SPECIAL ISSUE FOR THE NORDIC MEETING IN AALBORG
THE SOCIAL BRAIN: DEVELOPMENT AND DYSFUNCTION
It was – «in the words of» former International Olympic Committee president Juan Antonio Samaranch – the best Nordic meeting ever. Even though the weather gods did not cooperate fully, the Danish organizers and volunteers put up an extensive and very well organized program, and the Danish well known kindness made us all feel at home. The social program was fantastic; a little rain does not stop a Nordic picnic!

The topic of the meeting – the social brain – was timely and well received. The methods and concepts of social cognition were initially used autism and neuroimaging studies, and they are now being extended to both neurologic and psychiatric patient groups. It was the aim of the conference to present the relevance of this new and exciting field of social cognitive neuroscience for neuropsychologists. As this issue of Neuropsykologi on the meeting attests, this aim was met.

The meeting started on Sunday August 15th with workshop sessions at the University Hospital, led by Lucia W. Braga (family based approach to rehabilitation of the child after brain injury), Maria Råstam (assessment of adults with Asperger syndrome), Paul Burgess (assessment of executive functions) and Anders Fjell (developmental aspects of brain and cognition).

There were two oral paper sessions during the meeting, where mostly nordic contributors presented their work and invited to discussion. The first oral session was on the topic of “childhood: development and dysfunction” and the second oral session focused on the topic of “intervention studies in experimental and clinical neuropsychiatry”. These sessions, as well as the poster sessions, showed the extent to which our Nordic colleagues are involved in state-of-the-art research on a variety of topics, many with cooperation with international groups. This meeting did indeed give opportunities for new such international cooperations to form.

The invited speakers were many (15), and they were all international experts within their fields. Also, the meeting presented ten very interesting symposia, where both Nordic and international colleagues presented and contributed to the discussions. In this issue we will give summaries from these presentations and symposia.

This issue is full of pictures from the meeting, all provided by Anders Gade who also served as a co-editor. Thank you Anders!

Thanks also to all the contributors to this volume.
Everyday social interaction is often dominated by categorical thinking, with generic knowledge structures (e.g., stereotypes) guiding people’s dealings with others. The primary benefit of categorical thinking is thought to reside in the cognitive economy it provides during impression formation and response generation. Challenging this viewpoint, the Macrae’s talk suggested that the origins of categorical thinking lie instead in the workings of early perceptual operations. A series of behavioural and imaging experiments were described that explore: (i) the efficiency of person categorization; (ii) the conditions under which person categorization and identification go awry; and (iii) how stereotype-based beliefs shape the neural operations that support person understanding.
Social interaction plays a critical role in early brain development. Recent research demonstrates that the social brain undergoes protracted development, and that adolescence in particular represents a period of reorganization of the social brain.

**HUMANS ARE EXQUISITELY SOCIAL**

Humans are an exquisitely social species. We are constantly reading each others’ actions, gestures and faces in terms of underlying mental states and emotions, in an attempt to figure out what other people are thinking and feeling, and what they are about to do next. This is known as theory of mind or mentalising. Developmental psychology research on theory of mind has demonstrated that the ability to understand others’ mental states develops over the first four or five years of life. While certain aspects of theory of mind are present in infancy, it is not until around the age of four years that children begin explicitly to understand that someone else can hold a belief that differs from one’s own, and which can be false. An understanding of others’ mental states plays a critical role in social interaction because it enables us to work out what other people want and what they are about to do next, and to modify our own behaviour accordingly.

**THE SOCIAL BRAIN**

Over the past 15 years, a large number of independent studies have shown remarkable consistency in identifying the brain regions that are involved in theory of mind or mentalising. These studies have employed a wide range of stimuli including stories, sentences, words, cartoons and animations, each designed to elicit the attribution of mental states (see Amodio & Frith, 2006, for review). In each case, the mentalising task resulted in the activation of a network of regions including the posterior STS at the temporo-parietal junction (TPJ), the temporal poles and the dorsal medial PFC (mPFC; see Burnett & Blakemore, 2009). The agreement between neuroimaging studies in this area is remarkable, and the consistent localisation of activity within a network of regions including the pSTS/TPJ and mPFC, as well as the temporal poles, suggests that these regions are key to the process of mentalising.

Brain lesion studies have consistently demonstrated that the superior temporal lobes (e.g. Samson et al., 2004) and PFC (e.g. Stuss et al., 2001) are involved in mentalising, as damage to these brain areas impairs mentalising abilities. Interestingly, one
study reported a patient with large PFC damage whose mentalising abilities were intact (Bird et al., 2004), suggesting that this region is not necessary for mentalising. However, there are other explanations for this surprising and intriguing finding. It is possible that, due to plasticity, this patient used a different neural strategy in mentalising tasks. Alternatively, it is possible that damage to this area at different ages has different consequences for mentalising abilities. The patient described by Bird and colleagues had sustained her PFC lesion at a later age (62 years) than most previously reported patients who did show impairments in mentalising. Perhaps mPFC lesions later in life spare mentalising abilities, whereas damage earlier in life is detrimental. MPFC may be necessary for the acquisition of mentalising but not essential for later implementation of mentalising. Intriguingly, this is in line with recent data from developmental fMRI studies of mentalising, which suggest that the mPFC contributes differentially to mentalising at different ages.

**DEVELOPMENT OF MENTALISING DURING ADOLESCENCE**

Developmental fMRI studies of mental state attribution have consistently shown that mPFC cortex activity during mentalising tasks decreases between adolescence and adulthood. Each of these studies compared brain activity in young adolescents and adults while they were performing a task that involved thinking about mental states (see figure 1 for details of studies). In each of these studies, mPFC activity was greater in the adolescent group than in the adult group during the mentalising task compared to the control task. In addition, adolescents tend to show stronger connectivity between mPFC and other parts of the mentalising network than adults (Burnett & Blakemore 2009).

It is not yet understood why mPFC activity decreases between adolescence and adulthood during mentalising tasks. Two non-mutually exclusive explanations have been put forward (see Blakemore, 2008, for details). One possibility is that the cognitive strategy for mentalising changes between adolescence and adulthood. A second possibility is that the functional change with age is due to neuroanatomical changes during this period. Decreases in activity are frequently interpreted as being due to developmental reductions in grey matter volume, presumably related to synaptic pruning. However, there is currently no direct way to test the relationship between number of synapses, synaptic activity and neural activity as measured by fMRI in humans (see Blakemore, 2008, for discussion). If the neural substrates for social cognition change during adolescence, what are the consequences for social cognitive behaviour?

**ONLINE MENTALISING USAGE IS STILL DEVELOPING IN MID-adoLESCENCE**

Most developmental studies of social cognition focus on early childhood, possibly because children perform adequately in even quite complex mentalising tasks from around the age of four. This can be attributed to a lack of suitable paradigms: generally, in order to create a mentalising task without ceiling effects for older children, the linguistic and executive demands of the task must be increased. This renders any age-associated improvement in performance difficult to attribute solely to improved mentalising ability. However, the protracted structural and functional development in adolescence and early adulthood of the brain regions involved in theory of mind might be expected to affect mental state understanding.
Recently, we adapted a task that requires the online use of theory of mind information when making decisions in a communication game, and which produces large numbers of errors even in adults (Keysar et al., 2003). In our computerised version of the task, participants view a set of shelves containing objects, which they are instructed to move by a “Director,” who can see some but not all of the objects (Dumonthiel et al., 2010; figure 2). Correct interpretation of critical instructions requires participants to use the director’s perspective and only move objects that the director can see. We tested participants aged between 7 and 27 years and found that, while performance in the director and a control condition followed the same trajectory until mid-adolescence, the mid-adolescent group made more errors than the adults in the director condition only. These results suggest that the ability to take another person’s perspective to direct appropriate behaviour is still undergoing development at this relatively late stage.

