when isolating facial features to increase selective attention.

Amyotrophic lateral sclerosis

A percentage of amyotrophic lateral sclerosis patients suffer from altered bifrontal cortical function, what leads to abnormal emotion perception. A notable impairment in perception of anger, surprise and disgust of individuals with preserved frontotemporal function [74] has been reported. Until quite recently, it has been assumed that amyotrophic lateral sclerosis coursed with preserved cognitive functioning, so they were allowed to take important end of life decisions. But these types of decisions are based, in part, on interpersonal judgment, the ability to perceive emotional signals and to act appropriately, and therefore any alteration in emotional processing can limit decision-making capacity and to participate in their own care [75]. These considerations, therefore, should be taken into account given the association of amyotrophic lateral sclerosis with frontotemporal dementia and the objective decline in social cognition.

Therefore, a better understanding of cognitive alterations in the disease will help clinicians and caregivers promote a better quality of life for the affected individuals.

Epilepsy

Epilepsy encompasses a broad spectrum of diseases with clinical similarities but varied etiology. Studies on temporal lobe epilepsy –the most common form of the disease– have detected a worse recognition of fear and, to a lesser extent, in disgust and anger [76].

Because of the heterogeneity of epilepsy, studies show inconsistent results depending on the age of onset and severity of the disease, surgery and lateralization of surgery, subtype of epilepsy [77] and premorbid intellectual quotient [78] Research on FER in epilepsy is very recent and neither the impairment nor the moderating variables are yet fully understood. Nevertheless, knowledge in this field can provide with an excellent toehold for a better understanding of the neural correlates of social cognition and the complex neural networks involved in it.

Traumatic brain injury

Traumatic brain injury is an alteration in brain function, or other evidence of brain pathology, caused by an external force, and it is a leading cause of disability and mortality worldwide [6]. Due to its heterogeneity, it is classified according to the severity of the alterations, from mild to severe.

The few studies that can be found in the literature on FER and traumatic brain injury in moderate-severe degree, confirm an alteration in the recognition of all basic emotions, with special difficulty in negative emotions [79]. Other authors showed that reduced subjective experience, especially of sadness and fear, was associated to poor emotion matching but not emotion labelling [80]. The same authors explain that affective semantic knowledge and face perception appeared to be relatively intact in these patients, and they improve their results in FER tasks with contextual clues.

Demyelinating diseases

Multiple sclerosis

Besides being an inflammatory, demyelinating and degenerative disease, multiple sclerosis also courses with cognitive impairment, fatigue and affective disorders. For this reason, researchers hypothesised that social cognition, and most specifically FER, could also be impaired. Confirming their hypothesis, multiple sclerosis patients are significantly worse

at FER than healthy controls amongst other domains of social cognition [81]. The impairment is independent of the physical disability or disease severity [82] although it has been found a substantial correlation between FER impairments and psychological and social aspects of quality of life [81]. A more recent study found that depression, facial discrimination and fatigue were the biggest predictors of emotion recognition accuracy [83].

Although cognitive decay and depression could account for part of FER impairment cause, it seems a relatively independent domain when a sample with very low depression scores is analyzed [81]. Improvements in the approach to treating multiple sclerosis have yielded to a better quality of life and have increased lifespan. Because it is not known how to improve their social aspects of quality of life could impact the wellbeing of the patient, developing research in this field further could provide clinicians with tools to address affective and social problems associated to the disease.

Treatments in facial emotion recognition and neurological diseases

As it has been shown, there are difficulties in FER in all the described neurological pathologies, in some cases being a clear early marker of deterioration. Since FER is related to other cognitive areas, to a good neuronal functioning and to the psychosocial functioning of the person, it is of special relevance to find treatments that can improve these alterations. Therefore, the purpose of this section is to describe the current pharmacological and psychotherapeutic treatments for FER in the neurological disease exposed.

Pharmacological treatments

One of the treatments that has been more studied to date is oxytocin, a neuropeptide secreted by the posterior pituitary, key in social behavior [84]. Recent meta-analyzes confirm that treatment with a single intranasal dose of oxytocin significantly improves the recognition of fear, anger and happiness [84], both in controls and patients with various neurological and psychiatric pathologies. Related to specific neurological disease, for example, it was found that intranasal oxytocin improved social cognition of patients with the behavioral variant of frontotemporal dementia [84].

