CHAPTER 33

THEORY OF MIND AND PSYCHOPATHOLOGY

Theory of mind (ToM) is a fairly new concept and field of study. It promises to advance our understanding mankind in general and psychopathology in particular.

Social functioning depends on managing social relationships. The necessary skills include ToM, recognition of social signals, social knowledge, emotion processing, attention, working memory and decision making (Green and Leitman, 2008).

ToM refers to the ability to attribute mental states (such as thoughts, beliefs, desires and intentions) to people (yourself and others). In lay terms it roughly means “being able to tell” what other people are thinking and feeling.

ToM has received attention, not only because it is fascinating, but also, because it is a new way of studying clinical conditions, particularly autism and schizophrenia, but also others (see below)

The term, ToM, was first used by primatologists and psychologists Premack and Woodruff (1978) when they asked: “Does the chimpanzee have a theory of mind?”

Don’t worry about chimpanzees. Of greater importance at this point, is whether you have a ToM. Please attempt the following test:
Illustration. (Adapted from Baron-Cohen et al, 1986). If your answer was: you thought Sally would think her ball was in Anne’s box, we have a problem. It would be better if you had thought that Sally would think something which you knew to be wrong. Now, read on.

ToM involves “thinking about people thinking about us”, and recognition/understanding, that others have minds like your own.

ToM may give an evolutionary advantage. It reduces our susceptibility to deception. In social situations, people with good ToM skills outmanoeuvre those with poor ToM skills. And as mentioned, ToM dysfunction is a feature of some mental disorders.

ToM is distinct from many cognitive functions, but how ToM and the executive functions are related is yet to be fully explained (Yeh, 2013; Mitchell R, Phillips, 2015). Various tests of ToM utilized different cognitive mechanisms (Ahmed & Miller, 2010).

ToM can be viewed as having two components 1) cognitive ToM, which refers to the ability to make inferences about beliefs, thoughts, desires, motivations and intentions of others, and 2) empathetic/affective ToM, which refers to the ability to infer the feeling/emotions of others (Shamay-Tsoory et al, 2007). A recent fMRI study suggests that both cognitive and affective ToM are associated with activity in the superior temporal sulcus/temporo-parietal junction (STS/TPJ). In addition, affective ToM was associated with activity in the medial prefrontal cortex (mPFC; Sebastian et al, 2011).

The process by which ToM conclusions about others are reached may involve a two stage process, 1) the individual must first, inhibit perception of the self, and then 2) apply reasoning. Inhibition of one’s own perspective is believed to depend on activity in the inferior frontal gyrus (IFG; Russell et al, 2000). The reasoning component of ToM is believed to be mediated by the left superior temporal gyrus (STG) and TPJ (van der Meer et al, 2011).

**Evolution, theory of mind and psychopathology**

ToM is almost exclusively a human attribute.

ToM probably emerged during the late Pleistocene (sometime before 10 000 years ago), when social environments became increasingly complex and placed increasing evolutionary pressure on the primate brain (Whiten, 2000).

Increased size (and therefore increased complexity) of social groups raised the demand for information processing capacity. This is consistent with the positive association between the size of the group and the size of the neocortex of the different primate species (Dunbar, 2003).

Evolution requires the transmission of genes, and this is facilitated when the individual lives a long life, and is attractive to members of the opposite gender.
In our ancestral human environment (and to some extent in current life) homicide by gangs was/is a major cause of the shortening of life of males. Accordingly, fear of gangs may be adaptive. This may be reflected in the common psychiatric delusion that one is being observed by groups, such as police, bikies and drug dealers.

In the wild, the attractiveness of the male to female primates depends largely on status: the higher the status, the “better” the male genes, and the better are the chances of survival of female genes when combined with high status male genes. This may be reflected in the psychiatric disorder of erotomania, in which a female (usually) believes a high status male wants her as a partner.