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Uta Frith started out with two claims: 1) Claim one says that there is a start-up kit in the human brain which allows humans to develop the ability to mentalise. Human beings from early infancy on attribute mental states to others and to themselves. In this way they can predict and explain behaviour. For instance, John will bring an umbrella, because he believes it is going to rain. 2) Claim two argues that the autistic brain lacks this start-up kit.

To remind us of the current knowledge of autism, Uta Frith shortly explained that autism is a neuro-developmental disorder with a basis in the brain and with a genetic predisposition. It affects almost 1% of the population with a 3:1 male:female ratio. The biological cause is not yet identified, but the cognitive mechanisms are partly identified. Autism is usually diagnosed in the second or third year of life but can be identified later as well. The behavioural criteria differ according to severity, age, ability, education, compensation, temperament, etc. It is therefore difficult to talk about autism as an entity. Rather it is a spectrum of autistic disorders where Aspergers syndrome is in the mild end of the spectrum because they are better at compensating.

The core feature of autism is the lack of mentalising which may explain why autistic individuals have impairments in reciprocal interaction and communication. The impairments in reciprocal interaction and in verbal and non-verbal communication give a restricted and repetitive repertoire of activities.

To understand how mentalising failures interact
with the biological factors, the environmental factors and the social factors have to relate to cognitive mechanisms. In a 3 level framework (biological, cognitive and behaviour) where environmental factors operate on all levels, the cognitive level (with mentalising and start-up kit) links the biological and behaviour level. For instance, the way a child mentalises will be seen in pretend play, where the child can have poor make-believe but good factual memory. Or poor joint attention but good focused attention.

When children grow up they develop their ability to mentalise. The ability in mentalising reached by normal children at 5 years may be reached by children with autism at perhaps 8 or 10 years of age (Happé, 1995). At this age most children with autism can pass the False Belief tasks in the Sally & Ann and similar tests. It is also the case that children with Asperger syndrome may pass the classic mentalising tasks at the same age as normal children. But then how can mentalising deficits be central to autism?

According to Frith, in real life children with Asperger syndrome and adult autistic people still find it hard to infer what other people are thinking, and to predict what they are going to do. The hypothesis is that they lack spontaneous mentalising ability, and that they have learned to pass false belief tasks by compensatory learning. To test this hypothesis Uta Frith has studied spontaneous mentalising by using animated triangles (White et al., in press). This task has also been used by Kana et al. (2009) in fMRI studies, demonstrating both an activation of the entire ToM network in normal subjects, and a lack of functional connectivity of these areas in autistic subjects during the attribution of mental states.

Another test was developed to test implicit mentalising. It is called the Eye-Gaze paradigm, and it is difficult enough that even normal adults tend to produce errors (see figure 2 and description in Sarah-Jayne Blakemore’s article, this issue). With this task Senju et al. (2009) studied adults with Asperger syndrome who could easily solve the False Beliefs tests, but who still showed persistent naivety in social interactions and poor understanding of non-literal language (sarcasm, jokes, teasing). In the Eye-Gaze paradigm, their eye movements betrayed a lack of spontaneous mentalising.

Uta Frith then turned to feelings: many people with autism have difficulties describing and controlling their own feelings, some even doubt they have feelings. Could this reflect a lack of spontaneous mentalising, she asked. In an fMRI experiment, high functioning autism/Asperger individuals and controls reflected on their feelings to unpleasant pictures (Silani et al., 2008). Both groups showed a strong relationship between alexithymia questionnaire scores and their activation in the anterior insula. Regardless of self-reported degree of emotional awareness, the autism group showed reduced activation in self-reflection/mentalizing regions. Thus, difficulties in emotional awareness may not be related to mentalizing per se.

Uta Frith ended up by concluding that there is still work to do on her two claims – that spontaneous mentalising ability has a firm brain basis (the ‘start-up kit), and that this neural substrate allowing automatic attribution of mental states is deficient in autism, being less active than it should be. However, explicit mentalising is likely to recruit different cognitive processes. Which means you can still learn even though you may not have the start-up kit.

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In her talk at the 10th Nordic Meeting in Aalborg 2010, Francesca Happé first raised the question of whether or not the triad of traits considered to be characteristic of autism form as tight a unity as has been generally believed. The triad refers to the three core features of autism spectrum disorders: Social impairments, communication impairments, and restricted/repetitive behaviours and interests. Francesca Happé suggested that this triad may be fractionable, that is, that the three traits may occur as separate features with distinct causes.

The fractionable triad view was founded in data from a large twin study (Ronald, Happé & Plomin, 2005). These data indicate that all three traits are heritable and, secondly, that they do not always co-occur (Figure 1). Their overlap is above chance level, but phenotypic correlations are only modest (r .2 to .4). Genetic correlations vary from .18 to .50 and suggest that at least half the genetic influences on, say, social difficulties are specific to that trait and not overlapping with influences on communication impairment. In line with this, many children have only one or two of these traits. There does not seem to be any single genetic disposition that could explain all the traits. The search for genes contributing to autistic spectrum behavior may be more fruitful if directed towards genetic dispositions to the separate traits.

The study also indicated that the autistic-like traits vary continuously. They occur in varying degrees forming a normal, not a bimodal distribution. They may also occur in non-ASD individuals.

The search for a parsimonious single cognitive deficit underlying the triad has also been unsuccessful. However, there are satisfactory theories for distinct parts of the triad. For example, non-social features are well explained by a detail-focused cognitive style, that is, ‘weak central coherence’. One expression of his cognitive style may be the distress at «tiny» changes in environment and routines that occurs in many affected individuals. Restricted/repetitive behaviours may be explained by executive function problems, but also by weak coherence, i.e., detail-focused cognitive style. Other examples were also given.

However, detail focus and planning problems dissociate. For example, boys with ADHD, who have executive dysfunction and show poor planning, do not show weak central coherence. Similarly, using a drawing task, Booth et al. (2003) showed that poor planning was not correlated with detail-focus (e.g., starting the drawing of a house from a pane in the window) or coherence (e.g., fragmentation).

Concerning neural underpinnings there does not seem to be a single neural substrate for the triad of ASD traits. In functional imaging studies theory of mind and executive functions have been related to different neural networks while weak central coherence may, possibly, be related to reduced functional connectivity.
The latter part of Francesca Happé’s talk reviewed and updated the concept of central coherence. This concept was put forward by Uta Frith. In her classic book Autism: Explaining the Enigma (1989) Uta Frith stated: “In the normal cognitive system there is a built-in propensity to form coherence over as wide a range of stimuli as possible, and to generalize over as wide a range of contexts as possible». Weak central coherence is reflected as a processing style which favours segmented over holistic processing.

In 2006 Francesca Happé and Uta Frith suggested some modifications of the concept: Weak coherence is not a deficit, but a processing style or bias. It is independent of, but interacts with, social (e.g. Theory of Mind) deficits. Further, problems processing global information may be secondary to strength in local processing.

Resolving this question is complicated by the fact that many tasks confound demands, such that local processing superiority/bias and global processing inferiority cannot be untangled. For example, good performance on the Embedded Figures task, characteristic of ASD, may be due either to being good at spotting small parts, or to being bad at perceiving the cohering gestalt.

