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Social cognition dysfunctions in patients with epilepsy: Evidence from patients with temporal lobe and idiopathic generalized epilepsies

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ABSTRACT

Background and aim: Despite an extensive literature on cognitive impairments in focal and generalized epilepsy, only a few number of studies specifically explored social cognition disorders in epilepsy syndromes. The aim of our study was to investigate social cognition abilities in patients with temporal lobe epilepsy (TLE) and in patients with idiopathic generalized epilepsy (IGE).
Materials and methods: Thirty-nine patients (21 patients with TLE and 18 patients with IGE) and 21 matched healthy controls (HCs) were recruited. All subjects underwent a basic neuropsychological battery plus two experimental tasks evaluating emotion recognition from facial expression (Ekman-60-Faces test, Ek-60F) and mental state attribution (Story-based Empathy Task, SET). In particular, the latter is a newly developed task that assesses the ability to infer others' intentions (i.e., intention attribution — IA) and emotions (i.e., emotion attribution — EA) compared with a control condition of physical causality (i.e., causal inferences — CI).
Results: Compared with HCs, patients with TLE showed significantly lower performances on both social cognition tasks. In particular, all SET subconditions as well as the recognition of negative emotions were significantly impaired in patients with TLE vs. HCs. On the contrary, patients with IGE showed impairments on anger recognition only without any deficit at the SET task.
Discussion: Emotion recognition deficits occur in patients with epilepsy, possibly because of a global disruption of a pathway involving frontal, temporal, and limbic regions. Impairments of mental state attribution specifically characterize the neuropsychological profile of patients with TLE in the context of the in-depth temporal dysfunction typical of such patients.
Conclusion: Impairments of socioemotional processing have to be considered as part of the neuropsychological assessment in both TLE and IGE in view of a correct management and for future therapeutic interventions.

1. Introduction

During the last century, the interest on cognitive deficit in the course of epilepsy considerably increased. A large body of research has been indeed collected, defining the cognitive profile of patients with epilepsy [1] and the importance of the assessment of cognitive functions on the comprehensive care program for persons with epilepsy [2]. While the first behavioral studies of patients with epilepsy mostly tested global intelligence level, only recently, researchers tried to disclose specific neuropsychological profiles according to the different subtypes of epilepsy [1]. Overall literature findings suggest that the neuropsychological evaluation must take into account the subtype of the epileptic syndrome, in particular its localization, the possible etiology, and the pharmacological therapy. All these elements undoubtedly influence the presentation of the cognitive deficits [1]. Although an intensive research led in the last few decades in this field, it is still unclear whether such cognitive impairments are generally related to the chronic and stigmatizing condition the epilepsy patients live or are mainly due to a specific neuropathological process [3].

Temporal lobe epilepsy (TLE) is the most common focal epilepsy syndrome. Cognitive functions may be variably impaired in people with TLE. Even if memory deficits are usually the core of the cognitive phenotype of TLE, impairments largely vary also including low global intelligence level and deficits on verbal learning, visuospatial skills, and

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executive functions (e.g., problem solving) [1,4,5]. On the contrary, idiopathic generalized epilepsy (IGE) is defined as an epilepsy syndrome that has no apparent cause and is assumed to have an underlying genetic etiology. Compared with cognitive impairments in TLE, those in IGE are less prominent and, thus, have been less investigated by researchers. At the cognitive evaluation, most of patients with IGE show just a low global intelligence level [6]. Nevertheless, some of these patients can, however, present mild deficits on attention, visuospatial skills, and nonverbal memory tasks [6–10].

Social cognition is a high-level cognitive function that broadly includes all the processes used to understand and store information about the interactions with other people in a social context [11]. Perception of social signals pertaining to others' mental states is a fundamental prerequisite in order to obtain a correct formulation of the appropriate responses. In addition, a correct processing of such signals, attributing independent mental (knowledge, beliefs, and motives) or emotional (feelings) states to other individuals, is also fundamental to understand and predict others' behavior.