In the wild, the attractiveness of the female to male primates is based on beauty/physical health (the more healthy the female, the more chance the male genes will survive). This may be reflected in the psychiatric disorders in which individuals have delusions about their state of health or physical appearance (delusions about having diseases and blemishes when none exist).

Jealousy may provide an evolutionary advantage. Jealousy is a characteristic of species in which the male contributes resources (energy and materials) to his offspring after their birth. In this manner, the male is increasing the chances of the survival of his own genes. Accordingly, the male is particularly concerned about the sexual fidelity of his partner, lest he contribute resources to the genes of another male. This may be reflected in pathological jealousy - the incorrect belief that a partner has been sexually unfaithful, which is most often encountered in males.

The female is concerned to receive continued support (and resources) from a male, to increase the chance of survival of her young (genes). The female is therefore particularly concerned about the emotional fidelity of her partner (as this is likely to translate into longer term support).

The above paragraphs suggest a connection between evolution, ToM and delusions. While ToM deficits are substantiated in schizophrenia (see later) they do not appear to be fundamental to delusions (Garety & Freeman, 2013). Further work is needed to examine these ideas.

**Ontogeny**

Spindle cells (see below) are not present at birth in humans, but appear at around 4 months of age (Allman et al, 2001).

The ToM system (see below) probably begins to operate in the human from about 18 months of age. Before 3-4 years of age the child is unable to distinguish between his/her knowledge of the world and that of other people. At 5-6 years of age the child can hold beliefs about another person’s beliefs. At 9-11 years, faux pas (breach of etiquette) can be understood (Frith and Frith, 2003).
Refinement of the ToM capacity continues through adult human life (Brune and Brune-Cohrs, 2006).

**Cells**

Three types of cells (at least) appear to be important in ToM.

1. **“Mirror neurons” (MNs)**
   MNs are unique cells which are activated in two circumstances, 1) when the individual performs an action, and 2) when the individual observes another individual performing that action (DiPellegrino et al, 1992).

   They are found in humans and to a lesser extent in other great apes, and some monkeys. They are located predominantly in the inferior frontal gyrus (IFG; BA 44 & 45) and the inferior lobule of the parietal lobe (IPL) (Gallese & Goldman, 1998).

   Studies in which screens have been used to obscure components of movements by actors have shown that although complete movements have not been observed, MNs are nevertheless activated. This has been taken as evidence that the MNs are activated according to the “intention” of the movement, rather than the movement per se. And, thus, MNs allow us to understand the “intentions” of others. MNs are specific for particular movements: grasping, reaching, holding and tearing neurons have been identified. Other MNs respond to sounds; there are neurons for the “sound of scratching” and others for the “sound of tearing”.

   Non-humans have very limited capacity for behavioural copying/imitating. This is consistent with a relative absence of MNs. Performing monkeys which wear clothes and behave like humans have been taught, rather than developed their own routines.

   Mehta et al (2013) using a transcranial magnetic stimulation (TMS) methodology suggest a deficit MN system underpins the social cognitive deficits of schizophrenia. However, Andrews et al (2015) failed to find support this suggestion.

2. **von Ecomomo neurons (VEN)**
   VEN (spindle cells) may be important in inhibiting mirror cells, and enable suppression of immediate social responses. They are located in the anterior cingulate cortex (ACC) of humans and the great apes, but not monkeys. The density of these cells in different species correlates inversely with genetic distance from humans (Nimchinsky et al, 1999). The presence of spindle cells in human brains is evidence that the ACC (an old structure) has undergone recent (last 15-20 million years) evolutinal change.

3. **Special somatosensory cells**
   Certain cells of the somatosensory cortex II (SCII, located on the parietal operculum – upper edge of the Sylvian fissure) are activated when a subject observes another person being touched (Keyser et al, 2004). This “vicarious recruitment” of cortical somatosensory cells suggests that SCII should be added to the premotor cortex as part of the system which underpins empathy and useful social perception (Keyser et al, 2010).
**Chemistry and the “social brain”**

“The social brain hypothesis” (Dunbar, 1998) includes ToM and other notions of social information processing (Krah et al, 2010).