There may be means to separate the effects of local bias and global weakness, however. Francesca Happé reported new studies using tasks assessing perception of fragmented figures, discrimination of impossible figures, and phoneme segmentation (work by Rhonda Booth, 2006, in prep.), which may specifically target global integration rather than superior detail processing. Similarly, the drawing task by Booth et al. (2003) demonstrated that local superiority and global deficits can be tapped independently; a drawing may lack the global configuration in spite of no evidence of a local bias, or vice versa.

A separate question is whether top-down (concept-driven) processing influences perception of social vs. nonsocial stimuli to the same degree. In other words, is ASD characterized by reduced top-down processing in general or more specifically in social perception (e.g. faces)? There is some evidence in favor of the latter view. People with ASD have difficulties using prior knowledge, providing top-down cues, to perceive a face as a face. Thus, when faces are shown first as degraded pictures, then as complete figures, people with ASD still have problems perceiving the same face when presented in the form of a degraded picture. In contrast, perceiving objects from degraded pictures is facilitated if the person is allowed to see the complete figure first (Loth, Gomez & Happé, 2010).

Implications of the fractionable triad approach include recognizing the need to assess the traits separately and awareness that they may occur in non-ASD individuals as well. Concerning the central coherence concept, newer views maintain that both reduced global processing and superior local processing are characteristic of ASD.

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Martin Brüne is a medical doctor trained in both neurology and psychiatry. He is a professor at the University of Bochum and has worked on the topic of social cognition in schizophrenia for a number of years (see i.e. Brüne, 2005). In his talk on this topic, he started out by stressing that the term social cognition is not particularly precise and that it overlaps with other constructs, such as metacognition. Also, theory of mind (ToM), one social cognitive domain which has received much attention in the schizophrenia field during the last years, is not included in the perhaps most common definition of social cognition which was put forth by Leslie Brothers in 1990: “Social cognition is the processing of any information which culminates in the accurate perception of the dispositions and intentions of other individuals”. ToM, or mentalizing ability, was given particularly attention during Martin Brüne’s talk.

The initial part of the talk focused on the “social brain hypothesis” and evolutionary aspects of social cognition. The first scientific paper to deal with ToM did not focus on humans. Premack & Woodruff (1978) asked whether the chimpanzee has a ToM and can infer the mental state of a human being. The answer is that they can. However, although experimentally hand reared chimpanzees are able to understand the intention of others; they cannot in general pass false belief tasks or under-
stand fairness. Thus, there is something uniquely human to ToM. Brüne claimed that ToM developed through human evolution due to increasing social complexity of the environment, and that the evolved function of ToM is to predict or even manipulate the behaviour of others which will subsequently lead to social success. An example of one evolved cognitive mechanism is the detection of cheaters or free-riders. The capacity to detect a cheater depends on social cognitive abilities such as inferring what is on another person’s mind and to evaluate the truthfulness of that person’s actions.

From this Martin Brüne went on to the neural correlates of social cognition. The amygdala, the orbitofrontal cortex and the ventromedial prefrontal cortex are central brain areas for this type of cognition. He gave us arguments for the importance of studying social cognition in schizophrenia, such as mounting evidence that ToM deficits in schizophrenia are trait-like (Janssen et al., 2003) and reminded us of the clinical importance of social cognition in schizophrenia through its role as a predictor of functioning. The explained variance ranges from 20 to 50 % depending on the study. A recent meta-analysis investigating the predictive validity of neurocognition and social cognition found that both types of cognition are powerful predictors, with social cognition, especially ToM, possibly overriding neurocognition in importance (Fett et al., 2010).

Brüne presented some preliminary evidence of social or mirror neuron dysfunction in schizophrenia: one study has found atypical facial mimicry reactions in people with schizophrenia when observing emotions in others (Varcin et al., 2010).

To end his talk, Martin Brüne drew our attention to the von Economo neurons (also called spindle cell neurons) located in the anterior cingulate cortex (ACC), parts of the insula and in the dorsolateral prefrontal cortex. Their density is known to depend on human age, and they have been claimed to play a specific role in social information processing. In a recent study Brüne and colleagues (2010) examined the density of these neurons in the ACC in persons with schizophrenia and found it to be positively correlated with age of onset and negatively correlated with duration of illness. The authors speculated that this could implicate the von Economo neurons in the ACC in both neurodevelopmental as well as neurodegenerative processes in schizophrenia.

In his talk, Martin Brüne was able to convey to his audience both the importance of social cognition in schizophrenia, as well as how social cognition constitutes a primary asset of what it means to be human through his focus on evolutionary theories.

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The concept of the “executive functions” of the frontal lobe originated in neuropsychological studies of neurological populations in humans aimed at determining the roles of the frontal lobes in cognition. It is an umbrella term for a very wide range of abilities that enable us to adapt new ways of behaving. Since many, if not most, social situations are “novel” in the sense that they are not exactly the same as previously encountered, it is not surprising if deficits in executive function have effects upon social behaviour. Moreover, some aspects of frontal lobe function seem dedicated to social and emotional behaviour. However, research into frontal lobe executive functions and investigations in the field of social cognition have historically developed largely independently. But recent years have seen the importing of many of the procedures and techniques from human neuropsychology into fields relevant to understanding social cognition, and vice versa. The most obvious of these relates to the study of cognition in autism spectrum disorders (ASD) and other developmental conditions. At the head of this advance lies the huge recent step forward in our understanding of a large part of the brain: rostral prefrontal cortex (approximating BA 10).

We now know that rostral PFC supports processing relating to a wide variety of cognitive abilities which are critical to competence in everyday life. These include multi-tasking and prospective memory, high-level memory control (e.g.) source and context memory, metacognition, social cognition (including some forms of mentalizing), and dealing with “open-ended” situations. Recent evidence from Professor Burgess’ lab and others shows that these abilities can be impaired independently from “IQ” and many other mental capacities. The new methods developed at Professor Burgess’ lab in order to investigate competence in these domains, ostensibly for neurological populations, are now starting to be applied to the study of atypical development in children. As this occurs, it is becoming clear that people with ASD often show disruption of the abilities supported by rostral PFC. However, this disruption cannot be captured within a “deficit model”. Instead, it is characterised by abnormal variation which might be the hallmark of a disruption in the course of functional specialisation of this brain region. This kind of interaction between the methods and theories prevalent in the two erstwhile separate fields (neuropsychology and developmental psychology), typified by the emerging field of social neuroscience, promises to radically change practice in both fields.
Essi Viding’s research combines cognitive experimental measures, twin model-fitting, brain imaging and genotyping in order to study different developmental pathways (environmental and genetic) to persistent antisocial behaviour.

The aim of her talk was to explore whether genetic vulnerability may underlie neurocognitive ‘abnormalities’ associated with psychopathic traits. More specifically, her research focuses upon whether children with callous-unemotional traits are at risk of developing psychopathy as adults?

Antisocial behaviour has been divided into different subtypes of which callous-unemotional traits are seen as a sub-typing index, and growing evidence indicates that children with conduct problems/callous-unemotional traits show more severe behavioural problems than children who ‘only’ have conduct problems. The children with callous-unemotional traits are also more likely to have long term difficulties and may be resistant to traditional interventions (Frick & Viding, 2009).

The children with both conduct problems and callous-unemotional traits have been shown, in several studies, to have problems with recognising emotions such as fear and sadness, both vocally and visually, in others (Blair & Viding, 2008). These children are also poor at recognising others’ (and their own) distress and guilt, but appear to have good mentalising skills otherwise. From a behavioural perspective there is reasonably good evidence that callous-unemotional traits may characterize a subgroup of children with conduct problems. One of the central questions for research is the respective contribution of heritable and environmental factors in the development of antisocial behaviour.