Epilepsy condition can variably impair social cognition abilities according to the localization of the epileptic focus and to the associated pathology. However, the clinical significance of such deficit is largely unexplored. Some studies provided evidence of the presence of deficits of negative emotion recognition in patients with TLE as well as in patients with IGE [12,13]. These authors showed that fear and disgust recognition is impaired in both epilepsy syndromes, with TLE deficit also extending to facial identity recognition [12]. Other studies have previously proved selective impairment of fear recognition in the TLE syndrome [13,14]. Reynders et al. also showed a fear recognition deficit in a small sample of patients with IGE [13].

In addition, in the last years, researchers also investigated the mentalizing abilities of patients with TLE and patients with IGE. Low performances on different Theory of Mind (ToM) tasks [15–20] have been reported in TLE. Both cognitive and affective facets of ToM have been proved to be impaired [19]. Identification and comprehension of sincere, deceitful, and sarcastic social exchanges are also impaired in individuals with TLE [20]. This deficit seems to be partly related to the presence of mesial temporal lobe sclerosis and the early age at seizure onset [20], as also supported by Giovagnoli et al. [16]. Cohn and coworkers also proved in a voxel-based morphometry MRI study a significant relationship between left hippocampal atrophy and overall social inference abilities, as well as between left anterior temporal neocortex atrophy and sarcasm comprehension [20]. These results support a critical role of the anterior temporal cortex as converging zone of higher-order perceptual and emotional processes and of stored representations. Studies assessing different facets of ToM ability in patients with TLE proved, however, contradictory results, ranging from wide ToM impairments [15,19,21] to normal performances [22] or selective deficits [23]. Such variability is probably due to the intrinsic heterogeneity of the TLE syndrome which may include lesional and nonlesional patients, as well as cases with unilateral (left or right) or prevalent medial or lateral TLE. The same authors suggested a role of executive functioning in performances of patients with TLE on ToM tasks, although in disagreement with a previous study supporting a dissociation between these cognitive functions [15].

At the opposite, only a few studies investigated other aspects of social cognition (e.g., social judgment, and empathy) in patients with IGE [13,24], consistently proving impaired social cognition abilities compared with healthy controls. Cognitive and affective empathy has been indirectly investigated by Jiang et al. by means of the Interpersonal Reactivity Index (IRI) questionnaire. Study findings provided evidence of the presence of a selective perspective-taking deficit in patients with IGE with preserved ability of the affective component supporting the more limited damage of social cognition networks in this epilepsy syndrome [24].

The aim of this study was to investigate in patients with TLE and in those with IGE the type and the severity of ToM deficits, assessing for the first time the ability to attribute mental states (either intentions or emotions) to others with a single newly ad hoc developed task. We also assessed the ability to recognize emotions from facial expression in both epilepsy syndromes and explored possible correlation of social cognition performances with executive measures. In particular, we used the Italian version of the Ekman-60-Faces (Ek-60F) test [25] and the Story-based Empathy Task (SET) [26]. According to the neural correlates of socioemotional processing, which involves specific frontotemporal and limbic networks [27,28], we expected low social cognition performances in both IGE and TLE but with a wider impairment in patients with TLE. We also hypothesized that deficits of basic cognitive functioning may result in patients with TLE and in those with IGE in poor performances on specific social cognition tasks.

2. Methods

2.1. Subjects

We recruited 39 consecutive patients with epilepsy (21 patients with TLE (8 males; mean age = 37 ± 12.5 years) and 18 patients with IGE (6 males; mean age = 26.3 ± 7.2 years)) referred to the Centre for the Diagnosis and the Treatment of Epilepsy of University of Palermo (Palermo, Italy). All participants underwent electroclinical phenotyping using a validated seizure questionnaire and review of medical records to investigate age at seizure onset, ictal semiology (described by both the patient and an external observer), seizure frequency, and response to treatment. Selected cases underwent a prolonged video-EEG monitoring for seizure recording. Seizure semiology in patients with TLE was based above all on the clinical history in all cases. Unfortunately, since we observed only unspecified interictal discharges and no seizures during the EEG monitoring, we were not able to identify specific lateralization.

