

DYSFUNCTIONS OF FACIAL EMOTION RECOGNITION IN ADULT NEUROPSYCHIATRIC DISORDERS: INFLUENCE ON INTERPERSONAL DIFFICULTIES

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Emotional facial expression (EFE) accurate recognition is needed to ensure good interpersonal communication and relationship.

Impaired recognition of emotional facial expressions has been described in many neuropsychiatric disorders. Neuronal circuits dysfunctions and/or inappropriate learning processes could explain these recognition difficulties.

EFE decoding disturbances in adult neuropsychiatric disorders are reviewed.

For some of these disorders, a correlation has been evidenced between poor recognition of EFE and interpersonal difficulties.

Treatment of EFE dysfunctions seems possible but it is not yet known if such a treatment could improve the interpersonal life of patients with neuropsychiatric disorders.

Introduction

Studies initiated in the 1960s by Paul Ekman (Ekman, Sorenson, & Friesen, 1969) and others (Izard, 1968) have shown that nonverbal communication is partly a universal language. The ability to use this language and to understand its meaning is present very early in human beings, pointing to the possibility that brain structures are pre-wired regarding this means of communication (Field, Woodson, Greenberg, & Cohen, 1982). There are other arguments in favour of an innate system: for example, blind children and adults display appropriate Emotional Facial Expressions (EFE) without having ever seen them (Charlesworth & Kreutzer, 1973). Humans are also able to decode quite accurately EFE in apes, pointing to a language partially crossing species (Chevalier-Skolnikoff, 1973; Redican, 1982).

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However, the ability to use this language is not fully mature at birth. Newborns and children must have models in order to refine their knowledge of EFE and of the possible messages they convey (De Sonnevile et al., 2002). Parents' education may have an impact. Grossly inappropriate behaviour by parents as with maltreated or neglected children alters the decoding capacity of children (Dunn & Brown, 1994; Jones, Bowling, & Cumberland, 1997).

Studies of EFE decoding abilities have been made possible through the development of standardised sets of photographs, most of the time issued from the work of Paul Ekman (Ekman & Friesen, 1976). The use of standardised evaluation procedures allows for the study of EFE decoding in the normal population, testing the influence of gender (Hall, 1978), personality (Mufson & Nowicki, 1991), and social position (LaFrance & Henley, 1994).

Overall the ability to accurately decode EFE is not uniformly distributed in the population: there are persons who are particularly skilled regarding this function and others who have difficulties to identify correctly emotions in the face of others. Being female (Hall, 1978), of high social position (LaFrance & Henley, 1994), and with a dominant personality (Hall, Halberstadt, & O'Brien, 1997) are generally considered as factors associated with better skills. Extreme recurrent negative emotional expressions by the parents during childhood (Dunn & Brown, 1994) or maltreatment (Camras et al., 1990) are usually associated with poorer skills in adulthood.

The link between the ability to decode correctly EFE and interpersonal relationships in normal populations has also been studied and results show that the accurate perception of nonverbal language is associated with better social functioning (Boyatzis & Satyaprasad, 1994; Carton, Kessler, & Pape, 1999; Philippot & Feldman, 1990).

A flourishing stream of research has also emerged, with the help of standardised EFE evaluation procedures, in order to localise the processing of EFE in the brain through imaging techniques. Moreover, these EFE evaluation techniques have also allowed the study of pathological populations, comparing them to normal controls on this ability, and tracking the eventual differences in EFE brain processing among populations. We will review results from cerebral lesions patients as well as the main results in neuropsychiatric populations.

Cerebral lesions

Numerous studies testing patients with neurological problems have studied the impact of cerebral lesions on EFE decoding ability. In fact, these studies were the only way to explore the circuits involved in this task before the

emergence of functional cerebral imaging techniques.

The amygdala seems to be an important component although its exact role is not yet fully understood. Most studies have explored the impact of amygdala lesions either in individual patients (Adolphs, Tranel, Damasio, & Damasio, 1994; Anderson & Phelps, 2000; Sprengelmeyer et al., 1999; Young, Hellawell, Van de Wal, & Johnson, 1996) or in groups of patients (Adolphs et al., 1999; Broks et al., 1998; Schmolck & Squire, 2001) and found that these lesions were associated with a deficit in EFE recognition. This deficit was mostly present for the recognition of fear but could also be associated with the recognition of several negative emotions (Adolphs et al., 1994; Adolphs et al., 1999; Anderson & Phelps, 2000; Broks et al., 1998; Schmolck & Squire, 2001). Poor EFE recognition could have important social consequences as patients with amygdala lesions judge persons presented on photographs as more reliable than controls and could therefore be more susceptible to be deceived in social interactions (Adolphs, Tranel, & Damasio, 1998).