However, the mechanisms of action of intranasal oxytocin are still quite unknown: given the extensive distribution of their receptors in the brain, it is hypothesized that it may improve attentional, learning, or act on the neural activation of the amygdala [84].

Finally, there are authors who point out that increased serotonin [85] or dopamine combined with light therapy [86] could facilitate FER, especially in the recognition of fearful faces. However, this has only been studied in psychiatric pathologies such as anxiety or depression, and more investigation is needed to apply it in neurological disorders.

Neurological treatments

Recently, how therapies like transcranial magnetic stimulation can improve FER. In patients with schizophrenia it has been observed that, applying a repetitive electric current in the left dorsolateral prefrontal cortex significantly improves the accuracy of FER after only 10 sessions [87].

In the field of neurological diseases, this technique has been shown to be effective by enhancing neurogenesis and suppressing apoptosis in the hippocampus of rats [88]. It has also been tested in healthy aging and Alzheimer's disease patients, obtaining an improvement in diverse cognitive functions that seems to remain in time [89]. However, it is still a very novel technique and the results must be replicated to ensure the promising results obtained to date.

Psychotherapeutic treatments

Despite the great relevance of FER in the diversity of neurological pathologies, there are few psychotherapeutic programs focused on its treatment and rehabilitation. In fact, the first treatments focused on FER were in the field of psychiatric pathology, specifically in schizophrenia, and they are still the more currently developed.

Programs like GAÏA s-face [90] or Microexpression Training Tool (METT) –a self-guided online training program [91]– have demonstrated their effectiveness by improving the accuracy of FER in patients with schizophrenia. They all combine virtual exposure sessions with static faces, videos and personal sessions with therapists to extrapolate learning to real life.

Based on the positive results, some of these programs have been used for neurological disorders with alterations in FER. This is the case for Huntington's disease, in which the METT program was used for patients in presymptomatic and symptomatic stages. Through intensive instructions to direct visual attention during 8 sessions in 1 month, the

program produced a significant improvement in the accuracy in FER [92].

In the case of Alzheimer's disease, a combined treatment of emotional rehabilitation and cognitive stimulation was innovatively developed in 36 patients with mild Alzheimer's disease. After 40 sessions in six months, a significant improvement in recognition of sadness, disgust, surprise and neutral expression was found, which was maintained a month later [93]. It should be noted that this treatment did not only improve FER, but also processing speed, basic activities of daily living and maximized Mini-Mental State Examination scores. Although they have not yet been replicated, the results are very promising.

For patients with traumatic brain injury, a multifaceted treatment for social cognition and emotion regulation (T-ScEmo) has been developed. It consists in a compensatory strategy training for impairments in FER, theory of mind and social behavioral skills. The intervention takes from 16 to 20 weekly 1-hour sessions, and patients not only had improved all areas including FER, but this improvement was maintained up to five months after treatment [94]. Nevertheless, and for further improvement, new treatments targeting FER in persons with traumatic brain injury, could benefit from taking into account theories of affect recognition, strategies used in autism and teaching techniques commonly used in traumatic brain injury.

In general, it seems that training with guided instructions to certain specific facial areas can increase the accuracy and speed of FER. Possibly, future psychotherapeutic approaches for FER will be oriented in this line, given the promising results obtained in different psychiatric pathologies to date.

Recommendations to improve future intervention programs, such as those by Vianin [95] can help develop future strategies. These point out that, in order to maximize their effectiveness, virtual applications must be developed so that the person can work in a safe, directed environment and without the emotional influences of others, although with the presence of a therapist to guide the patient in their learning. Subsequently, it is crucial that this learning is extrapolated to the situations of daily life that can improve the functionality of the patient in order to ensure that the goals of the interventions are met.

Conclusions

The aim of this article is to synthesize the most recent studies on the FER in the most frequent neurodegenerative pathologies. Following an exhaustive narrative review on the current state of this topic, it is important to highlight certain aspects. Most of the scientific information consulted significantly supports Ekman's initial classification of the six basic emotions. Also the two neurological pathways activated to process invariant and variable facial characteristics [7], although it is not clear whether this double activation is simultaneous or independent; more studies support the latter hypothesis [26] although more research is still needed to confirm it.