Patients with TLE in our study did not show mesial temporal sclerosis or other structural brain lesions at the brain MRI; indeed, recognition of subtle cortical abnormalities is limited by actual neuroimaging resolution. So, we can define our patients as having “probably symptomatic” TLE, taking into account that some of them should have unrecognized subtle malformations of cortical development that correlate with the localization of the focus of epilepsy [29,30].

Exclusion criteria for patients' enrollment were an age younger than 18 years, a positive anamnesis for psychiatric disorders, and the presence of comprehension deficits or learning disorders that may influence the results of the neuropsychological evaluation. In addition, we recruited on a voluntary basis (i.e., partners or relatives of patients with epilepsy) a control group of 21 age-, gender-, and education-matched healthy subjects (HCs; 12 males; mean age = 31.95 ± 11.54 years) with no history of neurological or psychiatric illnesses. See Table 1 for demographic and clinic details of the enrolled sample.

All subjects or their caregivers gave informed consent to the experimental procedure, which was approved by the local ethics committee.

2.2. Standard neuropsychological battery

Both patients and HCs underwent a battery of neuropsychological tests in order to provide background information about their cognitive functioning. In particular, memory and executive functions (Rey Auditory Verbal Learning Test; Rey's Figure Recall Test; Verbal and Visual Digit Span Task; and Attentive Matrices) (see Lezak, 2000 for details) [31]; language abilities (Phonological and Semantic Fluency; Token test [32]; Aachener Aphasie Test (AAT) naming [33]); and visuoperceptual and visuospatial abilities (Rey's Figure Copy Test) (see Lezak, 2000) [31] were assessed in each patient. Depression and anxiety were investigated with the Beck Depression Scale (BDI, total score = 0–39) [34].

2.3. Experimental social cognition battery

A brief experimental battery including the Italian version of the Ekman-60-Faces (Ek-60F) test [25] and the Story-based Empathy Task

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Table 1
Demographic features and clinical features of the enrolled sample.

	Patients with TLE (n = 21)	Patients with IGE (n = 18)	HCs (n = 21)	Patients with TLE–HCs		Patients with IGE–HCs	
	Mean ± SD	Mean ± SD	Mean ± SD	T-value	p-Value	T-value	p-Value
Sex (male/female)	8/13	6/12	12/9	$X^2(1) = 1.13$	n.s.	$X^2(1) = 2.21$	n.s.
Age at interview (years)	37 ± 12.5	26.3 ± 7.2	31.95 ± 11.54	1.36	n.s.	−1.78	n.s.
Education (years)	10.8 ± 3.1	11.9 ± 2.6	12.5 ± 3.96	−1.55	n.s.	−0.58	n.s.
				TLE–IGE			
				T-value		p-Value	
Age at onset	24.3 ± 13.2	15.14 ± 7.7	–	2.58		***0.01***	
Duration of epilepsy	12.9 ± 10.0	13.5 ± 8.2	–	−0.23		n.s.	
Number of AEDs	1.3 ± 0.7	1.2 ± 0.5	–	0.74		***0.46***	

TLE = temporal lobe epilepsy; IGE = idiopathic generalized epilepsy; HCs = healthy controls; AEDs = antiepileptic drugs.

(SET) [26] was used to explore socioemotional processing both in patients and in healthy controls. Both tasks were developed by the Neuroscience Division of San Raffaele Scientific Institute (Milan, Italy).

The EK-60F test is a well-known neuropsychological tool assessing emotion recognition from facial expressions. It consists of 60 b/w pictures from the Ekman and Friesen series of Picture of Facial Affect [35], which depict the faces of 10 actors (6 females and 4 males), each one displaying six basic emotions (i.e., happiness, sadness, anger, fear, surprise, and disgust). A global score (GS) of 60 indicates the best possible performance, and each basic emotion has a subscore of a maximum of 10 points.