Traditionally, the right hemisphere is considered to be prevalent in the decoding of EFE (Etcoff, 1984; Bowers, Bauer, Coslett, & Heilman, 1985; Bowers, Blonder, Feinberg, & Heilman, 1991; Blonder, Bowers, & Heilman, 1991; Adolphs, Damasio, Tranel, & Damasio, 1996; Mandal, Asthana, & Maitra, 1998; Mandal et al., 1999; Anderson et al., 2000; Adolphs, Tranel, & Damasio, 2001) and particularly for negative EFE (Adolphs et al., 1996; Mandal et al., 1998; Anderson, Phelps, Spencer, & Fulbright, 2000; Adolphs et al., 2001). This is consistent with the theory that the right hemisphere is preferentially associated with withdrawal behaviours, while the left hemisphere is involved in approach behaviours (Davidson, Ekman, Saron, Senulis, & Friesen, 1990). Curiously, the supposed predominance of the right hemisphere to process EFE information has received little support from imaging studies (Gur, Skolnick, & Gur, 1994). This could be due to differences in methodology. Imaging studies involve the presentation of photographs but do not ask participants to identify the emotion displayed, while in cerebro-lesion studies participants are asked either to match two EFE presented simultaneously or to identify them (by naming). It is thus conceivable that the perception of EFE implicates different cerebral regions in the two hemispheres, but that the identification of EFE needs the use of a nonverbal lexicon seated in the right hemisphere.

Overall, EFE perception and decoding is based on a multi-component distributed brain system including mostly prefrontal regions and the amygdala, but also parietal and temporal lobes (Streit et al., 2000). The precise role of each component is not yet well understood, but clinical studies clearly show that cerebral lesion of numerous locations can lead to errors in EFE processing.

Parkinson Disease

There are contradictory data about the ability of EFE decoding in Parkinson disease. One study (Jacobs, Shuren, Bowers, & Heilman, 1995) shows a difficulty to process EFE, suggesting that the striatum could have a role in the decoding of EFE as they are implicated in the production of mimics. However, others studies failed to find any disturbances in EFE decoding in patients suffering from Parkinson disease (Adolphs, Schul, & Tranel, 1998; Dujardin et al., 2004). Discrepancies could be due to the influence of depression, frequently associated with Parkinson disease, and not always well controlled for. The study of Adolphs, Schul, and Tranel (1998) is particularly interesting since it shows that EFE mimicry is not necessary to recognise EFE, as Parkinson disease is frequently associated with a rigidity of the muscles of the face. The absence of an important role of mimicry in the recognition of EFE has been confirmed elsewhere (Blairy, Herrera, & Hess, 1999).

Alzheimer disease

Alzheimer patients present difficulties in decoding EFE in addition to the usual cognitive dysfunctions seen in this disease. However, compared with general cognitive ability, it seems that nonverbal emotional processing skills are relatively spared (Bucks & Radford, 2004). When compared to patients with vascular diseases, Alzheimer disease patients seem to perform better, even though no differences emerge between them in their general cognition and visuoperceptual abilities (Shimokawa et al., 2003). Recognition of facial emotions in Alzheimer disease decreases with the progression of dementia and could be related to the degeneration's progression of structures implicated in emotional processing systems (Lavenu & Pasquier, 2005).

EFE decoding impairments could be due to visuospatial dysfunctions rather than to amygdala damage: patients seem more impaired in matching tasks than in labelling tasks (Burnham & Hogevoorst, 2004).

EFE decoding impairments are specifically associated with impaired interpersonal relationships, a specificity not usually found to be associated with standard cognitive measures (Shimokawa et al., 2001).

Huntington disease

Difficulties to decode EFE, but also to recognise faces are present when Huntington disease is clinically apparent (Jacobs, Shuren, & Heilman, 1995; Sprengelmeyer et al., 1996). These patients have more difficulties when pro-

cessing negative emotions, i.e. anger, fear and disgust. Disgust is especially difficult to recognise, both when seeing emotional faces and when hearing the emotional tone of the voice. This relatively specific deficit might be due to a dysfunction of peri-amygdalar and pyriform regions (Sprengelmeyer et al., 1996). A specific deficit for the EFE of disgust is found in pre-clinical forms, i.e., in persons bearing the gene but with no clinical signs yet (Gray, Young, Barker, Curtis, & Gibson, 1997).