Research into genetic influences is primarily based on the classical twin design, where greater resemblance is expected in monozygotic twins than in dizygotic twins (this then allows for shared and non-shared environmental influence to be identified). Essi Viding and her colleagues look at callous-unemotional traits in the Twins Early Development Study. They found that the relationship between callous-unemotional traits and conduct problems is...
primarily heritable both across the normal distribution and at the extremes of the distribution at 7 years of age (Viding, Frick & Plomin, 2007). Non-shared environment has also been found to play a role (Viding et al., 2005).

Further research into whether there is a difference in the origin of conduct problems between children with and without callous-unemotional traits (differentiated by the use of SDQ Conduct Problems scale) demonstrated a stronger genetic influence in the group of children with callous-unemotional traits as opposed to children with conduct problems but without callous-unemotional traits (the groups were also controlled for variance shared with hyperactivity).

The first genome-wide association study of children with conduct problems and callous-unemotional traits has generated a list of SNPs that should be followed up in subsequent studies, but none have so far reached genome-wide significance (Viding et al., 2010).

Early callous-unemotional traits are associated with later psychopathology. This was shown by Fontaine et al. (2010) who found that elevated callous-unemotional traits are related to increased levels of behavioural difficulties and family risk factors at 7 years of age as well as higher levels of conduct problems and hyperactivity at 12 years. Viding and her colleagues argue that clinical approaches aimed at reducing psychopathy in children would benefit from an assessment of callous-unemotional traits, given their reliable association with later psychopathology. Furthermore, sex differences appear relevant in understanding how patterns of stable high callous-unemotional traits emerge; meaning that trajectory membership may be influenced by different etiological factors in girls.

Overall, the existing research into genetics and callous-unemotional traits supports that antisocial behaviour in the presence of callous-unemotional traits is strongly heritable and, as stated, that early callous-unemotional traits are associated with later psychopathology.

Finally, Essi Viding gave an overview of research into neuroimaging data on conduct problems/callous-unemotional traits. It is known that patients with amygdala damage have poor conditioned emotional responses and poor fear recognition - so do adult psychopaths and children with conduct problems/callous-unemotional traits (Blair & Viding, 2008). Marsh et al. in 2008 found lower amygdala reactivity to fearful emotional faces in adolescents with callous-unemotional traits as compared with healthy comparison adults and adolescents with ADHD. Jones et al. (2009) replicated this study with seventeen boys with a mean age of 11 years with conduct problems and callous-unemotional traits and thirteen comparison boys of equal age. They found that relative to the comparison group, the boys with conduct problems and elevated levels of callous-unemotional traits manifested lesser right amygdala activity to fearful faces. The findings from these two studies are in line with data from adults with psychopathy and suggest that the neural substrates of emotional impairment associated with conduct problems/callous-unemotional traits are present already in childhood.

It has also been shown in brain imaging that structural brain data is suggestive of aberrant development of brain regions implicated in social cognition including orbitofrontal and cingulate cortices (De Brito et al., 2009), and that brain structural differences associated with conduct problems/callous-unemotional traits may represent intermediate phenotypes (Rijssijk et al., 2010).

Essi Viding pointed out that many questions in research on the “Development of the psychopathic brain” remain unanswered. Amongst them are (i) What genes are involved in vulnerability to conduct problems/callous-unemotional traits? (ii) How specific are the brain endophenotypes to conduct problems/callous-unemotional traits? (iii) Are the intermediate phenotypes stable across time? (iv) Are biomarkers (genes/brain measures) predictive of long term outcome and treatment response?

A considerable future challenge is also to gain a better understanding of environmental contingencies and development.

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THE NEURAL BASES OF COMPETITIVE EMOTIONS

Simone G. Shamay-Tsoory, Professor of social neuroscience director of the Social and Affective Neuroscience Lab at Haifa University.

In her talk at the 10th Nordic Meeting in Aalborg 2010, Simone G. Shamay-Tsoory presented recent studies exploring the neural bases of competitive emotions. Although the neural correlates of basic emotions, such as fear, anger, happiness and disgust, have been extensively documented in the literature (Phan, Wager, Taylor, & Liberzon, 2002), the neuroanatomical correlates of competitive emotions have only recently started to receive attention.

First, Shamay Tsoory presented findings from a lesion study conducted in her lab. The study explored the notion that the understanding of social competitive emotions is particularly impaired in patients with ventro-medial (VM) prefrontal lesions. Shamay-Tsoory and colleagues (2007) speculated that the ability to understand competitive emotions, such as Schadenfreude and envy, is related to broader mentalizing and perspective-taking capacities and therefore that lesions in the VM prefrontal cortex may impair the ability to understand these emotions. In order to examine this hypothesis, patients with lesions in the VM prefrontal cortex completed the ‘Yoni’ task. This computerized task is based on another task previously described by Baron-Cohen et al. (1995), which involves the ability to judge mental states on the basis of verbal and eye gaze cues (the ‘Charlie’ task). The task was modified by adding social competitive conditions for envy, Schadenfreude, and identification. The results supported the aforementioned hypothesis by showing selective impairment in recognizing Schadenfreude and envy, but not identification, in patients with VM prefrontal cortex damage (Shamay-Tsoory, Tibi-Elhanany, & Aharon-Pe- retz, 2007).

A later study by Shamay-Tsoory and colleagues (2008) examined the ability of individuals with Asperger syndrome (AS) and high functioning autism (HFA) to recog-
In order to test the ability of individuals with AS/HFA to understand Schadenfreude, envy, and identification, the aforementioned ‘Yoni’ task was used. The results showed that while individuals with AS and HFA showed no difficulty on basic ToM conditions, they were impaired in their ability to identify envy and Schadenfreude. In addition, the ability to recognize these emotions was related to their scores on a self-rating scale of perspective-taking ability and the ToM task.

Recently, a functional magnetic imaging study was conducted by Shamay-Tsoory and colleagues (Dvash et al., 2010). The study examined the emotional and neural correlates of upward social comparison (comparison with those who have more) and downward social comparison (comparison with those who have less). The task used was a computerized game in which a reward was presented either absolutely (alone) or relatively (in comparison to another). The results showed that comparisons of the activations between an actual gain and a relative gain (when the subject won more or lost less money than the other putative player) suggests that even when a person loses money, merely adding information about another person’s greater loss may increase ventral striatum activations to a point where they resemble those of an actual gain. In other words, even a loss may seem like a gain when compared with another’s greater loss. Likewise, winning money elicited activations in the ventral striatum that resembled those of an actual loss when compared to another’s greater gain. That is, even a gain may seem like a loss when compared with the other’s greater gain. These results support the role of the ventral striatum in competitive emotions and suggest that an absolute gain is processed in a similar pattern as relative gain and an absolute loss is processed similarly to relative loss. Furthermore, the results indicated an increased activation in the medial prefrontal cortex and temporal poles in relative outcome events as compared to absolute outcome events. The importance of these areas for mentalizing about other people’s minds has previously been demonstrated (Gallagher & Frith, 2003), and their involvement in relative reward points to a role for the mentalizing network in competitive emotions.

Exploring the neurochemical bases of competitive emotions, Shamay-Tsoory proposed a role for the oxytocinergic system in mediating these emotions. Contrary to the prevailing belief that the oxytocinergic system is involved solely in positive cooperative behaviors, Shamay-Tsoory presented findings from a recent study showing that oxytocin plays a key role in a wider range of social emotion-related behaviors (Shamay-Tsoory et al., 2009). In this study subjects played the game of chance described earlier (Dvash et al., 2010), following the administration of oxytocin or a placebo. While the oxytocin had no effect on feelings toward colors or on general mood, increased ratings of envy and Schadenfreude were reported. These results suggest that the oxytocinergic system is involved in modulating envy and Schadenfreude. Thus, it is proposed that the oxytocinergic system is responsible for modulating the salience of social agents in social contexts. As such, the administration of oxytocin may evoke a wide range of emotions and behaviors related to social behavior and parenting, such as trusting collaborators, attacking potential intruders, and competing with rivals.