The SET is a nonverbal task of mental state attribution to other individuals (see [26] for more details on test construction and stimuli selection). In summary, the whole task, lasting about 15–20 min, consists of two main experimental conditions, i.e., identifying intentions (SET − IA) and emotional states (SET − EA), plus a control condition entailing the inference of causality reaction based on knowledge about the physical properties of objects or human bodies (SET − CI). The task requires a subject to describe the story that is presented in a comic strip composed of three pictures in the upper half of the page, formulate a possible story ending, and then select the correct ending present in the lower half of the page (from three possible endings to the story: plausible, implausible, or plausible but incorrect) [26]. A global score (GS) of 36 indicates the best possible performance and each condition has a subscore of a maximum of 12 points.

The whole battery (including basic neuropsychological assessment; see above) lasted approximately 80 min. Group differences and correlation analyses were computed using either parametric or nonparametric statistics after testing for the normal distribution of data. Group differences in demographic and cognitive neuropsychological data were analyzed through two sample t-test analyses comparing each experimental group with HCs. Because of the nonnormal distribution of social cognition data, we performed nonparametric statistics. We controlled for multiple comparison using False Discovery Rate. The Mann–Whitney U test was used to analyze group differences in social abilities, while Spearman's correlation analyses were performed to investigate the relationship between social and basic cognitive abilities. All statistical analyses were conducted using Statistica 8.0.

3. Results

No significant differences between patients and HCs in gender (patients with TLE vs. HCs: $X^2(1) = 1.13$, $p = 0.28$; patients with IGE vs. HCs: $X^2(1) = 2.21$, $p = 0.14$), age (patients with TLE vs. HCs: $t(40) = 1.36$, $p = 0.18$; patients with IGE vs. HCs: $t(37) = -1.78$, $p = 0.08$), or educational level (patients with TLE vs. HCs: $t(40) = -1.55$, $p = 0.13$; patients with IGE vs. HCs: $t(37) = -0.58$, $p = 0.57$) were found.

Compared with HCs, patients with TLE showed significant deficits on all the explored cognitive functions except for selective attention (Table 2). On the contrary, patients with IGE compared with HCs showed impaired performance only on semantic fluency ($t(37) = -3.45$, $p < 0.01$) and naming tasks ($t(37) = -3.68$, $p < 0.01$) (Table 2). The group with TLE and the group with IGE did not significantly differ between each other in any cognitive task (Table 2).

Patients with TLE showed impaired performances in both mentalizing and emotion recognition abilities (Table 3). Compared with HCs, this group of patients showed significant lower scores in all the SET subconditions (SET − EA: $Z = -2.54$, $p = 0.03$; SET − IA: $Z = -2.20$, $p = 0.04$; SET − CI: $Z = -2.57$, $p = 0.03$). Moreover, TLE presented also a global deficit at the Ek-60F ($Z = -2.36$, $p = 0.04$), which was related to negative emotion recognition ($Z = -2.09$, $p = 0.05$). On the contrary, compared with HCs, the group with IGE showed a limited impairment of social cognition abilities, which was restricted to low performances on the Ek-60F anger recognition score ($Z = -2.69$, $p = 0.04$) (Table 3). No significant difference at the SET task was found in the group with IGE compared with HCs.

It is worth noting that, with regard to controlling for multiple comparison, no significant difference at the Ek-60F fear recognition score was found in the IGE vs. HC comparison. Taking into account that a deficit of fear recognition at the Ek-60F was consistently reported in the IGE literature [12–14], we also computed statistical comparison without correcting for multiple comparison to see whether our data showed also a trend in this direction. According to previous literature, we thus proved low performance on Ek-60F global score ($Z = -2.12$, $p < 0.05$), with not only low anger ($Z = -2.69$, $p < 0.01$) but also low fear ($Z = -2.02$, $p < 0.05$) recognition scores.

Correlation analyses between social cognition performances and basic cognitive functions highlighted in the group with TLE a positive correlation between a measure of working memory (i.e., the adjusted score at the Verbal Digit Span Task) and the global scores of both SET ($r = 0.52$, $p < 0.05$) and Ek-60F ($r = 0.54$, $p < 0.05$) tasks. Story-based Empathy Task — causal inference condition also correlated with working memory performance ($r = 0.52$, $p < 0.05$). However, these results did not survive correction for multiple comparisons.