Schizophrenia

Schizophrenia is the neuropsychiatric disorder that is most studied regarding the ability to decode EFE. The literature is almost unanimous to describe a severe deficit in particular for negative emotions (Walker, McGuire, & Bettes, 1984; Novic, Luchins, & Perline, 1984; Bellack, Mueser, Wade, Sayers, & Morrison, 1992; Bell, Bryson, & Lysaker, 1997). The difficulty to decode negative emotions could be congruent with a dysfunction of the right hemisphere which is usually considered to more impaired in schizophrenia, and known to be specialised in the processing of negative emotions (Davidson et al., 1990).

Most of the studies report a generalised deficit, i.e. that schizophrenics have difficulties regarding visuo-spatial tasks in general, including the recognition of faces and that EFE decoding impairment is only one facet of a larger right hemisphere functioning problem (Novic, Luchins, & Perline, 1984; Archer, Hay, & Young, 1992; Kerr & Neale, 1993; Borod, Martin, Aler, Brozgold, & Welkowitz, 1993; Archer & Hay, 1994; Mueser et al., 1996). EFE decoding impairments also seem to be linked to negative symptoms in schizophrenia (Kohler, Bilker, Hagendoorn, Gur, & Gur, 2000). However, several studies report impairment in decoding EFE specifically and independently from other cognitive tasks such as the recognition of faces and the guessing of age using facial cues (Cutting, 1981; Heimberg, Gur, Erwin, Shtasel, & Gur, 1992; Edwards, Pattison, Jackson, & Wales, 2001). It seems also that only part of the variance of the results on emotional tests is explainable by cognitive variables (Bryson et al., 1997). Social isolation, lack of social contacts could also contribute to the deficit observed, because the patients would not have sufficient opportunities to exercise their EFE decoding skills.

Schizophrenia is associated with dysfunction of several brain regions, namely prefrontal cortex, thalamus and striatum (Andreasen, 1997). Recognition of EFE in schizophrenia could be impaired if circuits involved in EFE processing were altered (Streit et al., 2001). Hypo-activity of cerebral regions, particularly prefrontal regions (Streit et al., 2001) and amygdalae

(Kosaka et al., 2002), has been described in association with difficulties to decode EFE by schizophrenic patients. Prefrontal cortices are known to be damaged in schizophrenia and are important in the decoding of EFE (Streit et al., 2001). Failure to activate limbic regions during emotional valence discrimination has also been reported (Gur et al., 2002) but did not impact performance probably because the task used was not demanding enough.

The clinical consequences of an EFE decoding deficit in schizophrenia have been investigated using different techniques. There seems to be a relationship between deficits in EFE decoding and social skills in this population (Bellack et al., 1992, Mueser et al., 1996). General social functioning, i.e. the possibility to live an autonomous life, to work, to have good relationships with the family or significant others, is linked to EFE perception skills but not to the prosody recognition or the recognition of faces per se in one study (Hooker & Park, 2002). Another study has found that emotion perception difficulties in general (facial emotion, voice emotion and affect perception) are associated with decreased work functioning and altered possibility to live independently, when patients are followed 12 months after assessment (Kee, Green, Mintz, & Brekke, 2003).

Depression

Most studies have emitted the hypothesis that depressive patients would display a bias towards negative emotions when decoding EFE. This would be congruent with Beck's cognitive conception of depression (Beck, Rush, Shaw, & Emery, 1979). Early negative schemas interacting with personality factors and negative life events could lead to the development of depressive episodes associated with a negative bias in the processing of information, including social information. This biased processing of social information could include a misperception of emotions in the face of others (Bouhuys, Geerts, & Gordijn, 1999). Indeed a negative bias is found in several studies (Mandal & Bhattacharya, 1985; Gur et al., 1992; Bouhuys, Bloem, & Groothuis, 1995; Hale III, Jansen, Bouhuys, & van den Hoofdakker, 1997; Bouhuys, Geerts, & Mersch, 1997; Hale III, 1998; Geerts, & Bouhuys, 1998; Bouhuys et al., 1999).