In summarizing these results, Shamay-Tsoory proposed that the mentalizing network (through the mPFC and TP) and reward system (through the ventral striatum) process competitive emotions, and that the oxytocinergic system is involved in mediating these emotions.

REFERENCES:


The presentation emphasized the importance of music in relationship to an attribute of behaviour which would seem unique to humans, namely that of emotional crying. The approach taken were from neuroanatomical and evolutionary perspectives, discussing the development of crying as a form of communication in ancestors of Homo sapiens, noting some neuroanatomical developments in the human brain which would seem to distinguish it from that of our nearest living primate ancestors, which may explain the development of emotional crying.

The presentation then returned to music, and the relationship between psychiatric illness and creative abilities in relationship to poets and musicians. Neuroanatomical associations between the non-dominant hemisphere of the brain, psychiatric illness and creativity were outlined, linking the artistic expressions of such creativity with the early development of religion, again from an evolutionary perspective.
The human brain undergoes tremendous development in childhood and adolescence, along with the emergence and improvement of cognitive and behavioral skills. Development of better neuroimaging techniques and tools for analysing images of the brain now yield new possibilities for studying brain development. Recent neuroimaging studies have shown that the volume of the cerebral cortex increases in preschool years, before later volumetric reductions set in. Dramatic developments also occur in white matter, which consists mainly of myelinated long-distance nerve fibers.

The brain is largely unmyelinated at birth, and myelin growth is an important part of brain development that continues well into adulthood. How does development of cognitive abilities follow the maturation of specific brain structures as measured by neuroimaging techniques, in particular morphometric and diffusion tensor imaging (DTI) measures? In addition to normal development of brain and cognition, neuroimaging studies of how risk factors such as prenatal substance exposure may alter the developing brain and later cognitive skills, were also discussed.
The development of the social brain relies crucially on the early parent-infant relationship and these social pleasures have been shown to strongly influence future levels of pleasure and well-being. The talk discussed how social pleasures and in particular the early parent-infant relationship are related to other fundamental pleasures. The talk further outlined the evolving nature of the relationship, starting with basic orienting and recognition processes, and culminating in the infant’s attainment of higher socio-emotional and cognitive capacities.

Key social and affective interactions, such as communication, cooperative play and the establishment of specific attachments propel the development of the parent–infant relationship. Overall, the talk discussed how this research can help find more effective ways of alleviating the lack of pleasure, anhedonia, in mental illness both acutely and longer-term.

**SUGGESTED READING**

Frontotemporal dementia (FTD) is an umbrella term for a diverse group of neurodegenerative disorders that primarily affect the frontal and anterior temporal lobes of the brain. Most patients with FTD undergo dramatic changes in their personality and become socially inappropriate, impulsive or emotionally blunted, while some lose the ability to use and understand language.

FTD is the second most common cause of neurodegenerative dementia among young and middle-aged people, with Alzheimer’s disease being the most common among all age groups. The neuropathology of FTD is extremely complex, and there is no simple association between the clinical phenotype and the underlying pathology. Genetic factors play an important role in FTD, and a family history of dementia can be found in 30-40% of the cases.

In his presentation at the 10th Nordic Meeting in Neuropsychology, Christopher Kipps first gave an overview of the current understanding and classification of FTD. Three main clinical subtypes of FTD are proposed: The frontal or behavioral variant of FTD characterized by the typical behavioral changes associated with frontal damage and two language variants, together classified as primary progressive aphasia: Semantic dementia and progressive non-fluent aphasia.

Patients with the behavioral variant of FTD (bvFTD) typically become apathetic and less empathetic. They lose interest in other people and withdraw from social interaction. They tend to neglect responsibilities, self care and social rules, and they often develop stereotypic and ritualistic behaviors such as watching TV or eating according to certain rules. Disinhibition due to impulsivity and poor judgment is common and may be expressed in tactless or socially inappropriate behavior. Very few patients with bvFTD are aware of the changes, and most will deny or minimize complaints put forward by their relatives. Some, however, admit to difficulties in concentration or memory, or they may present with diffuse somatic complaints. The early symptoms of bvFTD are often mistaken for depression, but the lack of negative emotions and subjective complaints is important in distinguish-
ing bvFTD from depression. Also, relatives to patients with bvFTD very often describe personality changes that manifest as selfishness and insensitivity to other’s feelings, which is seldom (if ever) the case in depression.

The two language variants of FTD are very different in their clinical presentations. Semantic dementia (SD) refers to a syndrome characterized by gradual loss of semantic memory but preserved episodic memory. Loss of semantic memory manifests in a gradual thinning of the patient’s vocabulary. Speech remains fluent and grammatically intact, but with a profound lack of nouns. As the loss of words reflects an underlying loss of semantic knowledge, word comprehension is also affected, and despite intact perceptual abilities there is a general loss in object recognition and knowledge about the use of objects (associative agnosia). Patients with SD typically complain of the loss of memory for words, but many have a more general feeling of having “forgotten everything”.

Patients with progressive non-fluent aphasia (PNFA) present with a history of isolated difficulties in language production. Typically patients complain of word finding difficulties, and their speech is slow and accompanied by pauses when they try to retrieve words. Semantic as well as phonological errors are frequent, and in some cases sentence construction is abnormal. Also repetition of multisyllabic words is typically impaired. Patients seldom have difficulties understanding simple words, but formal testing shows difficulties understanding more complex language structures. Their personality, behavior and other cognitive functions are unaffected for the first many years of the disease.

Clinical presentations are seldom pure. Few patients present with a clinical picture that clearly fit into one of the three boxes, and several symptoms are shared across the different variants. Patients with the behavioral variant of FTD often also have language impairments, and most patients with semantic dementia exhibit changes in personality and behavior.

While diagnosis is usually easy in the more advanced phases of the disease when many abnormal features are present, it can be extremely difficult to diagnose FTD in the early phases. Many patients with bvFTD go through several neurologic, psychiatric and neuropsychological assessments in the first years of the disease without this leading to the correct diagnosis.

Kipps pointed out that this might be due to two problems:
1) That the current diagnostic criteria for FTD need to be revised as only few patients exhibit all the core features early in the disease. It’s also a major problem that the current criteria are purely descriptive and extremely difficult to operationalize.
2) That the traditional methods of testing for cognitive deterioration might not be appropriate for assessment of early bvFTD. Many of these patients are able to perform within normal range on the traditional tests for executive dysfunction, as their pathology is primarily localized in the medial and orbital areas of the frontal lobe. Tests sensitive to deficits in social cognition and emotion processing seem to be of much greater relevance, but few such tests are available for clinical use at present.

SUGGESTED READINGS:

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Working memory is the ability to keep information in mind for a brief period of time, typically a few seconds. In daily life, we use working memory to remember plans or instructions of what to do next, and for controlling attention. Lower working memory is associated with distractability, and deficits in working memory is a key deficit in attention deficit/hyperactivity disorder (ADHD).

Klingberg and collaborators have developed and tested a computerized method for training working memory. Several studies have shown that working memory can be improved by this method, and that this decreases the symptoms of inattention. This has now been confirmed by several, independent studies. Klingberg and colleagues have also shown that training of working memory increases brain activity in frontal and parietal regions, and is associated with changes in the density of dopamine D1-receptors in the cortex.