4. Discussion

In this study, we investigated two key aspects of social cognition abilities in patients with TLE and in patients with IGE. In particular, we assessed emotion recognition from facial expressions and the ability to infer others' intentions and emotions and correlated the performances with basic cognitive functioning. As expected, we confirmed mild cognitive impairments on language, memory, and executive domains in patients with TLE mainly related to the damage of the temporal lobes and its connections [36,37]. Mild deficits on language domains have

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Table 2
Neuropsychological characteristics of patients and controls at basic cognitive tests. Mean adjusted score ± standard deviation is reported. TLE = temporal lobe epilepsy; IGE = idiopathic generalized epilepsy; HCs = healthy controls; RAVLT = Rey Auditory Verbal Learning Test.

	Patients with TLE (n = 21)	Patients with IGE (n = 18)	HCs (n = 21)	Patients with TLE–HCs		Patients with IGE–HCs		Patients with TLE–patients with IGE	
	Mean ± SD	Mean ± SD	Mean ± SD	T-value	p-Value	T-value	p-Value	T-value	p-Value
Semantic fluency	25.38 ± 10.80	24.27 ± 4.50	33.43 ± 8.01	−2.74	***0.03***	−3.45	***0.01***	0.35	n.s.
Phonological fluency	21.43 ± 12.13	21.56 ± 15.08	28.90 ± 9.63	−2.21	***0.05***	−1.83	n.s	−0.02	n.s.
Digit span	5.09 ± 1.10	5.34 ± 1.38	5.79 ± 0.88	−2.23	***0.05***	−1.19	n.s.	−0.63	n.s.
Corsi Block span	4.05 ± 0.64	4.7 ± 0.9	4.7 ± 0.82	−2.84	***0.03***	−0.97	n.s.	−1.57	n.s.
RALVT — immediate recall	36.97 ± 8.75	42.97 ± 7.35	42.99 ± 7.25	−2.42	***0.04***	−0.007	n.s.	−2.29	n.s.
RALVT — delayed recall	6.8 ± 3.33	8.5 ± 1.8	9.6 ± 2.26	−3.18	***0.03***	−1.69	n.s.	−1.92	n.s.
Rey–Osterrieth Complex Figure — copy	30.74 ± 4.89	32.42 ± 1.86	33.32 ± 1.55	−2.30	***0.04***	−1.61	n.s.	−1.34	n.s.
Rey–Osterrieth Complex Figure — recall	10.69 ± 6.26	13.2 ± 6.24	14.39 ± 6.90	−2.31	***0.04***	−0.54	n.s.	−1.23	n.s.
Attentive matrices	49.17 ± 6.77	49.2 ± 5.56	49.09 ± 3.99	0.05	n.s.	0.08	n.s.	−0.02	n.s.
Token test	30.83 ± 4.66	32.8 ± 2.42	33.58 ± 2.37	−2.39	***0.04***	−0.93	n.s.	−1.60	n.s.
Aachener Aphasie Test Naming	117.14 ± 4.69	117.05 ± 3.42	119.86 ± 0.65	−2.62	***0.04***	−3.68	***0.01***	0.06	n.s.

been proved also in patients with IGE, as previously reported by few studies [38–40].

In addition, an extended disruption of social cognition abilities has been proved in the group with TLE involving both mental state attribution and global emotion recognition. According to previous literature findings [15–20], showing a wide damage of mentalizing skills in patients with TLE, our patients presented statistically significant differences compared with HCs in both aspects of ToM (cognitive and affective ToM). In addition, statistical analysis proved also a significant lower performance in patients with TLE compared with HCs at the SET — CI control condition (i.e., causal inferences) supporting the presence of a wider deficit in the basic cognitive processes (e.g., executive control) that underlie the performance in a nonverbal task such as the one employed here.

