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Review

Theory of Mind in normal ageing and neurodegenerative pathologies

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ABSTRACT

This paper reviews findings in three subcomponents of social cognition (i.e., Theory of Mind, facial emotion recognition, empathy) during ageing. Changes over time in social cognition were evaluated in normal ageing and in patients with various neurodegenerative pathologies, such as Alzheimer's disease, mild cognitive impairment, frontal and temporal variants of frontotemporal lobar degeneration and Parkinson's disease. Findings suggest a decline in social cognition with normal ageing, a decline that is at least partially independent of a more general cognitive or executive decline. The investigation of neurodegenerative pathologies showing specific deficits in Theory of Mind in relation to damage to specific cerebral regions led us to suggest a neural network involved in Theory of Mind processes, namely a fronto-subcortical loop linking the basal ganglia to the regions of the frontal lobes.

1. Introduction

Social cognition has been defined as the ability to interpret and predict others' behavior, based on their beliefs and intentions, and to interact in complex social environments and relationships (Baron-Cohen, 2000). The ability to understand and respond to the emotional content and cues present in the environment and the ability to remember emotional information are integral parts of social cognition (Adolphs, 2003; Grady and Keightley, 2002). Social cognition guides both automatic and volitional behavior, being composed of a variety of cognitive, emotional, and motivational processes that modulate behavioral responses. Memory, decision-making, attention, motivation, and emotion are all prominently recruited when socially relevant stimuli elicit behavior (Adolphs, 2009). Most empirical studies in social cognition have focused on developmental diseases (e.g., autism, Asperger's syndrome), or analyzed the consequences of acquired lesions (e.g., brain injury, stroke) on social cognition. Less is known about the integrity of social cognition in elderly individuals (see Table 1 for a description of studies on social cognition in normal ageing).

Due to the increasing prevalence of dementia with ageing, early diagnosis is important in enabling better support for these patients. The assessment of social cognition during ageing appears fundamental to this diagnostic approach. This point of view is supported by the American Psychiatric Association, which, by the intention to include impairment of social cognition in the new diagnostic criteria for "Major Neurocognitive Disorder" (i.e., former "dementia") in the DSM-V (Diagnostic and Statistical Manual of Mental Disorders-V), demonstrates an awareness of the increased vulnerability of social cognition mechanisms with ageing and highlights the importance and necessity of addressing social cognition in patients suspected of dementia. Assessment tools in social cognition may be particularly sensitive in the diagnosis of pathological ageing and may be valuable in guiding the diagnosis toward a particular neurodegenerative disease.

1.1. Theory of Mind

The most representative mechanism of social cognition is "Theory of Mind" (ToM), which designates the ability to attribute the full range of mental states (both goal and epistemic states) to ourselves and to others, and to use these attributions to make sense of and predict behavior. The term "Theory of Mind", was first introduced by Premack and Woodruff (1978), but there is a set of synonyms frequently employed, namely "mind reading", "mentalizing" and "mental state attribution". One recent model (Shamay-Tsoory et al., 2010) distinguishes cognitive (or "cold") from affective (or "hot") sub-processes of ToM (see Fig. 1).

Cognitive ToM refers to the ability to make inferences about the cognitive states, beliefs, thoughts, intentions and motivations of other people (Brothers and Ring, 1992; Coricelli, 2005), while affective ToM refers to the ability to infer the feelings, affective states and emotions of others (Brothers and Ring, 1992). Cognitive ToM can be assessed by a first- and second-order false belief task

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Table 1
Studies on social cognition in normal ageing.

Authors (published)	Mean age (years)	SC function assessed	Main SC results	
Happé et al. (1998)	21	Cognitive ToM	First- and second-order FBT	>
	73			
Maylor et al. (2002)	19		First- and second-order FBT	<
	67			
	81			
Sullivan and Ruffman (2004)	30		First-order FBT	<
	73		Video task	<
			Emotion labeling	<
McKinnon and Moscovitch (2007)	~20.5		First-order FBT	=
	~76.8		Second-order FBT	<
Duval et al. (2010)	23.8		Visual attribution of intention task	–
	52.5			
	70.1			
Charlton et al. (2009)	55.8		First- and second-order FBT	<
	65.6			
	75			
	83.8			
MacPherson et al. (2002)	28.8	Affective ToM	Faux-Pas Test	=
	50.3			
	69.9			
Phillips et al. (2002)	29.9		RME Test	<
	69.2			
Bailey and Henry (2008)	19.5		RME Test	<
	72.2			
Phillips et al. (2002)	29.9		RME Test	<
	69.2			
Slessor et al. (2007)	20		RME Test	<
	67			
Pardini and Nichelli (2009)	<55		RME Test	
	>55			
Duval et al. (2010)	23.8		Basic emotions	=
	52.5		Complex emotions	<
	70.1			
Keightley et al. (2006)	25.7	Emotion recognition	Negative emotions (fear, sadness)	<
	72.5		Positive emotions	=
McDowell et al. (1994)	17–22		Negative and neutral emotions	<
	65–90		Positive emotions	=
Chaby and Narme (2009)	50–70		Facial identity	<
			Emotion facial expression	<
Brosgole and Weisman (1995)	<45		Negative emotions (anger)	<
	>45			
Calder et al. (2003)	25		Negative emotions (except disgust)	<
	65			
MacPherson et al. (2006)	Younger adults		Negative emotions (sadness)	<
	Older adults			
MacPherson et al. (2002)	Younger adults		Sadness	<
	Older adults		Happiness, anger, disgust, fear, surprise, contempt	V
Malatesta et al. (1987)	Younger adults		Anger	<
	Older adults			

SC: social cognition; ToM: Theory of Mind; FB: false belief; FBT: false beliefs task; RME: Reading the Mind in the Eyes; (<): impaired, (=): preserved, (>): improved.

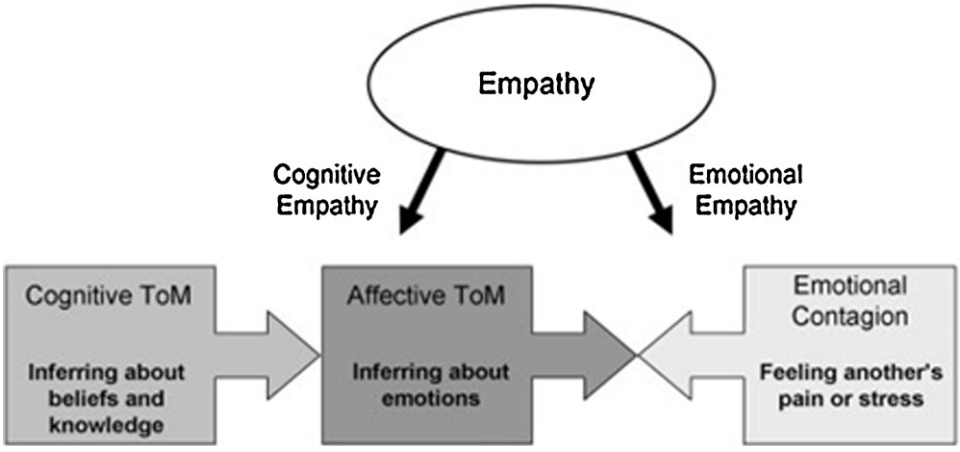

Fig. 1. Model of the relationships among cognitive ToM, affective ToM and empathy.
Source: From Shamay-Tsoory et al. (2010).

(see Appendix A for a task description), while affective ToM can be assessed using the Faux-Pas Test (Baron-Cohen et al., 1999) (see Appendix A for a task description). According to this model, cognitive ToM is a prerequisite for affective ToM, which also requires intact empathy processing.

ToM capacities first manifest themselves during early infancy (i.e., around 18 months of age), when children engage in shared attention and proto-declarative pointing (Baron-Cohen, 1995). Before that age, children are not able to decouple pretense from reality. Between 18 and 24 months, children begin to understand the mental state of "pretend" (Leslie, 1987), and, by age 2 years, they begin to have a firm grasp of the mental state of desire (Wellman and Woolley, 1990). Between ages 3 and 4 years, children can understand that another person may hold false beliefs (Gopnik and Astington, 1988; Johnson and Wellman, 1980; Wimmer and Perner, 1983). Prior to this age, a child does not understand that other people can hold beliefs about the world that differ from his or her own. Between ages 6 and 7 years, children begin to pass more advanced tests that examine "belief about belief" and begin to understand that other people can also represent mental states (Perner and Wimmer, 1985). Complex social skills first appear between ages 9 and 11 years, when children develop further ToM abilities, such as recognizing a social "faux-pas" or wrong behavior. Understanding that a faux-pas has occurred requires the representation of two mental states: that the person saying something does not know that he/she should not say it and that the person hearing it would feel insulted or hurt. Thus, there is both a cognitive component and an empathic affective component in this particular task (Baron-Cohen et al., 1997).

1.2. Empathy

Empathy, a second component of social cognition, is mainly implicated in prosocial behavior, morality and the regulation of aggression (Eisenberg and Eggum, 2009). Moreover, the term empathy is applied to a broad spectrum of phenomena, from feelings of concern for other people that create a motivation to help them, experiencing emotions that match another's, knowing what the other is thinking or feeling, to blurring the line between self and other (Hodges and Klein, 2001). Due to the complexity of the phenomenological experience of empathy, this construct has been broken down into component processes (Decety, 2010) (see Fig. 2).

These different components are intertwined and contribute to different aspects of the experience of empathy. Affective arousal is the first component that appears during development, having evolved to differentiate automatically hostile from hospitable stimuli and to organize adaptive responses to these stimuli. Emotion understanding develops later, beginning to be mature around age 2–3 years. This component largely overlaps with ToM-like processing. Emotion regulation enables the control of emotion, affect, drive and motivation. This component develops throughout childhood and adolescence, and parallels the maturation of execution functions. Humans also have the capacity to appraise and reappraise emotions and feelings; all of goals, intentions, context, and motivation are likely to play feed-forward roles in how emotions are perceived and experienced. Thus, empathy is not only a passive affective phenomenon; empathy is not simply a resonance with the emotions of others.

1.3. Facial emotion recognition

The recognition of emotions in faces is an essential component of social cognition. Social cognition has been defined as "the processing of any information which culminates in the accurate perception of the dispositions and intentions of other individuals" (Brothers, 1990, 2002). Among the information used for the recognition of dispositions and intentions are identity, category of posture, direction of movement, quality of vocalization, and facial expression (Brothers, 1990). According to this definition, emotion recognition and ToM are two core components of social cognition.

Facial expressions signal important information about the internal states of others and the external events that may have elicited those expressions (Ekman, 1997). Individuals use this information to guide their social behavior. Traditionally, the recognition of identity and emotion in human faces involves distinct processes linked by an initial visual processing. Recently, this partition has been shown to be an oversimplified model. The ability to recognize emotions from facial expressions is correlated with affective ToM, or the ability to attribute to others' mental states, beliefs, intents, and desires. Moreover, the areas of activation of these two components of social cognition overlap.

2. Cognitive ageing and social cognition

2.1. Cognitive ToM

Pioneering research in 1998 addressed the modularity/domain-specificity debate in a novel way by examining, for the first time, older adults' performance on ToM tasks (Happé et al., 1998). Two groups of participants, of mean ages 21 and 73 years, were asked to read short stories and answer questions based on their memories of each. Half of these were ToM stories involving double bluffs, mistakes, persuasions, and white lies, and half were control, or non-ToM stories. The ToM stories required participants to make inferences about what a protagonist understood about the mental state of another individual by decoding subtle cues to sarcasm, deception, and social rule violation. Although older participants read both the ToM and the control stories at slower rates than the younger participants, the older participants showed higher performance, as determined by numbers of questions answered correctly, on the ToM stories, whereas the two groups performed equally well on the control stories. These novel findings indicated that performance on ToM tasks remains intact and may even improve over the later adult years.