Training of working memory might thus be a nonpharmacological way to address a key cognitive dysfunction of ADHD and thereby significantly and sustainably reduce the inattentive symptoms of this disorder.

Future questions concern which other cognitive functions that can be trained, and how strong transfer is between functions. Training of inhibitory functions has given negative results, but one study with training of nonverbal reasoning in 4-year old children shows evidence of transfer to non-trained reasoning tasks as well as working memory.
Finding putative neural correlates of the self has been one of the holy grails for the aspiring generation of brain mappers. This move is not unproblematic. "Selves" are not trivial things to identify, and the various paradigms employed come with particular problems and possibilities that tie in with age-old discussions in Western philosophy and psychology (Zahavi & Roepstorff, in press). However, it is characteristic that most of these experiments and approaches have taken for granted and hence explored a universalistic conception of self-hood. In that respect, the search for the neural correlate of the self is similar to the many other searches for neural correlates that are currently ongoing.

This assumed universality has recently been questioned by developments in the emerging Cultural Neuroscience field, a research endeavour that attempts to explore systematic differences in brain activity between people coming from different parts of the world (Chiao, 2009). One of the foremost researchers in the field, the Chinese cognitive neuroscientist Shihui Han from Beijing University, gave a keynote lecture at the 10th Nordic Meeting in Neuropsychology.

As in much other research in the field, Shihui Han takes his starting point in Markus and Kitayama’s (1991) neo-classic suggestion that there are large scale differences between cultures in how ‘the self’ is conceived, experienced and enacted. A key distinction here is a difference between ‘independent’ and ‘interdependent’ views of the self. People with independent selves tend to view the self as an autonomous entity separating from others and to behave according to their own internal attributes and thoughts. The interdependent view of selves, on the other hand, emphasizes the interconnectedness of human beings along with contingencies between the individual’s behaviour and the thoughts and actions of others (Zhu et al., 2007).

Professor Han’s first major contribution to this field was an elegant cultural extension to one of the neo-classic studies in the neuroscience of the Self (Zhu et al., 2007). Kelley et al. (2002) had used an adjective ascription task in an fMRI experiment to suggest that self-related activities may involve a fairly distinct set of brain regions, including in particular parts of the medial prefrontal cortex (MPFC). In the study, participants decided whether particular adjectives were good descriptions of either themselves or a famous person.

Han and colleagues made a cross-cultural version of this by scanning both Chinese and Western students in Beijing, and they also included an additional condition where people had to relate the adjectives to their mother (Zhu et al., 2007). In the critical comparison between relating to the mother and relating to the self, the authors could not find significant differences in MPFC activity in the Chinese students, whereas the Western students showed significantly more MPFC activity in the ‘mother’ condition. This was interpreted in accordance with the Markus and Kitayama distinction, that Western students, with an
independent view of self, would neurally differentiate self from mother, whereas to the Chinese students the pattern of activity to self and close others in medial prefrontal cortex appeared comparable, perhaps reflecting a more interdependent self-construal.

This finding could be interpreted as an instance of cultural determinism, where the surroundings ‘create’ the persons. However, in an important follow up study, Han and colleagues examined whether cultural priming could shift the MPFC activity to close others between a “self”-related and a “distant other”-related pattern. For this study, they went to Hong Kong to study students who had been exposed to both “English” and “Chinese” impressions during their upbringing. Indeed, they found that exposing the participants to simple icons of ‘western’ culture would increase self-mother differences in MPFC, while priming with icons of Chinese culture decreased neural differentiation of mother and self in MPFC. This finding, supported also by ‘bi-cultural’ work in the US (Chiao et al., 2010), suggests that also at a neural level, cultural self construals are context dependent and may be flexibly adapted to particular settings.

Such dynamic patterns of self-construals were further pursued in two experiments where Han and colleagues studied Buddhist and Christian communities in China. Here they also found divergent neural patterns of activity in the trait adjective task (Han et al., 2008, 2010). These findings led Shihui Han to conclude in Aalborg that “Neural mechanisms of self-related processing can be different between human communities within distinct cultural environments, reflecting the neural consequences of divergent cultural experiences and values”.

On another and very important level, the (re)discovery of culture-cognition interactions may provide an important case for a general discussion of human universality and relativity in the years to come, and it is likely that ‘the brain’ is one of the key arenas for this debate to take place (Roepstorff, 2011; Smith, 2011a, 2011b).

REFERENCES

Chris Frith has been a leading force in social cognitive neuroscience since its beginning and a natural choice for the final lecture at the conference. He was asked to sum up both the field and the conference, as well as to give his personal perspective of the future.

According to Frith, social cognition may be seen as a particularly complex aspect of cognition, i.e. information processing. In this sense, all cognition may be relevant for social cognition. For example, at this conference Burgess and Kipps talked about the importance of executive functions in social behaviour. Macrae showed how
gender stereotyping depends upon early visual object recognition processes. Kringelbach told us about the importance of the reward system for social development. Shamay-Tsoory talked about how the rewards of others are incorporated into the basic reward system. This means that in some sense the so-called social brain is in fact the entire (human) brain, and future research should focus on how the brain processes specific kinds of social cognition. We need to specify in what way different kinds of social cognition are associated with different brain systems. It is also necessary to make a distinction between implicit and explicit processes (as done at this conference by Macrae, U. Frith, Siebner, and Brüne).

Social cognition is not only a human phenomenon. It has been observed even in very primitive species, such as sticklebacks, where it helps the individual animal to learn from others and to function socially within the group (Galef & Laland, 2005). The question remains, however, whether there are aspects of social cognition that are unique to humans. Only in such a case would these depend on new regions of the brain and novel forms of computation, as was pointed out by Jack in the ‘interacting minds’ symposium.

The research regarding the neural basis of social cognition has concentrated on two major processing systems in the brain: The mirror system and the mentalising system.

**The Mirror System**

The mirror system is related to the ventral pre-motor cortex and the rostral part the inferior parietal lobule (Figure 1; Rizzolatti & Fabbri-Destro, 2008). These regions are activated largely without consciousness when we observe another person acting, as well as when we act ourselves. This can be described as social contagion. The overall function of this system is to favour the aims of the group over those of the individual.

It has been shown that we have a tendency to spontaneously mimic other peoples’ facial expressions, which can be measured as distinct facial electromyographic reactions in the emotion relevant facial muscles. However, we also show the same response to facial stimuli of which we are not conscious (Dimberg, Thunberg, & Elmehed, 2000). Imitating facial expressions such as fear and disgust is a way of preparing ourselves for danger: fear enhances vigilance (Susskind et al., 2008). At this conference, Viding told us how expression of fear also can elicit prosocial behaviour in others. Touch sensations have also been shown to be contagious and to occur unconsciously (Blakemore et al., 2005). However, the intensity of the contagion depends on whether it is about an in-group to which you can relate, or an out-group to which you do not feel any connection. This means that the behaviour of someone from another culture than your own will not be contagious (as shown by Han and his colleagues, Xu et al., 2009).

A recent study has indicated that reward areas of the brain are also activated when others agree with us (Campbell-Meiklejohn et al., 2010). Similar effects were demonstrated at this conference in Shamay-Tsoory’s research on competitive emotions. These mechanisms of emotional and motor contagion help us align with the group and prime group goals rather than selfish interests (van Baaren et al., 2004). However, even though mimicking other people enhances prosocial behaviour, it may weaken abilities dependent on theory of mind, e.g. detect deception in other people (Stel, van Dijk, & Olivier, 2009). Contagion helps us behaving in a group oriented...
manner, but it interferes with recognizing behaviour and intentions of others when these differ from the common group behaviour.