The neural circuits involved with social attachment (pair bonding, mother-offspring relationship) include a reward circuit which involves the ventral tegmental area (VTA), nucleus accumbens (NAc), pallidum, thalamus, and prefrontal/cingulated cortex (Insel, 2003; Everitt & Wolf, 2002). A component of this circuit is the mesolimbic tract.

Dopamine is a major neurotransmitter in this reward circuit. However, various other agents are involved, including oxytocin (OT) and vasopressin (AVP), which are both stored in the posterior pituitary. OT is released in response to sucking (and has a role in establishing the mother-offspring bond), and both OT and AVP are released by viginocervical stimulation during labour and copulation (and have a role in pair bonding; Insel, 2003). OT and AVP both have a role in “social memory”.

Illustration. Reward circuit (Insel, 2003)

Dopamine is the neurotransmitter of the mesolimbic tract. In a related manner, dopamine is also involved in the nigrostriatal tract, which is dysfunctional in Parkinson’s disease, a condition in which ToM is reduced (Bora, et al 2015).

[Insel (2003) makes the fascinating observation that the reward system associated with reproductive behaviour is “hijacked” by drugs of addiction.]
Mirror Neuron System (MNS)

The MNS (Iacoboni and Mazziotta, 2007) refers to the structures which activate in association with MNs. Debate continues as to the “key” structures (Ciaramidaro et al, 2007).

It is believed that actions performed by others are processed in the visual system, and then mapped onto the motor cortex of the observer, which performs the same action (Rizzolatti et al, 2009). The occurrence of this neural pattern in the observer enables an understanding of the actions of the observed.

Along with inferior frontal gyrus (IFG) and inferior parietal lobe (IPL), the posterior superior temporal sulcus (STS) is an important component of the MNS. This region contains cells which activate when actions are observed, but not when activation is executed. Gallagher and Frith (2003) proposed that initial analysis of social cues occurs in this region.

When a movement is observed there is a progression of neural activity across the brain in the following order (Nishitani et al, 2002).
1. occipital cortex
2. STS (superior temporal sulcus)
3. IPL (inferior parietal lobe)
4. IFG (with inferior frontal gyrus)
5. primary motor cortex.

Further, brain areas involved in emotional process (such as the anterior insula and cingulate cortex) are activated when we observe the emotions of others. And, it is proposed that “motor simulation may be a trigger for the simulation of associated feeling states” (Bastiannsen et al, 2009).

While discovery of the MN and the MNS has been exciting, some reservations have been expressed (Pascolo et al, 2010).
**Somatosensory activation contribution**

That certain SCII cells are activated when others are observed being touched is a recent discovery. It is probable that this phenomenon also contributes (along with the MNS and other potential systems) to ToM skills.

**ToM in clinical disorders**

ToM skills are recently evolved and finely tuned, and depend on a high degree of biological, psychological and sociological integration. Finely balanced equipment is easily disrupted, and it would be reasonable to expect that some psychiatric disorders are underpinned by disrupted ToM processes (Brune et al, 2003; Brune and Brune-Cohrs, 2006).

**Developmental disorders**

Children with autism have difficulties with emotional relationships and language acquisition. They have markedly impaired ToM skills (Baron-Cohen, 1988). With maturation, in the teen years, the performance of people with autism on ToM tests, may improve. Interestingly, the lack of ToM skills means these children are unable to deceive others, or to recognize when they are being deceived by others. [The parents of some children with autistic-spectrum disorders claim their children are “good”, because they do not lie – it needs to be remembered that, they cannot lie]. People with less severe autistic-spectrum disorders have less impairment of ToM skills.