In contrast, a later study found that, although there were no significant age differences in performance on the non-ToM stories, younger participants (mean age, 19 years) performed better than groups of mean ages 67 and 81 years on task conditions for ToM assessment (Maylor et al., 2002). When the cognitive charge was reduced, the group of mean age 67 years performed at the level of those of mean age 19 years, whereas the group of mean age 81 years showed continued impairment. Similar results have been reported (Duval et al., 2010), in that younger and middle-aged participants were significantly more efficient than older participants on the ToM condition of a ToM task (i.e., the visual attribution of intention task; Brunet et al., 2000).

To clarify these discrepancies, as to whether ToM abilities improve or deteriorate with ageing, Sullivan and Ruffman (2004) used the stimuli first described by Happé et al. (1998). Although their results (seemingly to those of Maylor et al., 2002) show that ToM skills decline with age, the two age groups did not differ significantly when fluid intelligence (i.e., pure, not knowledge based thinking ability, involving abstract and novel reasoning) was considered. This result is consistent with the idea that an age-related decline in social understanding may be partially mediated by declining fluid intelligence. Nevertheless, the ToM stories still required second-order reasoning abilities, whereas the control stories did not. This was one reason for including two measures of social reasoning that placed reduced demands on working memory (i.e., a task assessing the ability to decode thoughts and feelings from short, dynamic video clips and an emotion recognition task).

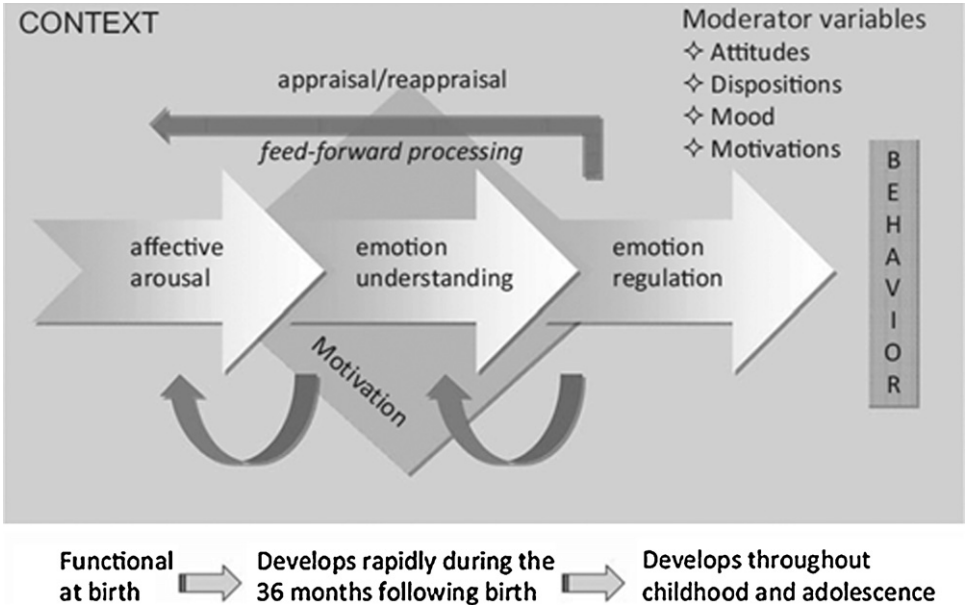

Fig. 2. Schematic illustration of the macrocomponents involved in human empathy

Source: From Decety (2010).

Using these new tasks showed a decline in social understanding in elderly participants, even after allowing for fluid and crystallized intelligence (i.e., the disposal of true and important knowledge and the intelligent use of this knowledge). Hence, changes in social understanding are not a simple function of domain-general changes in cognitive abilities; in other words, it is not reducible to a fluid deficit. Moreover, older participants performed less well on the Tom's taste Test than did younger and middle-aged participants. This ToM test assesses, by means of 16 cartoons drawings, the ability of an individual to judge the preference of another person in a given context, based on the content of his or her thoughts. This result suggests that performance on ToM tests declines significantly with age (Duval et al., 2010).

A more recent study confirmed the results of Maylor et al. (2002) and Sullivan and Ruffman (2004) by highlighting a negative association between performance on a ToM test and age (Charlton et al., 2009). Participants' age ranged over four decades (i.e., 50–89 years) and a subset of Happé's Strange Stories (Happé et al., 1998) was employed in testing. The authors found that participants' performance on this ToM task correlated negatively with age, whereas there was no such relationship with the control stories.

The effects of ageing on ToM were also investigated by focusing on first- and second-order ToM tasks separately (McKinnon and Moscovitch, 2007). Age-related declines in ToM were found when stories were used to assess complex second-order ToM, requiring participants to consider the thoughts of two different characters. In contrast, first-order ToM, which involved consideration of only one character's perspective, was not impaired with age. This could indicate that, rather than showing a specific deficit in the ability to represent mental states, older adults show a decline in the domain-general resources required for more complex ToM judgments (Slessor et al., 2007). These results are in agreement with earlier findings, showing that older adults are impaired on ToM tasks with high central processing demands (e.g., Maylor et al., 2002) (see Section 2.4 for a discussion of the cognitive functions that may mediate ToM performance).

Several hypotheses may explain these contradictory results: First, the impaired performance of the elderly reported by Maylor et al. (2002) may be due to the onset of dementia. Although this study excluded participants with a definite diagnosis of dementia, it did not test for dementia in study participants. To safeguard against this risk, however, Sullivan and Ruffman (2004) administered a screening test, the Mini Mental State Examination (MMSE; Folstein et al., 1975), to all participants. Thus, it is unlikely that the discrepancy was due to the onset of neurodegenerative pathology.

These discrepancies may also have been due to differences in study subjects. For example, the elderly participants in one study (Happé et al., 1998) may have been slower to show cognitive deterioration than in the other study (Sullivan and Ruffman, 2004), making the former a relatively gifted group of elderly people. Indeed, since the IQ of the participants was not measured, the elderly group, with a mean education of 14 years, 7 months, may have been more intelligent than the younger group (Happé et al., 1998). Further, Sullivan and Ruffman (2004) regarded their results as validated, since the participants in that study displayed the typical cognitive profiles of elderly individuals. Globally, their fluid abilities were relatively impaired whereas their crystallized abilities were intact, although a proportion of elderly individuals (between 13% and 33%) exhibited unimpaired social understanding on each task. These findings suggest that at least some elderly individuals demonstrate preserved or superior ToM abilities.

2.2. Affective ToM

The effects of ageing on "affective" ToM were assessed in three age groups, of mean ages 28.8, 50.3 and 69.9 years, by comparing their performance on an affective ToM task, namely the Faux-Pas Test (MacPherson et al., 2002). Although age did not significantly affect any of these faux-pas indices (MacPherson et al., 2002), other studies did not confirm these results. Indeed, affective ToM is also frequently assessed by another, visual task, namely the Reading the Mind in the Eyes (RME) Test (Baron-Cohen et al., 2001), in which mental state judgments must be made about pictures of the eye region (see Appendix A for a task description). Several studies have shown that older adults performed more poorly than younger adults on the RME Test (Bailey and Henry, 2008; Phillips et al., 2002; Slessor et al., 2007). Moreover, this decline in ToM capacity became statistically relevant after age 55 years (Pardini and Nichelli, 2009).

A task inspired by the classical RME Test consisted of the presentation to participants of 20 black-and-white photographs of the

eye region of a female actor who was asked to produce different facial expressions (Duval et al., 2010). Ten of the photographs depicted "basic emotions" (i.e., happy, sad, angry, afraid, surprised, and disgusted) and 10 depicted "complex emotions" (e.g., guilty, thoughtful, admiring, flirtatious). Although the difference did not reach statistical significance, age had a deleterious effect on recognizing complex emotions.

2.3. Emotion recognition

Subtle alterations in the recognition of facial identity and emotional facial expression have been observed, beginning at age 50 years and increasing after 70 years (Chaby and Narme, 2009). Indeed, when older and younger adults were asked to identify the emotion portrayed by facial expressions, the two groups had equal ability to identify happy expressions but older adults had significantly greater difficulties identifying negative and neutral expressions (McDowell et al., 1994). This finding, that difficulty identifying negative facial expressions increases with age, has been replicated by other researchers (Brosgole and Weisman, 1995; Calder et al., 2003; MacPherson et al., 2006), although one study, in which participants were exposed to color photographs of faces expressing different emotional states (e.g., happy, sad, angry, disgusted, frightened, surprised, and contempt; Matsumoto and Ekman, 1988) (see Appendix A for a task description), found that age-related impairment was restricted to sadness, but not to any of the other emotions (MacPherson et al., 2002). Other studies have also reported that the perception of anger (Malatesta et al., 1987) shows specific age-related changes. A study in which participants of mean ages 25.7 and 72.5 years were exposed to color photographs (Matsumoto and Ekman, 1988), found that, while the older group was slower, the two groups were equally accurate in identifying the emotional valence (i.e., positive, negative or neutral) of facial expressions, with the age difference in reaction time being largest for negative faces (Keightley et al., 2006). Further analysis showed that the older adults were significantly less accurate at identifying specific facial expressions of fear and sadness but were as accurate as younger adults in labeling happy, surprised, and neutral faces. These results suggested that age influences the judgment of negative facial expressions, as well as the time or effort needed to carry out the processing of these stimuli, while not affecting the processing of positive faces. A recent meta-analysis showed that older adults find facial expressions of anger, sadness, and fear particularly difficult to identify compared with young adults. In addition, older adults were less able to identify happy and surprised faces, but the magnitude of these difficulties was substantially smaller (Ruffman et al., 2008).

A possible explanation for the dissociation between recognition of positive and negative emotions may be provided by functional neuroimaging. Indeed, viewing faces with negative emotional expressions is associated with increased activity in the amygdala in younger adults (Anderson et al., 2003; Morris et al., 1998), but this activity is reduced in older adults (Gunning-Dixon et al., 2003; Iidaka et al., 2002).

2.4. Correlated deficits

Given the increased vulnerability of the frontal lobes in ageing (see Section 3.2), the observed ToM deficits may be associated with the relationship of executive functions, with neuroanatomical correlates within the frontal lobes, to ToM. Social reasoning skills may require a regulatory mechanism, allowing an individual to adopt the subjective perspective of another (Decety and Jackson, 2004; Eslinger, 1998; German and Hehman, 2006). Executive selection processes are necessary to determine the most appropriate mental state attribution from a number of potential candidates (German and Hehman, 2006). In addition, the self-perspective is regarded as the cognitive "default mode", driven by the automatic link between perception and action (Decety et al., 1997). Therefore, to evaluate another's perspective, some form of active inhibitory mechanism must regulate the prepotent self-perspective. This is particularly interesting because with cognitive ageing, individuals have been shown to suffer from declines in a range of executive capacities including aspects of inhibitory function (Bailey and Henry, 2008; Hasher and Zacks, 1988; Kramer et al., 1994). Older adults showed greater impairment on a high- than on a low-inhibition condition ToM task (Bailey and Henry, 2008). Furthermore, of the various aspects of cognitive functioning that were assessed, including memory, mental flexibility and cognitive speed, only cognitive disinhibition mediated age-related differences in ToM, suggesting that inhibitory control is an important mediator of ToM in late adulthood.

The eventual influence of higher intellectual functioning has been investigated directly with tests relating to intelligence. For example, when younger and older adults were matched on the vocabulary subtest of the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981), the older group performed more poorly on a composite ToM measure but on only one individual measure (Saltzman et al., 2000). Significant correlations have been observed between ToM and vocabulary, information processing speed and executive function; when controlling for these cognitive functions, the association between ToM and age remained (Maylor et al., 2002). In a subsequent test of the executive hypothesis in two groups, of mean ages 20 years and 78 years, neither group showed specific difficulties in reasoning about mental state as compared with non-mental state content when task inferential complexity was kept constant (German and Hehman, 2006). However, manipulations that systematically increased executive performance demands within belief–desire reasoning caused systematic decreases in task performance in both groups, although the effects of increasing executive demands were disproportionately greater in the older group. These results are consistent with other recent findings, showing that cognitive ToM scores were primarily correlated with performances on executive tests (Duval et al., 2010).