Happiness is the first emotion we learn to identify, a process that occurs around five months of age [13]. The rest of emotions are identified progressively, being the lower part of the face key to interpret disgust and surprise, and the upper part crucial in fear, anger and sadness recognition [11]. There are discrepancies regarding gender differences in FER: some authors consider that there is a greater female advantage due to neurological, cognitive [15] and hormonal [18] factors, others consider that the differences are secondary to observer biases and/or context [19]. By the age of 40-50, accuracy in FER decreases, especially in the recognition of negative emotions. This could be explained both by the neuropsychological and neurological impairment associated with age, and by a progressive tendency to focus gaze on the lower part of face ('focused-gaze strategy') [3].

The current review synthesizes the results of a meta-analysis published in 2009, where 105 studies exposes the neurological areas involved in each basic emotion [30]. To identify a facial emotion, a global brain activation occurs, although there are certain neuroanatomical areas highlighted, such as the amygdala, insula or fusiform gyrus. Although cognitive factors such as executive functions [37], attention [32] and working memory [38] are of special relevance in FER, in terms of better accuracy and speed of recognition, there is limited research in this regard, therefore, it is essential to consider this area in future research.

In the second part of this review, the main neurological pathologies and their relationship with FER are exposed. Mild cognitive impairment is the stage prior to the appearance of most of these diseases; different systematic reviews and meta-analysis confirm that, in this phase, there are already alterations in recognition of negative emotions [39]. This supports the natural evolution of FER in adults, as previously discussed.

Globally analyzed, it seems that the alteration of the recognition of negative emotions, especially of sadness, disgust and anger, appears both for corti-

cal pathologies, such as Alzheimer's [43] or frontotemporal dementia [69], subcortical as Parkinson's [52] or Huntington's disease [63] and demyelinating. Reasons refer, basically, to the degeneration of related neuroanatomical structures. Several studies confirm that the alteration in the recognition of sadness could become an early marker for Alzheimer's disease [43] and disgust for Huntington's disease [63]. Disgust recognition is also altered in Parkinson's disease, closely related to the neurodegeneration of insula; in this pathology, in addition, the deficit in FER has been correlated with deficit in facial emotion expression [54].

FER also altered in cases of epilepsy or traumatic brain injury, although, given the heterogeneity of the neurological disorder, it is difficult to draw conclusions. In epilepsy, studies focus on the alteration in temporal lobe, structure involved in emotions such as fear, so it is logical to find greater alteration in the recognition of this emotion [76]. In moderate-severe traumatic brain injury, the deficit in FER seems to be secondary to a lower subjective experience, especially of sadness and fear [80], and to visuoperceptive difficulties. In some of the rehabilitation programs, some studies have found improvement if an additional visual context is provided [79]. These data, however, open an interesting field of activity in terms of considering FER as a key element in the cognitive rehabilitation and global functional recovery of these patients.

People with multiple sclerosis also present worse performance in FER compared to controls, especially negative emotions such as fear or anger [81]. In some studies, these deficits can appear before the neuropsychological deterioration, and be of greater magnitude than the cognitive alteration [96]; however, others consider that both deficits in social cognition and neuropsychological are associated [81]. Somehow or other, deterioration in FER may implies *per se* a neuropsychological deterioration because, when recognizing facial emotions, we activate cognitive areas such as attention, executive functions, working memory, etc. Regarding clinical aspects, certain studies find deterioration in FER relatively independent of behavioral alterations [82], others consider that depression and fatigue present in multiple sclerosis are more predictors of poor FER performance than neurodegeneration [97]. Due to multiple sclerosis appears at younger ages than other neurodegenerative diseases, it is essential to further investigate this field, as well as more studies about the relationship between clinical/cognitive improvement and FER, and its possible relationship with an improvement in social functioning.

There is more controversy regarding positive emotions. Several studies affirm that there is a certain deficit associated with later stages of deterioration [43] and related to neuronal degeneration of especially frontal regions [70], while others do not detect alterations even in more advanced stages [46]. It is therefore necessary to consider this field as a possible line of future research.