THE MENTALISING SYSTEM
The mentalising system is related to the medial prefrontal cortex, the amygdaloid region, the superior temporal sulcus, the temporo-parietal junction and the basal temporal cortex (Figure 2; Blakemore, 2008). It involves perceiving the mental states of self and others and allows for optimisation of collaborative interactions. Some aspects of the mentalising system also function unconsciously. For example we have an automatic tendency to track the knowledge of other people (Samson et al., 2010). Even 13 months old babies are able to take account of false beliefs (Surian, Caldi, & Sperber, 2007) as related by Uta Frith at this conference. At age four this ability has developed further so that the child is aware of differences in knowledge and how this determines behaviour.

How does the mentalising system work? One suggestion is that we guess the goals of other people and then predict their actions by running a simulation in our own motor system (Kilner, Friston, & Frith, 2007). When people do not act as expected (a prediction error), we must modify our assumptions. Examples of this mechanism of top-down control based on prediction errors were given at this conference by Macrae and Happé. However, social interaction is a complicated process because while you are making predictions about other people, so are they about you! This leads to a recursive process whereby I have to think about what you think about me. For example Shihui Han showed how the concept of self is also influenced by what other people think of us. When intention prediction errors occur, higher activity may be seen in the right STS (Pelphrey, Morris, & McCarthy, 2004).

In contrast, when people need to mentalize in order to solve a task, studies reveal higher activation in mPFC and DLPFC (Hampton, Bossaerts, & O’Doherty, 2008). Computations regarding influences of both your own and other people’s actions on performing a task can be applied to the non-social domain as well. This point was made by Burgess and Kipps at this conference. Thus, this may not be an example of a uniquely social form of cognition. One possibly uniquely human form of social cognition is revealed by our recent study of interacting minds. Optimal performance in this collaborative task requires metacognition (the ability to introspect on one’s performance) and full communication between the partners (Bahrami et al., 2010).

CONCLUDING REMARKS
While the contagious mirror system primes group-oriented goals and optimises interaction, the mentalising system keeps track of the knowledge and goals of others when these are different from our own. Thus, these two systems may be fundamentally in conflict and mutually inhibitory. Human social interactions typically involve recursive representations of the mental states of self and others, but it may be that consciousness only breaks into social interactions when we need to explain to others the reason why we act as we do (based on knowledge and intentions). This process optimises social interaction and is uniquely human. It also creates culture and cultural differences.
REFERENCES


POSTER SESSIONS

Bettina Hornbøl

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INTRODUCTION:

Early-onset schizophrenia (EOS) is an illness with greater clinical severity and poorer outcome than the adult-onset form of the disorder, and it occurs in approximately 4% of all schizophrenia cases (Cannon et al., 1999). The clinical presentation of EOS is characterized by insidious onset and predominant negative symptoms (Hollis, 2000). These features, together with premorbid impairment which also characterizes this group (McClellan, Breiger, McCurry, & Hlastala, 2003), are the best predictors of a continuing diagnosis of schizophrenia, a more severe clinical course (Werry, McClellan, & Chard, 1991; Eggers & Bunk, 1997) and poor long-term outcome (Alaghband-Rad et al., 1995; Tolbert, 1996). In addition, the EOS-group is characterized by premorbid neurodevelopmental abnormalities (Nicolson & Rapoport, 1999), developmental deviation (Hollis, 1995; Vourdas, Pipe, Corrigall, & Frangou, 2003) and seem to have poorer treatment effects of antipsychotic medication (Hollis, 2000).

From research on adult-onset schizophrenia (AOS), we do know that cognitive impairment is a core feature of schizophrenia. Cognitive deficits are predictors of both poor functional outcome and prognosis (Green, Kern, Braff, & Mintz, 2000; Leung, Bowie, & Harvey, 2008). Cognitive deficits are also prevalent in the EOS group (Oie & Rund, 1999; Ueland, Oie, Inge Landro, & Rund, 2004; Fagerlund, Pagsberg, & Hemmingsen, 2006), but is examined to a much lesser extent.

Due to the lack of studies in this area, it has been hard to establish a typical, distinct neuropsychological profile in the EOS group. A major problem in establishing a profile of cognitive deficits in EOS is the use of different NP test batteries across different studies making direct comparisons difficult.

The lack of a consensus core battery to evaluate cognitive functioning has hampered both possibilities to compare findings across the now numerous studies on cognition in AOS and the development of treatments. This is one of the main reasons behind the National Institute of Mental Health “Measurement and Treatment Research to Improve Cognition in Schizophrenia” (MATRICS) initiative, which aimed at developing a consensus cognitive battery for use in clinical trials in schizophrenia (Kern et al., 2008).

The MATRICS group has identified 7 cognitive do-
mains that are dysfunctional in schizophrenia and thought to be a core feature of the disorder. The MA-TRICS test battery covers these domains and seeks to be a gold standard for measuring cognition in schizophrenia.

We wanted to examine the neuropsychological profile in the EOS group, using the MATRICS battery.

METHODS:
We examined 31 EOS patients and 67 healthy controls. Exclusion criteria were known brain injury or neurological diseases, or an intelligence quotient (IQ) below 70.

The patients were interviewed by clinical psychologists, and diagnoses were established using the Structural Clinical Instrument of Diagnosis for DSM-IV Axis I disorders (SCID-I), modules A–D. Psychopathology was assessed using the Positive and Negative Syndrome Scale (PANSS) and the Global Assessment of Functioning Scale.

Neurocognitive assessment was done using the MATRICS battery with the addition of IQ tests. The cognitive domains and their neuropsychological tests are as follows:

**Intellectual function**
Wechsler adult intelligence scale (WASI)

**Speed of processing**
Trail making Test A

**Category fluency**

**Brief Assessment of Cognition in Schizophrenia**
(BACS): Symbol-Coding

**Attention / vigilance**
Continuous Performance Test—Identical Pairs (CPT-IP)

**Working memory**
Wechsler Memory Scale (WMS-III): Spatial Span
Letter-Number Span

**Verbal learning**
Hopkins Verbal Learning Test—Revised (HVLT-R)

**Visual learning**
Brief Visuospatial Memory Test—Revised (BVMT-R)

**Reasoning and problem solving**
Neuropsychological Assessment Battery: Mazes

**Social Cognition**
Mayer-Salovey-Caruso emotional intelligence test (MSCEIT)

RESULTS:
We found that adolescents with schizophrenia were significantly more impaired than healthy control subjects on every measure, except social cognition. The differences are largest on the domains verbal learning, visual learning, and working memory. As a group, the patients performed between 0.8 and 1.8 SD below the control group.

The z score profile on the MATRICS battery is presented above, and shows that there was a clear difference in performance pattern across groups.

DISCUSSION:
We found a general and explicit cognitive deficit in the patient group. Our findings of impairments in all 4 higher order neurocognitive domains (executive function, working memory, visual learning, and verbal learning) support other studies implicating both frontal and temporal hippocampal involvement in the pathophysiology in schizophrenia.

There were no differences between the groups in social cognition, as measured by the MSCEIT. This could possibly be explained by test characteristics, as the MSCEIT was made for the adult population. The situations described in the test vignettes are far from adolescent every day life.

REFERENCE LIST


PhD candidate (dobbelkompetanse-prosjekt) and clinical psychologist Mari Strand is part of the Mood and Cognitive Function Group (MCF) at the University in Bergen, lead by associate professor Åsa Hammar. Hammar has been working with research on Major Depressive Disorder and cognitive functions for more than a decade. Her doctoral thesis (Hammar, 2002) had a focus on cognitive function in depression, where she followed patients from acute phase and into symptom recovery. The findings concluded that certain cognitive functions remained impaired six months following acute phase of depression, despite symptom reduction. Questions related to the understanding of how these functions develop over time, has initiated several doctoral projects, including the present project.