Similarly, rather than exhibiting a decline in social cognition *per se* with age, older individuals may present with reduced attention or working memory. These functions are mutually dependent, sharing (in part) the same neural substrates, including the dorsolateral prefrontal cortex (e.g., Baddeley, 1986; MacDonald et al., 2000). Indeed, functional neuroimaging studies have revealed age-related alterations in activity patterns within the prefrontal cortex during the conduct of working memory, encoding, and attentional tasks; activity is frequently less in older compared to younger adults (for a review, see Cabeza and Nyberg, 2000). Such declines in attention and/or working memory with ageing could result in difficulties in attentional control, which is the goal-driven allocation of attention toward processing of task-appropriate stimuli (here, social in nature) (Luks et al., 2007). However, some studies have found that the orienting network, one of the attention networks described by Posner and Petersen (1990) and which is involved in selection of information, is spared upon ageing (Zhou et al., 2011). This result is similar to those of previous studies suggesting that orienting of attention remained intact with age (Fernandez-Duque and Black, 2006; Hartley, 1993; Jennings et al., 2007). Moreover, Sullivan and Ruffman (2004), by employing two new measures of ToM which placed reduced demands on working memory still found an age-related decline in social cognition, indicating that ToM impairment associated with ageing is at least partially independent of an eventual decline in working memory.

Age-related declines have been observed in the detection of the most subtle differences in gaze aversion as well as in the ability

to engage in joint attention by following gaze cues, differences that could not be attributed solely to age-related impairments in visual perception and visual attention (Slessor et al., 2008). A mind-reading model has been proposed, with a specialized system responsible for eye-gaze perception, called the Eye Direction Detector (EDD; Baron-Cohen, 1995). Both eye-gaze detection and the ability to follow the gaze of others are thought to be precursors to ToM; therefore, deficits in these abilities may underlie the age-related declines previously found in more complex components of social perception such as ToM and the perception of threat (Ruffman et al., 2006). Since differences in gaze direction have also been found to be an important influence on the emotion perception of younger adults (Adams and Kleck, 2003, 2005), age-related reductions in eye-gaze detection may also have implications for older adults' emotion recognition.

Overall, these studies objectified a decline in ToM performances with ageing. Although ToM and executive functions can deteriorate independently, executive functions have been conceptualized as a "coopted" system, parallel to a "core" ToM system, which is necessary to succeed at least in particular variants of ToM tasks. That is, the decline in ToM with ageing could be mediated by age-related alterations in executive functions. Nevertheless, elderly individuals experience a decline in social understanding that is, at least partially, independent of a general decline in fluid intelligence or in executive functions (e.g., Duval et al., 2010; Maylor et al., 2002; Sullivan and Ruffman, 2004).

The reasons for the discrepancies among studies of ToM abilities during normal ageing remain unclear. Indeed, previous studies examining age-related changes in ToM have produced a contradictory pattern of results. However, these studies have a number of methodological issues. For example, the ToM tasks used in these studies have often been subject to ceiling effects (e.g., German and Hehman, 2006; Keightley et al., 2006; MacPherson et al., 2002; McKinnon and Moscovitch, 2007; Saltzman et al., 2000). Another major difference among studies was the use of regression across age (e.g., Charlton et al., 2009) in contrast to analyses directly comparing the performance of younger and older individuals (e.g., Happé et al., 1998; Sullivan and Ruffman, 2004). Moreover, some studies have had small samples, from 8 to 20 elderly participants (German and Hehman, 2006; Happé et al., 1998; McKinnon and Moscovitch, 2007; Saltzman et al., 2000), and some assessed small numbers of items (German and Hehman, 2006; Keightley et al., 2006; Maylor et al., 2002), raising questions about the reliability of their findings.

To summarize, different ToM measures assess different aspects of social understanding, which may be differentially affected by age, making it essential to assess performance on a range of ToM tasks in the same participants. It seems particularly important to gain more information about the effects of age on the analysis of dynamic social stimuli, such as the video tasks described by Sullivan and Ruffman (2004), as this is more akin to real-life situations and interactions (Saltzman et al., 2000).

3. Cerebral ageing and social cognition

3.1. Results in animals

Animal models may provide insight into the neural mechanisms thought to be involved in social cognition and its possible decline with age in humans, although animals do not seem to possess social cognition (i.e., ToM or emotional recognition) as defined and discussed in this review. Behavioral studies in rats indicated that emotional reactivity increases with age, resulting in age-related reduction in social interactions. For example, older rats spent less time in active social interaction than did younger animals (Salchner et al., 2004). Also, both exploratory behavior and investigation time were reduced in older compared to younger mice (Euteneuer et al., 2009). Furthermore, although evaluating memory rather than social cognition itself, some studies of social behavior in rodents have focused on social recognition memory. Although some works found subtle age-related changes in such memory (Markham and Juraska, 2007), others described a more pronounced age-associated effect, with older mice spending as much time investigating the same juvenile mouse during the recognition phase as during the first encounter, indicating disruption of social recognition ability (Prediger et al., 2005; Rial et al., 2009; Terranova et al., 1994). Further, ageing in male rats reduced the extent of investigation and recognition of females (Guan and Dluzen, 1994; Mencio-Wszalek et al., 1992) http://www.sciencedirect.com/science/article/pii/S0031938410003549 - ref_bb0070. Thus, these findings suggest that ageing seems to reduce social cognition/motivation, normally accompanied by a high-level emotional response (Shoji and Mizoguchi, 2011).

Although Premack and Woodruff (1978) asked: "Does the chimpanzee have a Theory of Mind?" more than 30 years ago, this question remains in the forefront of research on social behavior in nonhuman primates. Lesion studies may provide some information on the neural mechanisms implicated in social behavior. The results of most works on nonhuman primates support the general conclusion that the orbitofrontal (OF) cortex plays a critical role in species-typical social behavior (Deets et al., 1970; Franzen and Myers, 1973; Machado and Bachevalier, 2006; Myers et al., 1973). For example, studies in rhesus macaques allowed to freely interact with partner animals showed that lesioned animals (i.e., those with damage to the OF cortex) differed from control macaques in terms of social interest and fear-related behaviors (Babineau et al., 2011). Moreover, lesioned animals, compared to controls, showed more aggressive interactions and responded differently to both affiliative and threatening signals (Machado and Bachevalier, 2006). Lesions in the prefrontal or anterior temporal cortex resulted in major deficits in various aspects of social behavior, including major losses in the repertoires of facial expression and vocalization. This suggested that, in rhesus monkeys, such functions are primarily involved in emotional and social communication (Myers, 1972). Monkeys with hippocampal lesions showed an increased exploration tendency, enhanced excitability, and reduced responses to affiliative signals, whereas lesions in the amygdala caused several personality changes that precluded the conduct of positive social interactions. Exploration and excitability increased, affiliation and popularity decreased, and responses to threatening social signals were altered (Machado and Bachevalier, 2006). To the best of our knowledge, only one study to date has assessed the effects of age on social behavior in nonhuman primates (rhesus macaques), showing that older females spent less time in proximity to others, less time in social contact, and were groomed less often than were younger adult females. In contrast, older males spent more time in proximity to others, more time in social contact, and were groomed more often than were other adult males. These differences have been interpreted as stemming from sex-associated distinctions in ways of life, commencing at the beginning of adulthood, requiring male and female rhesus macaques to adopt different social strategies to ensure "successful" ageing (Corr, 2003).

Findings from these lesion studies can be paralleled to our knowledge in the field of social cognition in humans: the aforementioned brain areas involved in social behavior in non human primates (e.g., OF cortex, amygdala, anterior temporal lobe) correspond (at least partially) to the equivalent cerebral areas implicated in social cognition in humans (see Section 3.2 for a description of the latter).Nevertheless, a review of empirical studies of imitation, self-recognition, social relationships, deception, role-taking, and perspective-taking suggests that whenever nonhuman primate

behavior has been interpreted as a sign of ToM, this may have occurred by chance or may be a product of nonmentalistic processes (e.g., associative learning or inferences based on nonmental categories) (Heyes, 1998).

3.2. Neural substrates of social cognition in younger adults

To date, the neural bases of ToM have been mostly investigated in adults. More recently, however, studies have been performed on changes in ToM neural circuits during development (Kobayashi et al., 2007; Moriguchi et al., 2007; Ohnishi et al., 2004), and, until 2009 (i.e., Charlton et al., 2009), no study had focused on the relationship between structural or functional brain measures and ToM in normal ageing (see Section 3.4 for a discussion of the latter).

Functional magnetic resonance imaging (fMRI) in young adults has suggested that ToM abilities are related to activation of specific brain regions, such as the anterior cingulate (AC) (Gallagher and Frith, 2003; Gallagher et al., 2000; Gobbini et al., 2007; Sommer et al., 2007), which may be involved in the attribution of mental states to oneself and others (Vogeley et al., 2001); the temporal poles (TP) (Gallagher and Frith, 2003; Völlm et al., 2006), which may play roles in social and emotional processes, including face recognition and ToM, thus going beyond semantic memory (Olson et al., 2007); and the inferior parietal lobule (IPL) (Kobayashi et al., 2007). The temporo-parietal junction (TPJ) also appears to be particular important for ToM (Aichhorn et al., 2009; Gobbini et al., 2007; Kobayashi et al., 2007; Perner et al., 2006; Saxe, 2006; Sommer et al., 2007; Völlm et al., 2006; Young et al., 2007) as frequently shown by a paradigm using Happé et al.'s popular false-belief task (Happé et al., 1998) (or tasks inspired by the latter) during fMRI measures. The medial prefrontal cortex (mPFC) may also play a core role in social cognition abilities (Amodio and Frith, 2006; Frith and Frith, 2006; Saxe and Wexler, 2005). More precisely, Brodman's areas 8 and 9 (Fletcher et al., 1995; Russell and Sharma, 2003), and the OF cortex are important in ToM (Baron-Cohen et al., 1994). Indeed, this area is activated even during a simple ToM task, such as asking participants to recognize words qualifying mental states. Furthermore, positron emission tomography showed increased activation of the amygdala when individuals were asked to infer mental states from pictures of eyes (Baron-Cohen et al., 1999), whereas the superior temporal sulcus (STS) has been shown to respond selectively during observation of goal-directed actions, but not to movements lacking intention (Perrett et al., 1989). A recent review presents a quantitative meta-analysis of neuroimaging studies pertaining to ToM (Mar, 2011). The core mentalizing network, defined as the overlap between story- and nonstory-based studies (i.e., verbal and non-verbal tasks), includes the mPFC, bilateral posterior STS, more anterior temporal regions (i.e., STS, middle temporal gyrus) bilaterally and the posterior cingulated cortex and precuneus. The left side of the amygdala is activated during story-based studies, whereas the right side is activated during nonstory-based studies.

Structural imaging, more precisely voxel-based morphometry, has yielded further interesting information on the neural correlates of ToM. Indeed, in schizophrenic patients, poor performance on the Faux-Pas and RME Tests has been associated with ventromedial and ventrolateral PFC gray matter loss (GM), respectively (Hirao et al., 2008; Hooker et al., 2011). In healthy adults, GM volume in the posterior frontal poles, the medial OF cortex, the ventral portion of the medial frontal gyrus, the left TPJ, and the STS, varies parametrically with mentalizing ability (Lewis et al., 2011). White matter (WM) loss has also been shown to impair social cognition. WM loss in the frontal, temporal, and insular regions of patients with myotonic dystrophy type 1 may be especially associated with a decrease in emotional sensitivity to disgust and anger (Kobayakawa et al., 2010).

Lesion studies also provide valuable information concerning the brain areas implicated in ToM. Damage to the frontal lobes was found to impair the ability to infer mental states of others, indicating the importance of the frontal lobes in ToM (Stuss et al., 2001). More precisely, damage to the OF cortex (i.e., ventromedial PFC, vmPFC) resulted in impairment of the affective part of ToM. Although participants successfully completed false beliefs tasks, they failed the Faux-Pas Test, which demands more subtle social reasoning (Shamay-Tsoory and Aharon-Peretz, 2007). Bilateral damage to the amygdala also causes affective ToM impairment (Stone et al., 2003), confirming neuroimaging findings (e.g., those of Baron-Cohen et al., 1999). In contrast, cognitive ToM seems to rely on the integrity of the dorsolateral PFC (dlPFC), as shown by a transcranial magnetic stimulation (TMS) study (Kalbe et al., 2010).