In Beck's theory, negative cognitions should be responsible for a negative bias in the processing of information. However, a study using emotional states induction techniques in non-depressive subjects has shown that a negative mood could induce a negative perception bias when decoding EFE (Bouhuys et al., 1995). A depressive mood per se could thus induce the negative perception bias contributing probably to the maintenance of the depressive state. When the depressive state is severe, recognition of EFE is global-

ly affected (Feinberg, Rifkin, Schaffer, & Walker, 1986; Zuroff & Colussy, 1986; Persad & Polivy, 1993; Mikhailova, Vladimirova, Iznak, Tsusulkovskaya, & Sushko, 1996; Asthana, Mandal, Khurana, & Haque-Nizamie, 1998). There is usually no relationship between difficulties to recognise EFE and other alterations of other visuo-spatial measures. However, when patients have enduring and severe depressive disorders, there seems to be a generalised deficit like in schizophrenia, i.e., a deficit in general visuo-spatial perception including EFE (Asthana et al., 1998).

What happens when depression remits is not yet known. It seems that EFE decoding is partially improving, but this still has to be confirmed (Mikhailova et al., 1996). One study does not demonstrate an influence of the severity of the EFE deficit on the evolution of depression (Bouhuys et al., 1997), but this study included ambulatory patients who are probably less severely affected. For hospitalised patients, it seems that the severity of the EFE deficit could be linked to a bad prognosis of depression (Hale III, 1998; Bouhuys et al., 1999). Overall, long term studies with repeated measures are needed to further investigate this eventual relationship.

Alcoholism and drug dependence

Several studies have evidenced that alcoholism is associated with impaired EFE decoding encompassing both positive and negative emotions. Recently detoxified alcoholics decode less accurately EFE than normal controls (Oscar-Berman et al., 1990; Philippot et al., 1999; Kornreich et al., 2001a; 2001b) and they overestimate the intensity of EFE (Philippot et al., 1999; Kornreich et al., 2001a; 2001b). They also need more intensity of non-verbal signal to perceive an expression as being present (Frigerio, Burt, Montagne, Murray, & Perett, 2002), and they seem on some studies to display different patterns of interpretation of emotion as compared to controls, with a specific bias for hostile emotions (Philippot et al., 1999; Frigerio et al., 2002; Townshend & Duka, 2003).

This difficulty to decode EFE is present even in patients abstinent for at least 2 months (Kornreich et al., 2001a). This observation might imply that these deficits are either due to the influence of chronic alcohol consumption on the brain, that this influence is not easily reversed and could even be permanent, or that the deficits could be present before the development of alcoholism.

Drug addicts using opiates also display an EFE decoding deficit but less so than alcoholics (Kornreich et al., 2003; Foisy et al., 2005). Interestingly, polydrug addicts with alcohol and opiate dependence antecedents have similar results as alcoholics, which are worse than for opiate addicts not using

alcohol. This seems to indicate that chronic alcohol consumption is deleterious to EFE decoding function independently of potential impact of characteristics pre-existing the development of the addiction.

Finally, interpersonal difficulties seem to be partly mediated by EFE decoding deficits (Kornreich et al., 2002). Would this relationship been confirmed, it could have important clinical implications, as interpersonal problems are a major cause of relapse in alcoholism (Marlatt, 1996).

Anorexia Nervosa

Anorexic patients seem to have difficulties recognising both EFE and emotions in voices. This seems most marked for negative emotions in faces and for both positive and negative emotions in voices. These results seem not to be influenced by depressive scores and could contribute to poor interpersonal communication and lack of empathy in this group of patients (Kucharska-Pietura, Nikolaou, Masiak, & Treasure, 2004).

Obsessive compulsive disorder

The literature provides contradictory results regarding the ability of OCD patients to decode EFE. OCD patients recognise EFE as well as controls with the exception of disgust (Sprengelmeyer et al., 1997). These authors suggest that the caudate does play a part in the processing of disgust as they have found similar results in Huntington disease, a disease characterised by lesions in this region. However, poor recognition of disgust is not found in all studies about EFE in OCD (Buhlman, McNally, Etcoff, Tuschen-Caffier, & Wilhelm, 2004). Further, it is difficult to understand the clinical relevance of this finding even for washing patients. We would expect an overestimation of disgust or that other EFE would be confounded with disgust and this is not found in the study by Sprengelmeyer et al. (1997), where disgust is only poorly recognised.