In agreement with the epilepsy syndrome localization, precise pathologic temporal cortex damage has been suggested as a possible cause of the cognitive deficits of patients with TLE [41]. In fact, volume loss in this syndrome not only involves the hippocampus and the surrounding brain areas (e.g., amygdala and parahippocampal gyri) but also could be extended to extratemporal cortical and subcortical regions [27,42]. Therefore, this wide brain damage may cause a broad impairment of the cognitive functioning, as proved by the low performances of our group with TLE on basic neuropsychological evaluation and at the SET — CI subcondition. Similarly, the mentalizing system which involves many different regions along the cortical midline and in the temporal lobes, including the temporoparietal junction, the temporal poles, and the medial prefrontal cortex [11,43–46], may be impaired in such patients. A widespread microstructural derangement particularly involving the bilateral limbic circuit and the frontotemporal connections has been reported in TLE [47,48], supporting the impairment of ToM ability even in apparently individuals without lesional epilepsy, as the ones reported in our sample. Further, structural or functional MRI studies are, however, needed to unveil precise relationship between selective features of patients with TLE and specific social cognition deficits in order to better define cognitive profiles according to the epileptic syndrome.

While both experimental conditions (i.e., intention and emotion attribution) are broadly impaired in our sample of patients with TLE, the group with IGE did not show any deficit on the SET experimental conditions. This latter result is in contrast to the study of Jiang and coworkers [24] that indirectly documented in IGE syndrome a deficit of perspective-taking with the cognitive empathy subscale of the IRI questionnaire. The opposite result could be related to intrinsic differences between patient samples or to the specific characteristics of the two ToM tasks used (i.e., IRI questionnaire vs. SET — cartoon task). Larger studies are, however, needed to clarify this issue. Emotion recognition from facial expression, particularly with negative valence, was also significantly impaired in patients with TLE compared with HCs. The emotion recognition deficit was, on the contrary, more restricted in IGE which showed only anger

Table 3
Theory of mind ability and facial emotion recognition in patients and controls.

	Patients with TLE (n = 21)	Patients with IGE (n = 18)	HCs (n = 21)	Patients with TLE–HCs		Patients with IGE–HCs		Patients with TLE–patients with IGE	
	Mean ± SD	Mean ± SD	Mean ± SD	Z-value	p-Value	Z-value	p-Value	Z-value	p-Value
Story-based Empathy Task — SET									
SET — task global score	27.7 ± 5.5	29 ± 7.6	32.04 ± 5.45	−3.01	***0.03***	−1.54	n.s.	−1.17	n.s.
SET — intention attribution	9.3 ± 2.3	9.7 ± 2.9	10.76 ± 1.72	−2.20	***0.04***	−0.95	n.s.	−1.01	n.s.
SET — emotion attribution	9.5 ± 1.6	10.1 ± 2.2	10.62 ± 1.99	−2.54	***0.03***	−0.74	n.s.	−1.46	n.s.
SET — causal inferences	9.0 ± 2.4	9.2 ± 3.1	10.47 ± 2.23	−2.57	***0.03***	−1.38	n.s.	−0.69	n.s.
Ekman 60 Faces Test — Ek-60F									
Ek-60F — global score	47.6 ± 4.7	47.4 ± 5.4	51.19 ± 3.98	−2.36	***0.04***	−2.12	n.s.	0.07	n.s.
Ek-60F — negative emotions	21.71 ± 4.06	21.50 ± 4.32	24.09 ± 3.30	−2.09	***0.05***	−1.93	n.s	0.09	n.s.
Ek-60F — positive emotions	18.33 ± 1.49	18.88 ± 1.23	18.80 ± 1.32	−1.24	n.s.	0.20	n.s.	−1.24	n.s.
Ek-60F — surprise	8.6 ± 1.3	8.9 ± 1.1	8.95 ± 1.12	−1.30	n.s.	−0.01	n.s.	−0.90	n.s.
Ek-60F — happiness	9.8 ± 0.5	9.9 ± 0.2	9.85 ± 1.12	−0.94	n.s.	0.22	n.s.	−1.26	n.s.
Ek-60F — fear	6.0 ± 2.5	5.8 ± 2.0	7.29 ± 2	−0.5	n.s.	−2.02	n.s.	0.27	n.s.
Ek-60F — disgust	7.4 ± 1.7	7.4 ± 2.5	8.29 ± 1.76	−1.67	n.s.	−1.03	n.s.	−0.55	n.s.
Ek-60F — anger	7.6 ± 1.4	7.1 ± 1.3	8.28 ± 1	−1.64	n.s.	−2.69	***0.04***	1.28	n.s.
Ek-60F — sadness	8.4 ± 1.3	8.5 ± 1.7	8.52 ± 1.36	−0.40	n.s.	−0.4	n.s.	0.08	n.s.