A recent model of the neuroanatomical and neurochemical aspects of affective and cognitive ToM has suggested that these two forms of ToM engage distinct networks, with affective ToM being sustained by the vmPFC, the OF cortex, the ventral AC cortex, the amygdala, and the ventral striatum. Cognitive ToM is underpinned by the dorsomedial PFC (dmPFC), the dorsal AC cortex, and the dorsal striatum (Abu-Akel and Shamay-Tsoory, 2011). The TP appears to be critical for activity of both the affective and cognitive components. The TPJ, including the caudal part of the STS and the IPL (Zaitchik et al., 2010), intervenes during detection of a mental state; the representation obtained in this manner is next relayed further without any preference for processing of either the affective or cognitive component (Abu-Akel and Shamay-Tsoory, 2011). Nevertheless, although both ToM components seem to trigger distinct networks, these networks interact (LeDoux, 1995; Shamay-Tsoory, 2010).

Thus, if ToM performance is supported by a network of brain regions (Abu-Akel, 2003a; Völlm et al., 2006), the functional connectivity among these regions may be at least as important as the integrity of the localized neuronal structures. Indeed, the basal ganglia (BG) are thought to be connected to well delimited regions of the frontal cortex by five separate and parallel loops (Alexander et al., 1986). This model of fronto-subcortical circuits has given rise to detailed predictions regarding the causal relationship between the disruption of a particular loop and specific behavioral consequences (Lawrence et al., 1998; Mega and Cummings, 1994). These behavioral disorders may result from the disturbances in connectivity among cortico-subcortical networks due to damage to the BG. A recent case study found that damage to the head of the left caudate nucleus resulted in impairment of ToM and emotion recognition (Kemp et al., submitted for publication). Their behavioral results, supported by neuroimaging recordings, showed that the impairment was due to a disconnection of the subcortical OF circuit resulting from damage to the caudate.

3.3. Ageing and frontal-lobe dysfunction

Generally, many changes in the brain occur during the course of normal ageing, including decreases in brain volume (Matsumae et al., 1996; Resnick et al., 2000; Scahill et al., 2003), increases in the number and volume of white matter hyperintensities (WMH) (Breteler et al., 1994; Schmidt et al., 2005; van den Heuvel et al., 2006; Ylikoski et al., 1993) and, as recently demonstrated by diffusion tensor imaging (DTI), a decrease in WM integrity (Charlton et al., 2006; O'Sullivan et al., 2001; Pfefferbaum and Sullivan, 2003).

The effects of ageing on the frontal lobes have been well documented (Hawkins et al., 1983; Takada et al., 1992; Yanase et al., 2005). The frontal lobes are among the regions most affected by age, as determined by volumetric and functional analyses. Linear regression analysis has shown that whole brain metabolism decreases 38% and frontal lobe metabolism 42% with age (Tumeh et al., 2007). Current neuropsychological models propose that this frontal-lobe

deterioration is responsible for many age-related cognitive changes (Daigneault and Braun, 1993; Moscovitch and Winocur, 1995; West, 1996). Furthermore, although age-associated effects on activities of other regions of the brain have been reported (Greenwood, 2000), studies of cognitive ageing show that age effects are most evident on cognitive tasks and memory measures thought to be sensitive to frontal-lobe dysfunction.

However, less consensus has emerged on the differential structural vulnerability of the vmPFC and dlPFC. Using tasks requiring activity of the dorsolateral (DL) prefrontal region and tasks dependent on ventromedial (VM) prefrontal regions, (Salat et al., 2002) found that performance on all measures of prefrontal functioning were lower in older than in younger adults, suggesting a generalized decline within the prefrontal cortex in later life. However, others (e.g., Baena et al., 2010) found that vmPFC functions were more sensitive to the effects of age. In contrast, another report showed that age affected performance on tasks involving the DL, but not the VM, prefrontal region (MacPherson et al., 2002). It was suggested that the DL region was specifically involved in mediating cognitive changes occurring upon ageing; it was not necessary to propose a global decline in frontal lobe function. Again, some neuroimaging data have shown that accelerated gray matter loss is clustered in focal regions, including the DL frontal cortex (Grieve et al., 2005). A decline in cortical (i.e., DL) activation has been revealed by NIRS (Kwee and Nakada, 2003), thus confirming the former findings. In contrast, both cross-sectional and longitudinal studies have suggested that, within the prefrontal cortex, the vmPFC (i.e., the OF cortex) is more vulnerable to age-associated structural change (Convit et al., 2001; Resnick et al., 2000, 2003).

3.4. Neural basis of age related ToM modifications

For social cognition *per se*, the neuronal substrates involved in ageing were assessed using two main indicators, normalized whole brain volume and white matter integrity (WMI), as measured by volumetric analyses of WMH and DTI (Charlton et al., 2009), showing that performance on a subset of ToM-related stories (Happé et al., 1998) correlated significantly with DTI measures of WMI but not with WMH or whole-brain volume. In agreement with the hypothesis that activity of a network of different cortical areas underpinned ToM (Abu-Akel, 2003a; Gallagher and Frith, 2003; Völlm et al., 2006), the described association between diminished ToM performance and reduced WMI may be the consequence of disruption of the functional network of multiple brain regions necessary for ToM, caused by damage to white matter connections across the brain (Charlton et al., 2009).

Functional neuroimaging methods have also assessed the neural basis of mentalizing, relative to changes during normal ageing. To better understand the possible changes in brain networks underpinning ToM across the human life-span, the neural basis of mind-reading ability through the eyes was compared in healthy younger (mean age 25.2 years) and older (mean age, 65.2 years), who underwent fMRI scanning while performing the RME Test (Castelli et al., 2010). There were no between group differences in behavioral performance, and both groups showed activation of the posterior end of the superior temporal sulcus and the temporal pole, indicating that older people showed no impairment of mentalizing circuits. Despite their similar behavioral performance, there was a relevant shifting of the neural circuits utilized by each group to solve the task, with older subjects showing a more bilateral activation of frontal areas. The older group was similar to the younger group in showing activation of the right inferior frontal gyrus, but differed from the younger group in having increased activity in the left inferior frontal gyrus. The latter may represent a functional compensation by older participants, due to a preserved neuronal plasticity during "successful" ageing (Cabeza et al., 2004; Reuter-Lorenz, 2002; Reuter-Lorenz and Lustig, 2005).

4. Pathological ageing and social cognition

Linking specific neurodegenerative pathologies to social cognition deficits may help in the development of a theoretical framework for age-related declines in this ability. On the one hand, the apparent decline in social cognition with normal ageing is, at least partially, independent of a more general cognitive or executive decline. On the other hand, several studies have shown that different brain areas implicated in ToM (see Section 3.2 for a description of such brain areas) are specifically affected in certain neurodegenerative pathologies in older adults (see Table 2 for a description of studies on social cognition in neurodegenerative pathologies). Considering these two features together, it may be of particular interest to determine if age-related decline in social cognition can be linked to impairment of such abilities in patients with different pathologies, with the common denominator being the neuronal network underpinning ToM. To confirm this hypothesis, it is also necessary to consider pathologies in which deficits in ToM have been investigated, but in which no confirmed damage to the neuronal network underlying social cognition abilities has been observed.

4.1. Alzheimer's disease

Alzheimer's disease (AD) is a progressive neurologic disease of the brain that leads to the irreversible loss of neurons and dementia. Neurodegeneration is most evident in the hippocampo-cingulo-temporo-parietal network (Seeley et al., 2007), with progression to different disease stages being characterized by the brain regions affected. These are the transentorhinal cortex; the entorhinal cortex; the hippocampus; the anterior temporal cortex; the inferior temporal cortex; the medium temporal cortex; polymodal association areas (prefrontal, parietal inferior, temporal superior); unimodal areas; primary motor or sensory areas; and all neocortical areas (Delacourte et al., 1999). The clinical hallmarks of AD are progressive impairment in memory, judgment, decision making, orientation to physical surroundings, and language. Although poorly understood, social dysfunction frequently occurs during mild dementia states of AD. Although brain areas commonly associated with social cognition abilities are not necessarily among those first affected, impaired social cognition (e.g., erroneous inferring of mental state, faulty processing of emotional expressions) maybe present in such patients, may be related to social functioning, and may contribute to early changes in social behavior and impairment of autonomy and quality-of-life during the mild dementia stage of AD (Piquard et al., 2004; Shimokawa et al., 2001).

4.1.1. Cognitive and affective ToM

A consensus has been reached concerning cognitive ToM (i.e., first- and second-order false beliefs) in AD. Indeed, patients with AD generally perform well on first-order false belief tasks (Gregory et al., 2002), and may even reach ceiling performance on this type of task (Fernandez-Duque et al., 2009). Moreover, AD patients showed no difficulties in attributing a false belief to another person or in recognizing their own previous false beliefs (Zaitchik et al., 2004). In contrast, AD patients frequently fail at second-order false beliefs tasks (Fernandez-Duque et al., 2009; Gregory et al., 2002). A case study describing a 75-year-old woman with AD also reported this pattern of results (Modinos et al., 2009) and was consistent with results showing that 65% of AD patients with mild dementia could not pass a second-order false belief task, compared with 0 of 10 age-comparable healthy controls (Cuerva et al., 2001).

Table 2
Studies on social cognition in neurodegenerative pathologies.

Pathology	Authors, published	Sample (*n*)	SC function assessed	Main SC results	
AD	Gregory et al. (2002)	12 AD	Cognitive and affective	First-order FBT	=
		16 HC	ToM	Second-order FBT	<
				RME Test	=
				Faux-Pas Test	=
				Control questions	<
	Fernandez-Duque et al. (2009)	17 AD		First-order FBT	=
		12 HC		Second-order FBT	<
	Bediou et al. (2009)	10 AD	Emotion recognition	Facial expression task	<
		10 HC		Control tasks	=
	Drapeau et al. (2009)	7 AD		Sadness, disgust and fear	<
		16 HC		Happiness, surprise, anger	=
MCI	Bediou et al. (2009)	10 aMCI	Emotion recognition	Subtle emotional expressions	<
		10 HC		Higher intensities emotional	=
				expression	=
				Control tasks	
	Weiss et al. (2008)	21 aMCIsd		aMCIsd	=
		31 aMCImd		aMCImd: Sadness fear and neutral	<
		5 HC			
fvFTLD	Gregory et al. (2002)	19 fvFTLD	Cognitive and affective	First- and second-order FBT Control	<
		16 HC	ToM	questions	=
				RME Test	<
				Faux-Pas Test	<
				Control questions	=
	Modinos et al. (2009)	Case study		First- and second-order FBT	<
				RME Test	<
				Faux-Pas Test	<
	Lough et al. (2006)	18 fvFTLD	Emotion recognition	Negative emotions	<
		13 HC			
	Rosen et al. (2004)	fvFTLD		Negative & positive emotions	<
		HC			
PD	Saltzman et al. (2000)	11 PD	Cognitive ToM	FBT	<
		8 HC		Spy Test	<
				Perspective-Taking Test	=
				Knower/Guesser Test	=
	Bodden et al. (2010)	21 PD		First-order FB	=
		21 HC		Second-order FB	<
	Peron et al. (2009)	17 early PD	Affective ToM	Faux-Pas Test	=
		27 advanced		RME Test	=
		PD			
		26H HC			
	Bodden et al. (2010)	21 PD		Yoni Test	=
		21 HC		First-order FB	<
				Second-order FB	<
				RME Test	
	Sprengelmeyer et al. (2003)	36 PD	Emotion recognition	Fear, sadness, anger, disgust	<
		40 HC		Control tasks	=
	Dujardin et al. (2004)	18 early PD		Anger, sadness and disgust	<
		18 HC			
TLD	Bon et al. (2009)	Case study (SD)	Cognitive and affective	FBT	<
			ToM	Judgment of preference tasks	<
				RME Test	=
				Faux-pas test	<
	Rosen et al. (2002b)	9 tvFTLD	Emotion recognition	Basic emotions	<
		10 HC			
	Calabria et al. (2009)	Case study (SD)		Basic emotions	<

SC: social cognition; ToM: Theory of Mind; FB: false belief; FBT: false beliefs task; RME: Reading the Mind in the Eyes; HC: healthy controls; AD: Alzheimer's Disease; MCI: mild cognitive impairment; aMCI: amnestic mild cognitive impairment; aMCIsd: amnestic mild cognitive impairment simple domain; aMCImd: amnestic mild cognitive impairment multiple domain; fvFTLD: frontal version of frontotemporal lobar degeneration; tvFTLD: temporal version of frontotemporal lobar degeneration; PD: Parkinson's disease; TLD: temporal lobar degeneration; SC: semantic dementia; (<): impaired, (=): preserved, (>): improved.