Social Phobia

Shy children misclassify anger expressions interpreting them as disgust and classifying neutral expressions as sadness (Battaglia et al., 2004), suggesting a recognition problem in childhood improving perhaps in adulthood. This data seems relevant to social phobia as shyness in childhood is a predisposing factor to social phobia in adulthood (Biederman et al., 1990;

Hirschfeld et al., 1992).

Social phobics do not seem to present a deficit in the recognition of EFE, nor evaluative biases when decoding EFE (Philippot & Douilliez, 2005). However, they present attentional biases (Mogg & Bradley, 1998). As demonstrated by Mogg, Philippot, and Bradley (2004), when presented with expressive faces, social phobics present an initial selective attention to angry faces. This bias disappears at longer stimulus exposure. Further, imagery studies have shown an increased amygdala activation to angry and contemptuous faces in social phobia suggesting that this kind of faces is considered to be particularly threatening to these patients (Stein, Goldin, Sareen, Zorilla, & Brown, 2002).

Antisocial personality disorder

The literature does not provide unequivocal results for EFE decoding capacity in antisocial personality disorder. For example, a study by Pham and Philippot (in press) shows that the level of psychopathy among prison inmates was unrelated to their ability in EFE decoding. However, prison inmates, whether psychopaths or not, were less accurate than controls with no criminal history. In contrast, Kosson, Suchy, Mayer, and Libby (2002) found psychopaths to be less accurate than non psychopaths. Similarly, in a study using event related potentials, Campanella, Vanhoolandt, and Philippot (2005) found that students with psychopathic tendencies present an emotional deficit, both at the behaviour and brain activity level, and in the processing of facial expression. Still, overall, processing of verbal emotional material is probably more impaired than EFE processing in this population (Kosson et al., 2002).

Borderline personality disorder

Here again, the literature provides contradictory results: good EFE perception was found in one study (Wagner & Linehan, 1999) and accuracy problems in another (Levine, Marziali, & Hood, 1997). These discrepancies might be due to a difficulty to isolate borderline patients without co-morbidities like depression or substance abuse. One imagery study shows that borderline patients display hyperactivity of left amygdala compared to controls when viewing EFE, suggesting that processing of information conveyed by faces may be different in this population (Donegan et al., 2003).

Aetiology of EFE decoding in neuropsychiatric disorders

There are most certainly different mechanisms responsible for the presence of EFE decoding difficulties in neuropsychiatric disorders. Cerebral circuits involved in the decoding of EFE, i.e., mainly in amygdala prefrontal, temporal and parietal regions, may be lesioned, leading to decoding difficulties. It is certainly the main mechanism in cerebral lesions, Huntington disease and Alzheimer disease.

Learning problems could also be involved. For example, abused, maltreated or neglected children have difficulties to be accurate EFE readers (Camras et al., 1990; Dunn & Brown, 1994; Jones et al., 1997; Pollack, Cicchetti, Hornung, & Reed, 2000), probably because the emotional education has been badly flawed or incoherent. This mechanism could be present in borderline and antisocial personality disorders.

Both neuronal circuits dysfunctions and an inappropriate learning process could explain EFE decoding problems in pathologies such as alcoholism: alcoholics have frequently a disturbed childhood possibly leading to difficulties similar to neglected or maltreated children. But alcohol has also a toxic effect on the brain and could damage circuits involved in EFE decoding. Similarly, schizophrenic patients have brain abnormalities on the one hand, but have also a poor social life and a paucity of contacts on the other hand and may therefore have difficulties to exercise their EFE decoding abilities.

Mood disturbances and possibly anxiety problems are probably responsible for biased evaluation, even if brain circuits are intact and EFE related knowledge acquired during childhood and adolescence adequate.

Whether EFE dysfunction precedes the apparition of some neuropsychiatric disorders and constitutes a vulnerability factor or appears as a consequence of these disorders is not yet clear and should be investigated further.

Clinical relevance of EFE decoding dysfunction in neuropsychiatric problems

Psychological health may be reflected by three general areas of functioning: intrapersonal, interpersonal and social role performance (Sheffield, Carey, Patenaude, & Lambert, 1995). Within this framework, intrapersonal refers to affective, cognitive, and behavioural/ physiological indices of functioning; interpersonal refers to relationships with others; and social performance refers to the capacity to contribute meaningfully in a larger social context.