TLE = temporal lobe epilepsy; IGE = idiopathic generalized epilepsy; HCs = healthy controls.

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and fear recognition difficulties. Since anteromedial temporal and limbic (amygdala and anterior insula) structures play a crucial role in decoding emotions, especially in negative emotion (e.g., anger and fear) recognition [49–51], a wider deficit in TLE is expected. Nevertheless, patients with IGE have been proved to be also having impairment in the recognition of such emotions [12,13]. A derangement of the thalamofrontal pathways and of the communication between those regions and limbic (mainly amygdala) structures has been supposed at the basis of fear and disgust deficits in patients with IGE [12]. Thalamofrontal network dysfunctions have been indeed proved in such patients supporting this selective cognitive deficit [52]. Our data supported a more extensive impairment of emotion recognition, which could be related to dysfunction of the abovementioned networks including also the medial prefrontal cortex that is specifically engaged in the recognition of anger facial expression [50,53–55]. As previously suggested [13,56], disease duration may influence performances on emotion recognition, since IGE is typically a long-lasting epilepsy syndrome. Long duration or disequilibrium between critical and intercritical periods may indeed affect neuronal networking causing functional and then structural damages. In addition, the acquisition of this specific cognitive function starts at very early stages of the individual development [57]. So, the more precocious and repeated is this process over time, the less efficient is the communication between the sets of neural structures involved in emotion recognition, which need a deep synergistic integration in order to obtain a correct recognition of facial emotions [58].

Finally, we proved a correlation between the Verbal Digit Span Task and the global performances on both the SET task and the Ek-60F task. Notwithstanding, these results do not survive the FDR correction; they support the relationship between the executive control system and social cognition abilities, often commonly impaired in frontal lobe dysfunction [59,60]. In fact, both functions engage shared pathways involving the frontal associative neocortex related to abstract reasoning and cognitive control [61,62], which are abilities required during social functioning in resisting to the interferences and in inhibiting a spontaneous response in favor of the correct one [63,64]. Together with domain-general cognitive functions, social functioning requires, however, additional domain-specific abilities mostly related to temporal regions [65–67]. It is, however, to acknowledge that the main limitation of the study is the relatively limited sample size; large longitudinal studies on different populations are certainly needed not only to confirm our data but also to establish more in detail the role of social cognition disorders in patients with TLE and in patients with IGE.

5. Conclusions

Our results expand previous findings on social cognition disorders in patients with TLE and in patients with IGE. We suggest that such disorders, existing in both focal (TLE) and generalized (IGE) epilepsy subtypes, are related to structural and functional alterations of specific neuronal pathways involving frontotemporal and limbic regions within an extremely complex neuronal networking. According to this, our study findings support the integration of social cognition assessment in the standard neuropsychological battery of patients with epilepsy. A better definition of social cognition disorders in the context of a focal or generalized epilepsy syndrome may, thus, help clarify the role of these neuropsychological impairments on disease-related problems (e.g., stigma and isolation) and their relationship with the underlying pathology as well as with possible other yet-unexplored cognitive mechanisms. Specific psychobehavioral implications of social cognition disorders should also be explored. Social maladjustment is often reported in patients with epilepsy, and it is usually poorly explained by the underlying pathology or by psychiatric comorbidities [68,69]. The presence of specific ToM deficits in patients with epilepsy has specific influence on self-appraisal, coping, and overall intelligence level [70,71]. In conclusion, an in-depth assessment of socioemotional processing may also help in planning nonpharmacologic treatment, such as cognitive rehabilitation, psychotherapy, or work/social training, which is extremely useful in coping with daily problems.

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Conflict of interest

We report no conflicts of interest.

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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