Alternatively to a primary ToM impairment *per se*, the impairment in second-order ToM in AD patients may be secondary to their cognitive impairments. Indeed, second-order false belief tasks are cognitively more demanding, especially on working and episodic memory, than first-order false belief tasks, where cognitive demands are minimal. Compared with AD patients who passed second-order false believe tasks, those who did not pass this task had more severe deficits on tests of verbal anterograde memory, verbal comprehension, abstract thinking, and naming (Cuerva et al., 2001). Similarly, on a third ToM task, where the key information was embedded in a story narrative, AD patients performed significantly more poorly than controls, despite the task involving inferences of first-order false beliefs (Zaitchik et al., 2004). Correlations between the neuropsychological tests and performance on the third ToM task suggest that cognitive demands are important in false-belief task performance and that the basic ability involved in ToM (i.e., making inferences about beliefs held by another person) remains intact under simple conditions in patients with mild to moderate AD.

To our knowledge, only two studies to date have assessed the effects of AD on affective ToM. Both studies found that the "warm" part of ToM was relatively well preserved. Patients with AD performed as well as controls in terms of faux-pas detection, but they failed the "control" memory questions (Gregory et al., 2002).

Similarly, the second study, a case report, found that the tested AD patient performed adequately on the RME and Faux-Pas Tests (Modinos et al., 2009).

4.1.2. Emotion recognition

Another aspect of social cognition more frequently assessed in AD patients is facial expression recognition, which appears to be impaired in patients with moderate (Bediou et al., 2009) and severe (Albert et al., 1991; Hargrave et al., 2002; Koff et al., 1999) AD. However, whether a deficit is present in patients with mild dementia requires further consideration. Although one study found no differences between AD patients and healthy controls in the recognition of emotions (happiness, sadness, anger, fear or neutral) (Bucks and Radford, 2004), others did (e.g., Drapeau et al., 2009; Guaita et al., 2009; Lavenu and Pasquier, 2005). Indeed, studies showed that the degree of recognition impairment differed by type of emotion tested. Thus, emotion decoding performance in AD is likely impaired, particularly when relatively subtle expressions were presented (Phillips et al., 2010). Similarly, AD patients with mild dementia did not differ significantly from controls in the detection of prototypical full intensity facial expressions (100%), but showed a significant deficit in the detection of a larger range of emotional intensities (Bediou et al., 2009).

Differences may also depend on the valence of the emotions, but the results are not always consistent. As with the emotion recognition pattern observed during normal ageing (see Section 2.3), a positivity bias may be present in AD patients; positive emotions are better recognized than are negative emotions (Guaita et al., 2009). These results are consistent with the finding that AD patients were more impaired than were matched controls in terms of recognizing facial expression of emotions, especially those associated with sadness, disgust, and fear (Drapeau et al., 2009). Using the Multimodal Emotion Recognition Test (Banziger et al., 2009), AD patients were found to have lower objective recognition performances for disgust and fear (Wiechetek Ostos et al., 2011), whereas another group of AD patients showed diminished recognition, not only of sad and fearful, but also of neutral and happy, expressions. Furthermore, AD patients exhibited decreased differentiation between happy and sad expressions (Kohler et al., 2005). In contrast, another study (Lavenu et al., 1999) found that AD patients did not differ from controls in their recognition of anger, sadness and disgust, but showed poorer recognition of fear and contempt. An interesting longitudinal study assessing emotion recognition in patients with dementia (Lavenu and Pasquier, 2005) reported that the recognition of facial expression decreases as dementia progresses. Indeed, AD patients (as well as patients suffering from frontotemporal lobar degeneration) were tested on an emotion recognition test at their first visit and three years later. All AD patients' performances worsened with the progression of the disease.

However, some argue that the poor performances by AD patients in emotion recognition may not be due to ToM impairments. Rather, the difficulties in choosing between labels to describe an emotional face may be predicted by executive dysfunction, whereas impaired ability to match 2 emotional faces may be related to general difficulties with face perception (Phillips et al., 2010). Although another study showed significant impairments in several tests of facial emotion recognition in AD patients, these impairments may have been due to reduced ability to recognize non-emotional facial features and to reduced verbal processing in AD patients (Albert et al., 1991). These results suggest that the deficits in perception of affect tasks in AD patients are likely due to cognitive defects, not to a primary impairment in the perception of emotion.

4.1.3. Empathy

Empathy has also been assessed in AD patients, although less systematically than other social cognition abilities. In one study (Fernandez-Duque et al., 2010), participants were exposed to three videotapes, each depicting a different woman (non-actor) describing a recent personal experience (all experiences were real). After watching the videotapes, participants answered questions regarding the interviewee's feelings. AD patients inferred emotions as accurately as the healthy elderly, provided the emotions were displayed unambiguously and consistently across the interview. However, when the displayed emotions became more variable and ambiguous, the performance of AD patients became impaired relative to the healthy controls. These findings suggested that cognitive deficits lead to impaired empathy even in patients who do not display obvious social deficits, thereby highlighting the importance of cognitive deficits as a possible contributor to empathy problems.

4.2. Mild cognitive impairment

The concept of mild cognitive impairment (MCI) has evolved in recent years to represent the clinical transition between the cognitive changes found in normal ageing and those of early AD (Petersen et al., 2001, 1999). MCI may be heterogeneous, with the most common subtype being amnestic MCI (aMCI), a condition characterized by prominent memory impairment and likely progression to AD (Petersen, 2003). In addition, other subtypes, with different clinical criteria, have been proposed. It may therefore be worthwhile to assess social cognition abilities during the prodromal state of AD, since these findings suggest that social cognition, in particular facial emotion processing, may be impaired in MCI prior to the more marked cognitive deficits observed at clinically diagnosed AD (Teng et al., 2007).

4.2.1. Emotion recognition

Findings in this field are generally consistent. Patients with aMCI and healthy controls did not differ significantly on an emotion recognition task (Teng et al., 2007). Whereas patients with AD, even in the mild stage, showed impaired recognition of emotion in facial expressions, patients with MCI only exhibit difficulties when the emotion is weakly expressed (Spoletini et al., 2008). Moreover, low-intensity facial emotion recognition deficits may progress, from healthy controls to aMCI to mild AD patients. These findings have been confirmed in a study showing that individuals with aMCI responded similarly to individuals with mild AD dementia when emotional expression was more subtle (until 40%), whereas the former were more comparable to controls at higher intensities of emotional expression (80–100%) (Bediou et al., 2009). The intermediate performance of individuals with aMCI on facial expression detection may reflect the emergence of deficit worsening with the progression of AD neuropathology from the entorhinal cortex and hippocampus toward the superior temporal sulcus, a brain area critical for emotion recognition. This progression may also coincide with the appearance of social dysfunction, in that the evolution of the deficit in low-intensity fearful facial recognition in the aMCI phase progressed to deficiencies in all intensities and emotions in individuals with mild AD (Spoletini et al., 2008), deficiencies that may result from the progressive degeneration of brain structures that modulate emotional processing. Similarly, a recent study showed that individuals with simple-domain, but not multiple-domain, MCI performed normally on facial affect discrimination (Teng et al., 2007). In this and another (Weiss et al., 2008) study, performance across the facial affect recognition tests demonstrated intact performance by the single-domain aMCI group but significantly impaired performance by the multiple-domain aMCI group. Moreover, recognition of overall emotions, including sad, fearful, and neutral faces, was impaired, and the deficiencies increased with the severity of AD. Thus, multiple-domain MCI is generally associated with more diffuse neuropathological lesions and altered cognitive functioning than is simple-domain MCI; the

latter condition is characterized by neurofibrillary pathology affecting the entorhinal cortex, the hippocampus and the amygdale (Markesbery, 2010). On the other hand, multiple-domain MCI has been associated with reduced volume of all of the hippocampus, the middle temporal gyrus (bilaterally), the right inferior frontal gyrus and the bilateral superior temporal gyrus (Bell-McGinty et al., 2005).

In summary, the observed difficulties in ToM and emotion recognition in both AD and MCI patients seem to be secondary to their cognitive impairments and eventual difficulties with face perception and verbal processing, rather than a primary impairment in ToM. This hypothesis is in line with current knowledge about the neuropathology of AD and MCI and the neuronal correlates underlying ToM. That is, brain areas commonly implicated in social cognition, particularly the frontal lobes, are relatively spared in AD, especially in early stages of the disease.

4.3. Frontotemporal dementia

Patients suffering from the frontal variant of the frontotemporal lobar degeneration (fvFTLD) present with insidious changes in personality and behavior, including lack of empathy or concern for others, apathy, socially inappropriate and disinhibited behavior, impaired personal awareness and loss of insight (The Lund and Manchester Groups, 1994). Despite the gross alterations in interpersonal behavior, these patients may perform normally on traditional frontal executive tasks (Gregory et al., 1999; Rahman et al., 1999). Atrophy of the mesial frontal, OF, and anterior insular cortices is reliably observed (Davatzikos et al., 2008; Hornberger et al., 2011; Seeley et al., 2008). Combined atrophy of the frontal and anterior temporal cortex and the basal ganglia may also be seen in some patients (Whitwell et al., 2009). Selective atrophy of the AC and frontal insular cortices early during the course of fvFTLD has been objectified using voxel-based morphometry and cortical thickness mapping (Boccardi et al., 2005; Schroeter et al., 2007; Seeley et al., 2008).

Given the critical roles of the frontal lobes, especially the OF cortex, for the integrity of ToM and the neuropathology of fvFTLD, interest in the assessment of social cognition in these patients has increased. Social cognition was first assessed in case studies of patients with fvFTLD, with one of the first being a 47-year-old man diagnosed with fvFTLD and exhibiting severe antisocial behavior (Lough et al., 2001). Although he showed limited cognitive impairment, he failed both first- and second-order ToM tests and was unable to recognize a single faux-pas, he correctly answered the control questions. His ability to detect emotional states (measured *via* the RME Test) was intact. Similarly interesting results were found in a 57-year-old man presenting with marked behavioral symptoms (Lough and Hodges, 2002). General neuropsychological assessment showed limited cognitive impairment, but he was severely impaired on all ToM tests, with a clear dissociation between executive functions and ToM ability. These two case reports permit to highlight an interesting dissociation, which is that both patients showed a deficit in ToM ability independent of the level of executive functions.

4.3.1. Cognitive and affective ToM

The first group study testing the effects of fvFTLD on cognitive ToM found that, compared with healthy controls, the fvFTLD group was significantly impaired on first- and second-order ToM tasks, whereas they had no difficulties with the control questions designed to test general comprehension and memory (Gregory et al., 2002). These results were supported by findings obtained in a case study of a 64-year-old man suffering from fvFTLD, who showed clear deficiencies in ToM, failing the first-order and second-order false beliefs tasks (Modinos et al., 2009). In contrast, another study could replicate these findings only partially (Fernandez-Duque et al., 2009). Although the fvFTLD group was impaired relative to a healthy elderly group in second-order false-belief tasks, their mentalizing ability, as assessed by a first-order false-belief task, seemed to be spared during early stages of fvFTLD.