According to the publications, in general, all neurodegenerative pathologies exposed have in common a worst performance in negative emotions recognition rather than positive ones. One reason may be due to positive emotions are easier to discriminate than negative ones, even when there are associated clinical, neurological and cognitive alterations. Literature describes happiness as the first emotion we recognize, so their learning and discrimination could be more resistant to neurodegeneration. In addition, at a more advanced age, there is a greater tendency to focus gaze on the lower part of the face, a representative area of positive emotions [10], so that neurodegeneration could first alter the recognition of those emotions that do not receive as much attention. Further evidence supports this hypothesis, as the results obtained in rehabilitation programs, where patients are trained to look at certain representative facial areas, show good results maintained over time [92]. On the other hand, difficulties in discrimination between negative emotions could be explained by neuropsychological deficits; that is, the attentional, executive and visuoperceptive alterations present in most neurological pathologies could influence onto a bad discrimination between sadness and anger, etc. more than between sadness and happiness for example.

The specific differences between pathologies could be explained by the structures involved in the recognition of each emotion; that is, in Alzheimer's, where neurodegeneration begins initially in temporary medial structures, there is a worse recognition of sadness, emotion associated to areas such as the amygdala. In Parkinson's and Huntington's disease, there is a degenerative onset of thalamic structures, also implicated in recognition of disgust. In this review, we have shown how these deficits occur in FER.

Finally, the review synthesizes the most relevant pharmacological, neurological and psychotherapeutic treatments for FER to date. Pharmacological research has mostly focused in the use of intranasal oxytocin. Literature validates the use of intranasal oxytocin for the improvement of recognition fear, anger and surprise both in the general population and with mental or neurodegenerative pathology [84,98]. Although its mechanism of action is unknown, there

are certain hypotheses that point to a possible improvement of cognitive domains or stimulation of the amygdala. Neurological studies have only reported trials with transcranial magnetic stimulation in patients with schizophrenia. Transcranial magnetic stimulation acting on the dorsolateral prefrontal cortex showed that after 10 sessions, they improved in FER [87]. To date, studies in this field have not been applied to neurodegenerative pathology, but the results are promising. In addition, this technique seems to reduce apoptosis in the hippocampus, improve neurogenesis and cognitive functions in rats [88]; therefore, applied to humans, it could improve FER, but it is still a very novel technique that provides very promising results. At a psychotherapeutic level, research has focused on pathologies such Alzheimer's disease, Huntington's disease and traumatic brain injury: the treatment focus especially on guided self-instructions to certain specific facial areas. The use of computer programs such as METT, emotional rehabilitation or T-ScEmo, combined with cognitive stimulation and social skills training, it offers very positive results in FER that seem to maintain in time. In addition, it also seems to improve cognitive functions and quality of life [93], so clearly, future research should be oriented to this field of detection and intervention.

FER is a key construct of social cognition that allows us to interact and maintain optimal social functioning. Quality of life and good social functioning are one of the main objectives to achieve in neurological disorders; aspects that could be conditioned by a well recognition and interpretation of facial emotions. Likewise, a deficit in FER can, in some cases, not only modify social functioning, but also indicate underlying cognitive alterations or help in the early detection of diseases such as Alzheimer or Huntington. Despite its importance, the detection and treatment of FER deficits is not yet fully implemented in exploration and cognitive stimulation programs. The obtained results in FER treatment show promising results, which could even lead to an improvement in other cognitive or clinical domains, with an improvement in the disease burden. To date, more research is needed in this area, but it is a priority to consider FER as one key aspect to detect and treat in neurological pathology.

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Reconocimiento facial de emociones en trastornos neurológicos: una revisión narrativa

Resumen. El reconocimiento facial de emociones hace referencia a la interpretación de una persona sobre los rasgos faciales de otra para identificar un determinado estado emocional. Es esencial en la evolución humana y abarca distintas redes neuronales. A pesar de que el reconocimiento facial de emociones se ve alterado en la mayoría de las enfermedades neurodegenerativas, la bibliografía sólo se centra en patologías neurológicas individuales o en limitadas comparaciones con patologías psiquiátricas. Se desconoce si existe un patrón común de alteración entre las patologías o si el recono-

cimiento facial de emociones cambia según el trastorno subyacente. Esta revisión describe su desarrollo en población sana y sintetiza los estudios de reconocimiento facial de emociones en relación con las enfermedades neurológicas más comunes, así como los hallazgos más relevantes de neuroimagen y los tratamientos actuales. El reconocimiento facial de emociones, especialmente en emociones negativas, está alterado en todas las enfermedades neurodegenerativas descritas y podría constituir en algunos casos un marcador temprano de deterioro cognitivo.

Palabras clave. Emociones. Enfermedades del sistema nervioso. Expresión facial. Reconocimiento facial. Tratamiento terapéutico.