Regarding interpersonal functioning, nonverbal behaviour plays important functions in interaction and social regulation (Patterson, 1991), in emotion

contagion (Hess, Philippot, & Blairy, 1999) and more generally is an important medium for the communication of inner feelings and intentions (Cacioppo, Petty, Losch, & Kim 1986). The mastery of nonverbal behaviour is a critical competence for effective and harmonious social functioning (Feldman, Philippot, & Custrini, 1991). The link between EFE decoding and interpersonal relationships has been well studied in the general population: good EFE readers are the ones having the best interpersonal abilities and relationships (Carton et al., 1999; Rosenthal, 1979). They are better perceived by others, being described as open, empathic, attentive and caring (DiMatteo, Friedman, & Taranta, 1979; Funder & Harris, 1986). Therefore EFE decoding difficulties should be associated with impaired interpersonal relationships in neuropsychiatric disorders. Although intuitively appealing, we have only limited indications about the potential link between EFE decoding deficits in neuropsychiatric disorders and interpersonal relationships disturbances, and this is mostly due to a paucity of studies about this topic.

When cerebral lesions are associated with nonverbal signals decoding problems, there seems to be a negative impact on interpersonal relationships (Hornak, Rolls, & Wade, 1996). In Alzheimer disease, EFE decoding deficit is associated with interpersonal difficulties, an association not found with other cognitive measures (Shimokawa et al., 2001). There is also an association between EFE decoding abilities and social functioning in schizophrenia (Bellack et al., 1992; Mueser et al., 1996), social functioning being measured either through observed behaviours in role playing or through staff and family evaluation. General social functioning (the possibility to live independently, to work, to develop satisfying relationships with the family and significant others) is also associated with EFE recognition but not with face recognition in this population (Hooker & Park, 2002).

Finally, in alcoholism, a link between EFE decoding difficulties and poor interpersonal relationships has been found using a self-report measure through the use of a questionnaire (Kornreich et al., 2002).

Possibility to treat EFE decoding dysfunctions

Information about the possibility to improve the ability to read accurately EFE in the general population on the one hand and in patients on the other hand is limited. In one study, thirty children of elementary schools participated in several sessions teaching to discriminate and identify EFE. The ability to read emotions in facial expressions significantly improved in the intervention group compared to the control group. Improvement was associated with lower social anxiety and higher self-worth in girls but not in boys

(Grinspan, Hemphill, & Nowicki, 2003). Another study realised with a nursing staff caring for demented persons has shown that training to recognise nonverbal signals enhanced staff's satisfaction, diminishing negative affect without having an impact on demented persons' symptomatology (Magai, Cohen, & Gomberg, 2002). A computer programme has been developed in order to increase the ability to read EFE by Asperger and Autistic children. There was an improvement seen after the teaching programme on computer scores but no evaluation regarding an eventual impact on the functioning in the reality (Silver & Oakes, 2001). A study with a limited number of adults with acquired brain injury has shown that it was possible to increase the ability to decode EFE using a computer assisted teaching programme (Guercio, Podolska-Schroeder, & Rehfeldt, 2004). Finally, a training programme did improve facial affect recognition impairments in patients with schizophrenia (Frommann, Streit, & Wölwer, 2003). Again, results were only evaluated through computer scores and it is difficult to generalise these results to real life situations.

Conclusions

EFE decoding deficits are observed in numerous neuropsychiatric disorders and are not specific to a particular one. The aetiologies do probably vary depending of the type of disorder: damaged cerebral circuits are involved in brain lesions, Parkinson disease, Alzheimer disease, Huntington disease, mental retardation, autism, Asperger disease and probably in schizophrenia and alcoholism as well. Inappropriate learning process does probably have an impact in alcoholism and drug dependence as well as in some personality disorders. Mood disorders, and perhaps some anxiety disorders are associated with dysfunctional EFE decoding probably through interpretation bias.

As interpersonal relationships' quality is associated with appropriate EFE decoding in the normal population, it would be logical to infer that EFE decoding dysfunctions in neuropsychiatric disorders are partly responsible for interpersonal difficulties found in these pathologies. There are some indications that it is indeed the case in Alzheimer disease, brain lesions, schizophrenia and alcoholism but much remains to be done to explore this topic further in the above-mentioned pathologies as well as in others.

Evidence about the possibility to improve EFE decoding abilities is scarce even if there are some indications that it can be done in normal populations and in several pathological populations such as in brain-lesion patients and in Asperger disease. However, it remains to be demonstrated that an improvement of this skill through exercise in neuropsychiatric disorders has a positive influence on real life interpersonal relationships of these patients.

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