The link between ToM and executive function performances is unclear. Findings have suggested that executive function, semantic memory and general intellectual measures were not correlated with ToM performance (i.e., assessed by the Faux-Pas and RME Tests) (Gregory et al., 2002; Lough et al., 2001; Lough and Hodges, 2002). In contrast, fvFTLD patients who made errors on first-order false-belief questions performed significantly worse than those who did not on the Dementia Rating Scale (DRS), the comprehension section of the Western Aphasia Battery, digit span tasks, the Boston Naming Task, semantic fluency task, line orientation task and Rey copy figure, suggesting that the performance of patients with fvFTLD on false-belief tasks depends primarily on the cognitive demands of these tasks (Fernandez-Duque et al., 2009).

Another study investigated the ability of patients with fvFTLD to interpret social situations (such as humor appreciation, deception, bluff, and double bluff) and to attribute mental states to others (Snowden et al., 2003), by using single cartoons, pairs of cartoons, story comprehension (Happé et al., 1999), and judgment of preference tasks (Baron-Cohen et al., 1995). Compared with healthy controls, the performance of fvFTLD patients was severely impaired on all of the ToM tasks (Snowden et al., 2003). That study paid particular attention to fvFTLD patients' performance on the judgment of preference task. In this task, which is substantially undemanding and requires no active mental manipulation or integration of information, participants merely point to one of four pictures that a cartoon face prefers, with preference being determined by direction of eye gaze. Interestingly fvFTLD patients failed to ascribe preference ("Which one does he/she like?") but had no difficulty in reporting direction of eye gaze ("Which one is he/she looking at?"). As these two sub-tasks differ in mental state attribution but not in cognitive load required, the performance differences shown by the fvFTLD patients provide evidence for a specific ToM impairment. Although it is reasonable to presume that executive functions generally contribute to test performance, the impairments shown by the fvFTLD patients only secondarily affect ToM tasks and may mask specific deficits in mental state attribution (Snowden et al., 2003). Deficits in ToM independent of executive function may be expected early in the course of this disease, when pathological changes are confined to OF regions (Snowden et al., 1996). Later in the disease course, when the pathology extends into the dorsolateral regions, the picture will be increasingly colored by additional executive deficiencies.

Use of the Faux-Pas and the RME Tests objectified impairments of the warm part of ToM in fvFTLD patients (Gregory et al., 2002; Torralva et al., 2007). Performance by these patients on the Faux-Pas Test was significantly impaired relative to controls, with the errors made by fvFTLD patients showing their difficulty with several aspects of mental state inference. Some patients failed to detect when something hurtful or inappropriate had been said, whereas others inferred that something inappropriate had been said intentionally, indicating a failure to accurately infer the belief states of the story characters. Patients' performance on the RME Test was also significantly poorer than that of controls. A case report described similar results in a patient who failed both the Faux-Pas and RME Tests (Modinos et al., 2009). Errors were based on misattributing beliefs, assuming intentionality in comments that could hurt someone else's feelings, and attributing wrong emotions to eye expressions. Again, loss of memory was unlikely to be the cause, since the patient always remembered the story and the characters' names.

4.3.2. Emotion recognition

Emotion recognition in fvFTLD has been of particular interest, particularly because the emotion recognition impairments frequently observed likely contribute to the abnormal social behavior that is characteristic of this condition.

Most studies of emotional recognition in these patients have utilized the Ekman series of faces. This uniformity in test methodology has the distinct advantage of allowing direct comparisons across studies, as well as being a diagnostic tool for FTLD (Diehl-Schmid et al., 2007). Using a cut-off score of 46 points (total score, 60 points), the Ekman 60 Faces Test discriminated between patients with mild FTD and HC with 97% diagnostic accuracy (sensitivity: 94%; specificity: 100%).

Other studies focusing on patients with fvFTLD have observed difficulties in individuals with mild stages of the pathology, even if the results slightly differed across studies. Although one study showed impairments in FTD for all emotions (Snowden et al., 2008), another (Fernandez-Duque and Black, 2005; Lough et al., 2006) found that patients had difficulties recognizing negative emotions (e.g., anger, disgust, sadness and fear), but were able to recognize positive emotions (e.g., surprise and happiness). Similar findings were highlighted by Lough et al., (Lough et al., 2006). In their fvFTLD patients, emotion recognition was globally impaired, but was particularly so for anger and disgust. More heterogeneous observations have been made by other studies. For example, some patient groups exhibited difficulties recognizing disgust, happiness and fear (Bediou et al., 2009), whereas others showed impaired identification of happiness, sadness and anger, but not of fear and disgust (Keane et al., 2002). Other patients showed impaired identification of expressions of anger and surprise, but performed at control levels on expressions of disgust, happiness, sadness and fear (Kessels et al., 2007). In contrast, another study showed the opposite results, with low recognition scores for happy, sad, angry and frightened faces (Omar et al., 2011).

Patients with fvFTLD were found to be impaired in the recognition of both negative (e.g., sadness, anger and fear) and positive (e.g., happiness) facial expressions, whereas patients with the temporal version of the FTLD showed impairment only in recognizing negative emotions (Rosen et al., 2004).

The impairments observed in patients with fvFTLD may arise from various causes. Patients with fvFTLD were impaired in detecting facial expressions, as well as in determining gaze direction, suggesting that these patients do not focus their gaze on the eye region, which is crucial for the recognition of facial expression (Bediou et al., 2009). In contrast, other studies have found that patients with fvFTLD had no impairments of visual perception of faces (Rosen et al., 2004; Snowden et al., 2008). The performance of fvFTLD patients on a control test of facial identity matching was completely normal, indicating that the difficulties these patients had in detecting facial expression were not due to impaired visual perception or attention. Furthermore, the impairments in recognition of facial expression were present when tasks were minimized (two-choice tasks) and when verbal demands were eliminated (face-face-matching tasks), suggesting that the impairment observed in these patients were not due to reduced general mental demands or language. Despite the significant correlations between performance on emotion and standard executive tests, suggesting that general executive impairments influence performance, widespread deficiencies on emotion recognition tasks remained, even when executive demands of the task were reduced. Likewise, the absence of deficiencies on identity-matching tasks, with similar executive demands as emotion-matching tasks, provides strong evidence that emotion recognition impairments in FTD are primary. Moreover, impairments were observed across test modalities (visual and auditory), providing support for claims that the deficiencies observed in fvFTLD patients reflect a multimodal impairment of emotion recognition (Snowden et al., 2008). Similarly, participants with fvFTLD showed impaired recognition of facial expressions in the context of preserved recognition of facial identity, although deficiencies in vocal emotion recognition tasks were also observed (Keane et al., 2002). These results are consistent with the idea that fvFTLD affects the recognition of emotional signals from multiple modalities rather than from facial expression processing alone. Finally, when behavior and autonomic responses were used to assess the reactivity to video clips about happiness, fear and sadness, fvFTLD patients showed a general congruous reactivity, not connected to denomination or recognition deficits (Werner et al., 2007). These findings indicate that the socio-emotional decline typically seen in FTLD patients results more from an inability to process certain emotions in other people than from deficiencies in emotional reactivity.

4.3.3. Empathy

Empathy appears to be deficient in fvFTLD, with significant impairments on caregiver assessments of empathy (Eslinger et al., 2011; Lough et al., 2006). Similarly, using the same stimuli as those presented to patients with AD, both groups had the same patterns of results, namely a deficit in empathic accuracy when the displayed emotions were ambiguous or inconsistent across the interview (Fernandez-Duque et al., 2010). The similar profile of these two groups despite their differences in social skills, suggested that nonsocial cognitive processes affected in dementia may be important in drawing inferences about the feelings of other individuals.

4.3.4. Other aspects of social cognition

Impairments in other aspects of social cognition such as moral reasoning have also been reported (Lough et al., 2006). Knowledge of social rules was intact in patients with fvFTLD, but moral reasoning was defective, due, in part, to an inability to appropriately rate the seriousness of moral and conventional transgressions. The results of the moral–conventional reasoning test were particularly revealing, in that patients showed an inability to integrate social knowledge with its affective connotations. Not only did they fail to adequately recognize violations of the social norm, but they were also unable to distinguish judgmentally between moral and conventional transgressions. These findings suggest that executive dysfunction underlies some, but not all, of these deficiencies.

A recent report described four patients with FTLD, but also with a clear consciousness (i.e., when specific actions were pointed out, these were not denied; the subjects described the actions in detail, and agreed that they were both wrong and harmful) and sufficiently intact cognition (i.e., defined by neuropsychological assessment), who committed criminal violations (Mendez, 2010). They understood the nature of their acts and the potential consequences, but did not feel sufficiently concerned to be deterred. These findings suggest that patients with FTLD are particularly prone to sociopathic behavior while retaining knowledge of their acts and of moral and conventional rules.

In conclusion, despite the contribution of executive function to test performance, impairment of these abilities has only a secondary effect on social cognition performance. Moreover, patients with fvFTLD have specific deficiencies in ToM, emotion recognition, empathy and moral and conventional reasoning. This suggestion is supported by neuroanatomical data, since the brain areas crucial for ToM (i.e., the frontal lobes) are primarily affected by degeneration in fvFTLD.

4.4. Parkinson's disease

Parkinson's disease (PD) is a common neurodegenerative condition clinically defined by motor symptoms, including bradykinesia,

rigidity, resting tremor, and postural instability. Non-motor symptoms may also occur, including cognitive impairment and neuropsychiatric disturbances.

Given the neuronal correlates of cognitive and affective ToM and the neuropathology of PD, assessment of social cognition may be particularly interesting. Indeed, the cognitive and affective subcomponents of ToM have been linked to different frontostriatal circuitry (Bodden et al., 2010), neuronal pathways that are affected in PD (Yoshimura and Kawamura, 2005; Zgaljardic et al., 2006). When differentiating between affective and cognitive ToM, the affective subcomponent is regarded as mediated predominantly by the orbital frontostriatal (OFS) circuitry, whereas the cognitive subcomponent may also be associated with dorsolateral frontostriatal (DLFS) circuitry. During the early stages of PD, dopamine is depleted most from the most dorsolateral portion of the head of the caudate nucleus, an area involved in DLFS circuit, whereas the processes based on the OFS circuit are almost completely preserved. As PD progresses toward later stages, in which the prefrontal cortex is directly affected by the neuropathology of PD, dopamine depletion within the striatum also affects the OFS circuit, impairing related functions.

4.4.1. Cognitive and affective ToM

Findings concerning cognitive ToM abilities in PD are varied and seem to depend largely on the methodology. Heterogeneous results were obtained when assessing cognitive ToM (Mengelberg and Siegert, 2003), in that the PD patients did not perform as well as did controls on a false belief test, a short passage test (in which participants were asked to make inferences about mental states or physical causation), and on a first-order test, but performed equally well on a second-order story test. In contrast, another study found that PD patients were significantly less accurate than healthy controls in assigning second-order beliefs during a story comprehension task, suggesting that the ability to make a second-order mental state attribution declines in patients with PD (Monetta et al., 2009). PD patients were also less able to determine whether the final statement of a story should be interpreted as a joke or a lie, suggesting a failure in pragmatic interpretation abilities. A recent study, using the Yoni Test (Shamay-Tsoory and Aharon-Peretz, 2007), confirmed these findings, by showing that non-demented PD patients were impaired in cognitive ToM, and more precisely in second-order ToM, but that first-order ToM was preserved (Bodden et al., 2010). Another study found that cognitive ToM abilities (assessed using the cognitive subcomponent of the Faux-Pas Test) were impaired in patients with advanced, but not early, PD, whether or not such patients were medicated (Peron et al., 2009), suggesting that ToM dysfunction progresses as the disease develops in severity. A second study on the effect of PD medication on ToM, using the same assessment tool (i.e., the Faux-Pas Test) yielded different results (Roca et al., 2010). The latter work found a significant difference in performance evaluated using the cognitive component of the Faux-Pas Test, in that healthy controls outperformed both medicated and unmedicated early PD patients. However, the former study (Peron et al., 2009) used an abbreviated version of the Faux-Pas Test, which may have decreased test sensitivity. Moreover, assessing the same early PD patients under both "on" and "off" conditions may have induced a learning effect. The use of more comprehensive, and thus more sensitive, tests may allow detection of cognitive ToM deficiencies during early stages of PD (Roca et al., 2010). One study, employing a wide range of ToM tasks (Saltzman et al., 2000), found that, compared with healthy controls, PD patients showed poorer performances on the false belief test and in the Spy Test (Hala et al., 1991). This finding suggests that PD patients were less able to make correct predictions based on inferences about a story character's beliefs and that these patients had greater difficulties planning a course of action that could deceive another person. Nevertheless, the performances of PD patients on the Perspective-Taking Test (Chandler and Lalonde, 1996) and the Knower/Guesser Test (Povinelli et al., 1990) were preserved. Since these tasks involve cognitive aspects of ToM, the pattern of results using the four measures of ToM (Saltzman et al., 2000) suggests that these four measures may not evaluate the same underlying construct.