There have been conflicting reports about whether MN dysfunction is (Perkins et al, 2010) or is not (Fan et al, 2010) a feature of autism. However, there is no doubt that ToM is impaired in autism spectrum disorders (ASD; Schneider et al, 2013).

**Personality disorder**

People with psychopathy are described as being cold and lacking in empathy. This suggests they would perform poorly on ToM tasks. Contrary to expectations, however, people with psychopathy appear to have unimpaired ToM skills (Blair et al, 1996). It is believed that people with psychopathy are aware of the distress they cause others, but are not distressed by these facts.

**Delusional disorder (DD)**

ToM is believed to have roots in the evolutionary process, and the “pure” delusions of DD (fear of gangs, jealousy, erotomania and somatic) have been proposed as having had survival value. These delusions, which arise exclusively in a social context, have been labelled, “Theory of Mind Delusions” (Charlton and McClelland, 1999; Charlton, 2003).

However, people suffering DD may perform normally on ToM tests (Walston et al, 2000; Bommer and Brune, 2006). This may suggest that current ToM tests are not valid. Alternatively, there may be an explanation in differences between the DD delusions and the “bizarre” delusions of other psychoses.
A distinction has been drawn between DD delusions which begin with a single false premise (about the intentions of others), but which are thereafter logical, and the bizarre delusions of vindictive Gods, magic carpets and interplanetary travel of other psychoses (such as may occur in schizophrenia), where logic is repeatedly and clearly impaired (Charlton and McClelland, 1999; Charlton, 2003).

With respect to delusions in general, not specifically those of DD, Freeman (2007) found that ToM deficits may be present, but “they are certainly not specific or necessary”. With respect to delusions in schizophrenia, Pousa et al (2008) reported, “specific ToM deficits were found associated with delusions”.

Schizophrenia

Frith (1992) raised the possibility of ToM deficits underpinning schizophrenia. He offered a comprehensive theory, with different types of ToM skills impairment accounting for the different symptom groups: positive, negative and disorganization symptoms.

Spong et al, (2007) conducted a meta-analysis of studies in schizophrenia and found a highly significant ($P < 0.0001$) effect, with significant ToM impairment in all symptom groups (negative, positive and disorganized).

Bora et al (2009) conducted a meta-analysis of 36 studies. In remitted schizophrenia patients the degree of ToM impairment was less pronounced than non-remitted patients, but was still significant. They found evidence of a trait relating to ToM impairment. This is consistent with the finding of Dworkin et al (1993) of impaired social functioning (but not specifically ToM) in the relatives of people with schizophrenia.

Bora and Pantelis (2013) found that ToM was substantially impaired at the first episode of schizophrenia, and the deficits were comparable with findings in chronic patients. They also found measurable deficits in ultra-high risk individuals and unaffected relatives (replicated by Cella et al, 2015 and Ho et al, 2015).

ToM is, of course, not the whole story. Other components of social cognition and cognitive deficits are surely involved in schizophrenia, but the evidence indicates a role for ToM deficits in at least some individuals (Langdon et al, 2008).

Gavilan Ibanez and Garcia-Albea (2013) report, “In schizophrenia, the deficit in ToM appears to be specific and not dependent on more general cognitive abilities…”

Brune (2005) made the observation that people with schizophrenia, in contrast to people with personality disorder, rarely cheat or manipulate others (including their therapists). This makes theoretical sense: with impaired ability to understand the mind of others, the capacity to manipulate the minds of others would be reduced. The other side of this coin fits with the clinical observation that it can be difficult to establish an empathic relationship with some people with schizophrenia.

A recent study of patients with schizophrenia demonstrated ToM deficits were positively correlated with grey matter reductions in the STS and medial prefrontal cortex (Koelkebeck et al, 2013).
Depression
A recent study of females with major depression (Fischer-Kern et al, 2013) found a significantly lower ToM capacity compared to controls, and deficits were related to illness duration and number of hospitalizations. (See also, Cusi et al, 2013.)