Studies of affective ToM using the Faux-Pas Test have generally yielded consistent results. While PD patients showed impaired performance on cognitive ToM tasks, there was evidence for preserved performance on the affective component of the Faux-Pas Test. PD patients and controls were equally able to detect inappropriate remarks in stories (the affective component of the task) but patients had greater difficulty inferring the reason the character in the story had made an inappropriate remark (the cognitive component of the task) (Kawamura and Koyama, 2007; Peron et al., 2009; Roca et al., 2010).

The RME Test is also frequently utilized to assess effective ToM in patients with PD. Most studies have suggested that performance of medicated PD patients may be preserved during early (Euteneuer et al., 2009; Mimura et al., 2006; Roca et al., 2010) and advanced (Peron et al., 2010, 2009) stages of the disease. In addition, a group of unmedicated *de novo* PD patients showed similar performance on the RME test as both medicated PD patients and healthy controls (Roca et al., 2010). Moreover, a study investigating the effects of surgery (Peron et al., 2010) reported no differences in RME Test results between PD patients prior to deep brain stimulation and healthy controls, whereas post-operative PD patients had lower performance than healthy controls and pre-operative PD patients. Only two studies to date have reported lower RME performances in PD patients than in healthy controls (Bodden et al., 2010; Tsuruya et al., 2011). In both studies, however, the mean age of the control group was lower than that of the PD group (58.7 years vs. 63.7 years and 67.7 years vs. 70.5 years, respectively). This is particularly important, since RME performance has been reported to decline with age in healthy subjects (Pardini and Nichelli, 2009).

To date, little has been reported on the role of cognitive status in the processing of facial emotional expressions in patients with PD. Evidence that verbal working memory is affected in such patients (Ellfolk et al., 2006; Gilbert et al., 2005; Koerts et al., 2009) may partially explain their difficulties in the cognitive (i.e., false belief tests and the cognitive component of the Faux-Pas Test) but not in the affective (i.e., the RME Test and the affective component of the Faux-Pas Test) component of ToM tasks. A significant correlation has been reported between ToM and executive functions (Saltzman et al., 2000), and, in advanced PD patients, a significant correlation has been found between the Stroop Interference score and the explanation subscore of the Faux-Pas Test (Peron et al., 2009). Moreover, an association between ToM and decision making has been observed (Mimura et al., 2006), as well as an impairment in verbal working memory associated with a set-shifting impairment (Monetta et al., 2009).

4.4.2. Emotion recognition

Studies assessing emotion processing in PD have found that these patients are impaired in their ability to recognize facial emotional expression, although there are discrepancies depending on the type of emotions involved. Generally, PD patients are impaired in imaging, perceiving, and expressing emotional faces (Jacobs et al., 1995) and in recognizing fear and disgust from facial expression (Kan et al., 2002). In addition, 56 PD patients with bilateral motor symptoms showed limitations in recognizing all facial emotion, in particular fear and sadness, whereas 8 patients with right-sided PD were particularly impaired in the recognition of sadness and disgust (Yip et al., 2003). Another study (Sprengelmeyer et al., 2003) found that PD patients were generally impaired in their

recognition of facial emotional expressions, with the recognition of fear, sadness, anger, and disgust all being compromised. Further analysis showed that unmedicated PD patients were specifically deficient in the recognition of disgust compared with medicated PD patients. Using the facial expression hexagon, PD patients were selectively impaired in recognizing the facial expressions of disgust (Suzuki et al., 2006) and anger (Lawrence et al., 2007). Non-verbal emotional information processing is disturbed early in the course of PD, with unmedicated PD patients being significantly impaired in decoding emotional facial expressions (Dujardin et al., 2004). Moreover, these PD patients were less accurate than controls in decoding anger, sadness and disgust from facial expressions, regardless of the level of intensity of the expression (low or mild). These findings were partially confirmed by another study investigating the influence of intensity on the identification of a facial emotional expression, in that patients with PD recognized fewer low- and high-intensity facial expressions of disgust than did controls. However, this effect was selective, because their global ability to recognize emotions (including anger and sadness) was intact. Both patients with PD and healthy controls recognized high-intensity better than low-intensity emotions, except for disgust, which was recognized better at low intensity (Assogna et al., 2010). Thus, these studies suggest that PD patients find it particularly difficult to recognize disgust, but that almost all of the other facial emotions have been described as under-recognized in individual studies. In contrast, several studies failed to demonstrate any difference between PD patients and controls in facial emotional tasks (Adolphs et al., 1998; Biseul et al., 2005; Pell and Leonard, 2005; Smith et al., 1996).

A deficit in spatial contrast sensitivity related to impaired recognition of fearful facial expressions has been reported in medicated PD patients (Sprengelmeyer et al., 2003). Recognition of facial emotional expressions has been found to correlate with executive but not visuo-perceptual function (Dujardin et al., 2004), and a relationship has been observed between disadvantageous decision-making and decreased emotional response (Kawamura and Kobayakawa, 2009). Many patients with PSP or DLB also develop executive dysfunctions. Thus, although the relationship or potential interaction between the two concepts is not yet fully understood, executive dysfunction in BG disorders may impede ToM performance (Bodden et al., 2010).

4.4.3. Empathy

To the best of our knowledge, only one study has assessed empathy in patients with PD. Using the IRI, it was found that patients with PD, assessed by caregivers, had preserved empathy (Narme et al., 2011).

4.4.4. Other Parkinson-plus syndromes

Among other Parkinson-plus syndromes are, dementia with Lewy bodies (DLB), progressive supranuclear palsy (PSP) and corticobasal degeneration (CBD). Lewy bodies are intracytoplasmic inclusions and in DLB, they such inclusions are primary located in limbic and secondary in neocortical brain areas (Rezaie et al., 1996). Indeed, a VBM study found that such patients showed very little cortical involvement; regional GM loss was observed primarily in the dorsal midbrain, the substantia innominata and the hypothalamus (Whitwell et al., 2007). In most patients with PSP, the midbrain shows marked atrophy (Dickson et al., 2011), with neuronal loss being predominant in the substantia nigra, the globus pallidus, the subthalamic nuclei, the dentate nucleus of the cerebellum and the red nucleus (Inoue et al., 1996). Whereas cerebral lesions in CBD are often diverse, the affected areas of subcortical regions are rather uniform (Ikeda et al., 1996). Asymmetrical frontoparietal atrophy, especially around the central sulcus, is the most common atrophic presentation (e.g., Mori et al., 1994; Rinne et al., 1994), whereas the most prominent site of subcortical degeneration is the substantia nigra. The inner segment of the pallidum and part of the thalamus show moderate degeneration (Ikeda, 1995).

As in PD, in the light of the fronto-subcortical loops, given the prominent affection of subcortical areas and the neural correlates of social cognition, assessment of these abilities appear particularly interesting. Indeed, dysfunctions in emotional facial expression decoding have been described in patients diagnosed with DLB and PSP (Kluger and Heilman, 2007; O'Keeffe et al., 2007). Reduced empathy abilities have been reported in patients with PSP and CBD, operationalized through the "Measure of Empathic Tendency" (MET) (O'Keeffe et al., 2007). Furthermore, patients with PSP and CBD have lower scores than healthy control subjects in the affective and cognitive IRI (Interpersonal Reactivity Index, Davis, 1980) subscales (Rankin et al., 2006). A case report of a 72-year-old woman with DLB and an MMSE score of 24/30 found that she performed worse than patients with AD and MCI, but better than those with fvFRLD. This patient's impairment was associated with the affected areas of her brain, the medial prefrontal cortex and its networks (Modinos et al., 2009).

Hence, although some studies have suggested a link between ToM and performance of executive tasks, others have not. Indeed, several studies have found no correlation between emotion recognition and cognitive functions (Adolphs et al., 1998; Kan et al., 2002; Lawrence et al., 2007; Pell and Leonard, 2005), or between ToM and executive functions (Roca et al., 2010). In addition, even if executive functions were significantly decreased in their PD patients, they were not correlated with ToM performance (Bodden et al., 2010; Mengelberg and Siegert, 2003). At present, however, this issue remains unclear and requires further investigation.

The neurochemical hypothesis of ToM has suggested that dopaminergic and serotonergic dysfunction may cause impairments in ToM (Abu-Akel, 2003b). According to this hypothesis, (i) the neuroanatomic regions critical for ToM (i.e., the prefrontal cortex, the TPJ and the AC cortex) are innervated by the dopaminergic and serotonergic system; (ii) cognitive abilities such as executive functions that contribute to ToM, as well as the language that depends on ToM can deteriorate following abnormalities in neurotransmission within one of these two systems; and (iii) the dopamine system is relevant for signaling predictions about the consequences of future events, an ability closely related to ToM. With the substantia nigra, the BG encompasses crucial dopaminergic cells.

Thus, knowledge in this field is far from exhaustive and is sometimes contradictory. Findings to date, however, suggest that ToM dysfunctions occur in a variety of BG disorders and that executive functions or language may account for ToM deficiencies.

In summary, patients with PD (or other Parkinson-plus syndromes) show relatively spared affective ToM abilities, but impaired cognitive ToM abilities and facial emotional recognition. The link between cognition, particularly executive functions, and ToM in these patients has not yet been determined, although specific disturbances in ToM abilities, even if partially mediated by executive dysfunctions, cannot be excluded. This hypothesis is supported by the fronto-subcortical loop model (Alexander et al., 1986), according to which two fronto-subcortical circuits likely to mediate ToM components correspond to the neuronal pathways affected in PD. Furthermore, these findings are consistent with results suggesting that, during early stages of PD, dopamine is most depleted in an area involved in the DLFS circuit, but is almost completely preserved in processes based on the OFS circuit. Indeed, although PD patients show relatively poor performance on cognitive ToM tasks, they generally succeed affective ToM tasks. Thus, the functional connection between the subcortical and frontal regions may be at least partially affected in PD.

4.5. Temporal lobe degeneration

The temporal lobe is part of the limbic system (Heimer and Van Hoesen, 2006) and has been linked to the processing of emotional and arousing stimuli (Beauregard et al., 2001; Berthoz et al., 2002). It may also have functions related to social processing, including face processing (Mesulam, 1998; Seeck et al., 1993), storage of social concepts (Zahn et al., 2007), and the processing of social semantic knowledge (Ross and Olson, 2010; Simmons et al., 2010). While the amygdala and OF cortex are the two neuroanatomical regions most frequently linked to socio-emotional processing, less attention has been paid to the temporal pole (TP), a region that lies between the amygdala and OF cortex and receives and sends connections to both regions. Due to difficulties in lesion studies focusing on the temporal lobes, the assessment of social cognition in patients with neurodegenerative pathology may be of interest. Atrophy of the right anterior temporal lobe in patients with the temporal variant of FTLD (tvFTLD) has been largely associated with changes in mood (e.g., emotional blunting, depression, irritability, apathy), personality (e.g., from extroverted to introverted; sudden acquisition of peculiar new interests and hobbies) and socially appropriate behavior (Bozeat et al., 2000; Miller et al., 1997, 1995; Mychack et al., 2001; Thompson et al., 2003). In contrast, there have been few studies to date assessing social cognition in a disease in which neurodegeneration appears to selectively, and often asymmetrically, target the amygdala and anterior temporal lobes, as well as the posterior/medial portion of the OF cortex (Chan et al., 2001; Edwards-Lee et al., 1997; Galton et al., 2001; Mummery et al., 1999; Rosen et al., 2002a) (i.e., semantic dementia (SD), tvFTLD).

4.5.1. Cognitive and affective ToM

A case study of a 60-year-old man with SD found specific ToM deficits as indicated by poor performance on false belief and judgment of preference tasks (Bon et al., 2009). Affective ToM, as determined by the RME Test, was relatively spared in this patient. The ToM deficiencies in this patient may not be directly related to temporal lobe damage but to the observed hypoperfusion in the OF cortex or to a dysexecutive syndrome.

4.5.2. Emotion recognition

Patients with tvFTLD involving primarily the right temporal lobe showed greater impairments in emotional comprehension, as well as increased emotional blunting and loss of empathy, than did patients with tvFTLD involving primarily the left temporal lobe (Perry et al., 2001). Similarly, emotional comprehension, (i.e., comprehension of facial expressions of emotions) was significantly impaired in patients with tvFTLD compared with controls, with more severe performance decreases for negative emotions (e.g., sadness, anger, fear) (Rosen et al., 2002b). Further analyses indicate that this deficit was correlated with atrophy of the right amygdale and right OF, the areas most frequently affected in fvFTLD. A case study of a patient with SD showed objectification of emotion recognition deficits (Calabria et al., 2009), with spared emotional word knowledge and preserved emotional aspects of empathy, but below the cut-off for all basic emotions on the standard version of the Ekman 60 Faces Test.

4.5.3. Empathy

Empathy has also been evaluated in patients with this pathology. Family members of patients with tvFTLD frequently complain that the patient exhibits a pervasive lack of empathy (Mychack et al., 2001). Furthermore, a study of a group of FTLD patients using the IRI found that patients with SD were impaired in both the emotional and cognitive components of empathy (Rankin et al., 2005). Subsequently, tvFTLD was shown to be associated with disruption of emotional empathy and perspective-taking, a measure of cognitive empathy similar to ToM (Rankin et al., 2006).

There have been few studies assessing social cognition in patients with degenerative pathologies specifically affecting the temporal lobes, limiting our knowledge of the effect of these conditions on social cognition abilities. The presence of ToM deficits, even if biased by confounding factors, cannot be excluded. Overall, however, findings on emotion recognition abilities are consistent, in that these abilities seem to be impaired in pathologies affecting the temporal lobes, especially with regard to the additional areas of the brain involved in tvFTLD (i.e., the amygdala and OF cortex).

5. Conclusions

Several major findings have emerged from studies assessing the effects of ageing on social cognition. First, disturbances in social cognition abilities during the course of normal cognitive ageing are at least partially independent of a more general cognitive decline. Second, brain areas found crucial for the integrity of ToM are specifically affected by neurodegenerative processes in patients with age-related pathologies. Therefore, declines in social cognition in older adults can be linked to the declines observed in these neurodegenerative pathologies.

Major findings in assessments of ToM in pathological ageing showed that (a) in patients with AD and MCI, the observed difficulties in ToM and emotion recognition may be related to other cognitive deficits rather than to a primary impairment in ToM, in agreement with the neuropathology of AD and MCI and the neuronal correlates underlying ToM; (b) patients with fvFTLD, in which the impaired cerebral regions have been implicated in social cognition processes, show specific impairments of social cognition, including ToM, emotion recognition, empathy and moral and conventional reasoning; (c) since PD primarily affects the two fronto-subcortical circuits likely to mediate ToM, the functional connection between the subcortical and frontal regions is probably disturbed, resulting in the observed social cognition deficiencies; (d) impairments in emotional recognition in tvFTLD patients has been related to the additional brain areas affected in this pathology.

Together, these observations suggest that, when brain areas likely to underpin social cognition abilities are relatively spared in specific neurodegenerative pathologies (e.g., AD and MCI), social cognition abilities are relatively unaffected. Any observed difficulties in patients with these pathologies are more likely due to a more general cognitive decline, rather than to a specific deficiency in social cognition. However, when neurodegenerative conditions specifically affect cerebral structures involved in social cognition, there are ToM and/or emotion recognition deficits. In fvFTLD, for example, the frontal lobes are particularly affected and impairments in social cognition are frequently severe and extend to all of its aspects. This is consistent with studies evaluating social cognition in tvFTLD. In this variant of FTLD, where neurodegeneration primarily affects the temporal areas and affects other areas (e.g., the OF cortex and the amygdala) to a limited extent, deficits in ToM and emotional recognition are less pronounced.

Social cognition has also been evaluated during normal ageing. Although some (13–33%) elderly individuals show preservation of ToM ability (Sullivan and Ruffman, 2004), age-related impairments were objectified when all of second-order cognitive ToM, affective ToM, and emotion recognition abilities (especially negative facial emotions) were studied. These findings are paralleled by neuroimaging data, showing that the frontal lobes, strongly implicated in ToM abilities, are among the most deeply affected

regions in elderly individuals. Indeed, accelerated gray matter loss in the dlPFC (Grieve et al., 2005) and a decline of dlPFC activation (Kwee and Nakada, 2003), as well as structural changes within the vmPFC (i.e., the OF cortex) appear with ageing (Convit et al., 2001; Resnick et al., 2000; Resnick et al., 2003). These brain areas are likely critical in terms of social cognition abilities. Moreover, although age-related declines in executive functioning and fluid intelligence appear to mediate the deterioration in ToM with ageing, this is not reducible to a global cognitive decline (e.g., Maylor et al., 2002; Sullivan and Ruffman, 2004). Thus, test results obtained during normal ageing suggest that, when progressive declines in ToM abilities are present in elderly individuals, such declines may be related to the increase in age-related vulnerability of the frontal lobes. This hypothesis is supported by findings showing that, when behavioral performance on affective ToM tests are similar in younger and older adults, the latter showed more bilateral activation of frontal areas (Castelli et al., 2010). Hence, to perform at the same level as younger adults, older adults may require functional compensation, as shown by increased neuronal activation in the frontal areas.

Nevertheless, social cognition abilities cannot be attributed to these areas exclusively. Subcortical structures, including the caudate nucleus and amygdala, have also been shown to be crucial. Although their integrity appears fundamental for normal emotion processing, these areas are not exclusively involved in this ability, in that damage to the subcortical structures can also result in impairment of social cognition abilities, including cognitive and affective ToM, more commonly attributed to frontal brain areas. Indeed, the model of fronto-subcortical loops suggests that the poor performances of elderly individuals on ToM tasks may result from disturbances in the frontal areas, due to BG dysfunctions. The integrity of the entire circuitry may therefore be necessary to preserve social cognition. That is, damage to one of the structures belonging to one loop may cause disturbances in connectivity among cortico-subcortical networks. This may, in turn, result in the impairment of social cognition (e.g., emotion recognition and/or cognitive ToM and/or affective ToM) to different degrees, depending on the affected area. This argument is supported by findings showing that social cognition deficits are not only observed in pathologies affecting the frontal brain areas. Indeed, patients suffering from PD, as well as those with the temporal variant of FTLD, experience difficulties in emotion recognition, with PD patients also experiencing difficulties in affective ToM. PD patients seem particularly impaired in the recognition of disgust and negative emotions, deficiencies consistent with the neuropathology of PD, namely the primary impairment of the BG, an area likely to be crucial in emotion processing. For example, one of the cerebral structures precociously affected in PD, namely the CN, was found to be involved in the recognition of disgust (Phillips et al., 1998). Moreover, increased activation in the caudate in response to negative images has been observed (Carretie et al., 2009), objectifying the importance of this nucleus in processing negative emotions. Similarly, damage to the amygdala is frequently observed in patients with tvFTLD. This structure is likely to play a core role in negative emotion recognition (Blair et al., 1999; Breiter et al., 1996), consistent with findings in patients with tvFTLD (i.e., impairment in emotion recognition, especially for negative emotions). These findings are of particular interest in explaining the deficiencies observed during normal ageing. Indeed, elderly individuals have been shown to exhibit difficulties in recognizing facial expressions of emotions, especially of negative emotions. Again, functional neuroimaging provided information that enhances understanding of the underlying mechanisms of emotion recognition difficulties in older adults. That is, viewing negative facial emotional expressions has been associated with increased activity in the amygdala in younger adults, but reduced activity in the elderly.

Appendix A.

A.1. Main tests in social cognition

A.1.1. First- and second-order false beliefs tasks

First-order false beliefs tasks were designed to test a subject's ability to infer that someone may have a mistaken belief that is different from that subject's own true belief, or, in other words, the ability to infer mental states of the type "A thinks X". These tasks require consideration of only one character's perspective. False belief tasks typically involve one person putting an object somewhere in the presence of another person and then leaving the room. The second person moves the object to another location while the first person is away. The first person returns, and the subject is asked three questions: (1) the "belief question", which asks where the first person thinks the object is, thus requiring an understanding of others' mental states; (2) the "reality question", which asks where the object really is; and (3) the "memory question", which asks where the object was at the beginning.

Second-order false belief tasks were designed to test the ability to understand what someone else thinks about what another person thinks. In each story, Person 1 places an object somewhere and then leaves the room. Person 2 moves the object. While Person 1 is out of the room, he or she peeks back in and sees the object being moved, but Person 2 does not know that Person 1 has seen this. The subject is asked, "When Person 1 comes back in, where will Person 2 think that Person 1 thinks the object is?"

A.2. Faux-pas Test

The Faux-Pas Test assesses an individual's ability to infer another's mental state. A faux-pas occurs when someone says something they should not have said, not knowing or not realizing that they should not say it. To understand that a faux-pas has occurred, an individual has to represent two mental states: that the person saying it does not know that he or she should not say it and that the person hearing it would feel insulted or hurt. In other words, understanding a faux-pas requires understanding both a mental state of belief or knowledge and having some empathic understanding of how the person in the story would feel. Thus there is both a cognitive component and an empathic affective component. Subjects were read 10 stories that told about the occurrence of a faux-pas and 10 control stories reporting a minor conflict, but in which no faux-pas was committed. After each story, participants are asked the following types of questions: (1) did someone say something they should not have said? (Tests detection of faux-pas), (2) who said something they should not have said? (Tests understanding of faux-pas), (3) why should not they have said it? (Requires understanding of the mental state of the listener), (4) why did they say it? (Requires understanding of the mental state of the speaker), (5) did X know that Y, . . .? and (6) how do you think Y felt?

A.3. Reading the Mind in the Eyes Test

The Reading the Mind in the Eyes (RME) Test measures the ability of an individual to determine the mental state of another individual by looking at a picture of the latter's eyes. The task consists of 36 items, showing the eye region of black and white photographs of 36 different faces, both male and female. Each picture has four mental state terms printed below it, and the participant has to choose the word that best describes what the person on the photograph is feeling or thinking. Thus, this test evaluates the visual side of ToM. During the test, participants have the opportunity to

consult a glossary containing synonyms and examples of each word to ensure their comprehension of that word.

A.4. Emotion recognition

These tests are used to assess an individual's ability to identify emotions in others. Frequently, participants are presented with black and white photographs of faces (Ekman and Friesen, 1976; Matsumoto and Ekman, 1988, for a colored version) in the middle of a computer screen. Underneath each photograph is a series of adjectives describing different emotions (i.e., happy, sad, disgusted, frightened, surprised, angry or neutral). Participants are instructed to choose the adjective that best describes the emotion displayed on the face in the photograph. Each photograph remains on the computer screen until the participants make a decision and the participants do not receive any feedback on whether the decision is correct or not. The photographs are presented in a pseudorandom order, and the adjectives always remain in the same position on the computer screen.

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