Other disorders
Various other disorders have been associated with ToM deficits, including,
- bipolar disorder (Kerr et al, 2003), apparently associated with mood symptoms and cognitive deficits (Ioannidi et al, 2015),
- eating disorders (Laghi et al, 2013; Tapajoz et al, 2013),
- alcohol use disorder (Bosco et al, 2013),
- Huntington’s disease (Brune et al, 2011),
- frontotemporal dementia (Gregory et al, 2002),
- callosal agenesis (Booth et al, 2011),
- multiple sclerosis (Nature, 2013),
- Alzheimer’s disease (Moreau et al, 2015)
- Alcohol dependence (Maurage et al, 2015) and
- Parkinson’s disease, even at the early stages, appears to feature impaired ToM (Poletti et al, 2013). These deficits are not related with performance on executive functioning (Bora, et al 2015)

ToM tests
ToM testing commenced in the study of autism. There have been advances, but standardised techniques have not been widely agreed.

The Sally and Anne Test (Baron-Cohen et al, 1986) which the reader attempted (hopefully successfully) at the beginning of this chapter is a “second-order” task, by which is meant one other person’s mental state must be read/understood. Normal children will pass this test by 3-4 years of age. Children with autism may not pass such tests; if they do so, it is usually at an older age than those without autism.

“Third-order” false belief tasks involve reading/understanding what 2 other people think sequentially, such as, what John thinks Mary thinks. Normal children will pass such tests at around 6 years of age. High functioning children with autism may also pass these tests, but not before their teen years.

Other tests include the ability to understand metaphor, sarcasm, humour and faux pas (breach of etiquette; Baron-Cohen, 2001).

Theory of Mind Picture Stories Task
Dr Martin Brune of University of Bochum, Germany, (Martin.Bruene@ruhr-uni-bochum.de) developed this test (Brune 2003), and has given permission for these 2 (out of 6) stories to be reproduced.

Each story is 4 pictures which the subject is to arrange into a logical sequence. Subjects are then asked a short series of questions. A simple scoring system is then applied. Examples of the pictures arranged in the correct order and the appropriate questions follow:
Questions:
1a) What does the person with the red shirt believe, the one in blue shirt intends to do?
   (2nd order belief) (pointing to 2nd picture)
   Correct answer: Get apple from tree

1b) What does the person with the red shirt expect from the person in the blue shirt?
   (reciprocity)
   (pointing to 4th picture)
   Correct: Give him part of the apple; share with him
Questions:
2a) What does the person with the blue shirt believe is in the bag?  
   (false belief) (pointing to 2nd picture)
   Correct: Gift, present, flower, (bug is incorrect)

2b) What’s in the bag? (reality) (pointing to 2nd picture)
   Correct: Wasp, bee, insect, or bug

2c) What does the person in blue shirt believe the person in red intends to do?  
   (2nd order false belief) (pointing to 2nd picture)
   Correct: Give him a gift or present

2d) What does the person in red assume the person with the blue shirt believes,  
   regarding his (the one in red) intentions? (3rd order false belief)  
   (pointing to 2nd picture)
   Correct: Give him a gift or present

2e) What do you think the person in the red shirt intended to do?  
   (deception) (whole story)
   Correct: Scare him, frighten him, shock him

Faux Pas

A faux pas represent a breach of etiquette, and recognition calls for a high level of  
ToM.

Herold et al (2009) gave this example:
Jill had just moved into a new apartment. Jill went shopping and bought some new  
curtains for her bed room. When she had just finished decorating the apartment, her  
best friend, Lisa, came over. Jill gave her a tour of the apartment and asked, “How do  
you like my bedroom?”  
“Those curtains are horrible” Lisa said, “I hope you are going to get some new ones!”

Questions
1. Did Lisa know the curtains were new?  
2. Did some one say something she shouldn’t have said?

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References


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