The present project has included 30 patients suffering from recurrent Major Depressive Disorder and 30 matched healthy subjects. All participants have been examined with standardised tests and experimental paradigms at three test occasions (acute phase, 9 month and 24 month follow up) The project has generated a considerable amount of data and several papers have been published (Hammar et al. 2010; Hammar et al. accepted; Schmid et al submitted). Mari Strand has a special field of interest, and focuses on MDD and processing of emotional information during symptom recovery in her PhD project.

There are several ways to study emotional information processing, both verbally and pictorially. One way to study this processing has been to incorporate facial images in experimental designs. Studies using such designs have found that patients have responded significantly different towards negative emotional information (sadness) than healthy controls, and in addition report less bias towards positive emotional information (Bouhuys, 1999; Gotlib, et al., 2004; Hale, 1998; Joorman & Gotlib, 2006). Some studies report that this bias towards negative visual information remains in symptom reduction (Bouhuys, 1999; Joorman & Gotlib, 2006; Suslow, 2004). Karparova et al. found, however, that patients reported no particular bias towards negative information (Karparova et al., 2005). Emotional information processing during recovery is thus still not fully understood and needs further investigation.

A visual search paradigm was developed as a way to investigate cognitive functions (Hammar, 2002), and this paradigm was further developed by including emotional stimuli. The visual search paradigm including emotional information is based on Anne Treisman paradigms of visual search (Treisman, 1988). The stimulus material included in the test was taken from the FEEST collection of stimulus material, which is based on Ekman and Friesens facial images of basic emotions (Young, et al., 2002).

Results from the visual search test indicated that even though patients and controls were similar in their way of responding to emotional information in a visual search paradigm, the depressed patients had longer RTs the more depressive symptoms they reported, and there was a significant relationship between symptom load and RT, when there were positive and negative emotions involved simultaneously, however, not when neutral information was involved.

Secondly, we have carried out a study, using a translated version of an emotional Stroop design developed by Dr
Mike Oram at the University of St. Andrews, Scotland. The emotional Stroop task has traditionally been a copy of the original Stroop task often used in neuropsychological testing, however, with the exception that the words have had an emotional valence. A rather constant finding has been that acutely depressed patients have been more sensitive to words with negative content, and that this has increased response time compared to healthy controls. The emotional Stroop task used in this project does not only have words included as stimuli, it also incorporates facial images, developed at the Perception Lab, at the University of St. Andrews. Colour is thus not a feature in this test. The task was to identify if a word or face shown simultaneously on the screen is positive or negative. We have hypothesised that we would see an effect of facial emotions on word processing and that emotional and verbal congruency will yield shorter response time than when being incongruent, and that inhibiting incongruent emotions will increase response time. Furthermore, we expect that there will be a significant difference between patient and control groups regarding inhibition of negative emotions. A pilot study was conducted during the spring of 2010, on a small group of students. We have later this year conducted the main study on a group of 20 patients with MDD in recovery and 20 matched controls, and we are now analyzing the data in cooperation with Dr. Oram at the University of St. Andrews. This collaboration will continue during 2011, and the plan is to present the results from these studies during the coming year.

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The present PhD project is one in four PhD projects in the Bergen Mood and Cognitive Function Group (MCF) lead by associate professor Åsa Hammar, Institute of Biological and Medical Psychology (IBMP), University of Bergen, Norway. Together these four longitudinal projects cover different phases of depressive episodes, from first episode of depression to an 8-10 year follow up of patients experiencing recurrent episodes. The four sub studies are designed to answer questions and gain further knowledge about MDD individually. In addition, they all have a strong clinical focus and foundation in the same empirical tradition, making comparisons across studies possible. Cognitive impairment in the acute phase of recurrent Major Depressive Disorder (MDD) has frequently been documented in the literature in the past decade (see reviews Austin, Mitchell & Goodwin, 2001; Hammar & Årdal, 2009; Rogers et al. 2004). Further, there has been a growing focus on how the cognitive impairment develops over time, and on how it relates to reduction of depressive symptoms (Hammar & Årdal, 2009). Important factors to consider when investigating cognitive functioning in MDD patients is that findings suggest that cognitive impairment worsens for every episode of depression (Brown, Rush & McEwen, 1999; Sweeney, Kmiec & Kupfer, 2000) and that the impairment observed in the non symptomatic phase is related to number of previous episodes of depression (Kessing, 1998). In addition, previous findings suggest that subtypes of depression show different patterns of cognitive profiles (Elliot, 2002). However, longitudinal studies are few and divergent with respect to inclusion criteria and cognitive domain of interest.

The aim of the ongoing PhD project is to assess the role of diagnostic subtype on cognitive functioning in patients with MDD, both in the acute phase of illness, and in relation to symptom reduction. The main focus in the present project is the inclusion of a subgroup of patients that experience a major depressive episode for the first time in their lives. This subgroup of patients is seldom investigated in the literature, and following this group of patients over time may provide valuable knowledge about factors that contributes to relapse and recurrence often seen in this patient group. The project is now in the process of including an individually matched control group to the subgroup of patients that experience their first episode of MDD.

Focusing on the cognitive function of inhibition, results from a longitudinal study of 24 recurrent MDD patients, and preliminary results regarding the ability to inhibit in a group of 24 patients that experience their first episode of MDD, were presented at the 10th Nordic Meeting in Neuropsychology, 15-18 august 2010, in Aalborg, Denmark. Impairment in inhibition in recurrent MDD has been reported in longitudinal studies of various time spans, both in the acute phase and in phases of symptom reduction and recovery (Biringer et al., 2005; Hammar et al., 2009; Trichard et al., 1995; Årdal & Hammar, 2010). These studies may indicate that impaired inhibition is a prolonged or a stable cognitive impairment, contributing to the high relapse risk seen in this patient group. However, to our knowledge, the cognitive function of inhibition has not previously been investigated in patients experiencing a first episode of MDD.

Inhibition can be understood as a cognitive process which...
controls that unwanted internal or external stimuli do not compete for resources in a limited capacity system (Go
tier et al., 2009; Joormann, Yoon & Zetsche, 2007). De
pressed patients are characterized by rumination of nega
tive thoughts and negative self-evaluation. Joorman and col
leagues (2007) postulate in a review on cognitive inhi
bition in depression that the inability to inhibit irrelevant
stimuli may represent an underlying cognitive impairment
which contributes to sustained negative thoughts and ru
mination. An inability to inhibit negative thoughts may
represent an important vulnerability factor that can cause
enhanced risk of relapses and recurrences.
To measure Inhibition, the Color-Word Interference Test
from The Delis Kaplan Executive Function System (D-
KEFS) (Delis et al., 2001) was administrated. The Color-
Word Interference Test is a version of a Stroop like para
digm typically used to investigate inhibitory capacity. It
contains four conditions. In the first condition the subject is
to name the ink colors of color patches. In the second
condition, the subject is to read color words written in
black. This condition measures the automatic response of
reading. In the third condition, the subject is asked to
inhibit reading the colour words but to name the incongru
tent ink colour the words are written in, and thereby
inhibit the automatic response of reading. In the fourth
condition, the subject is asked to inhibit reading the co
lour words but to name the incongruent ink colour the
words are written in, except reading the colour words
when the words are presented within a frame. In the fo
urth condition, the ability to inhibit an automatic respon
se of reading and the ability to shift mental set (mental
flexibility) are measured.
The results show that in a group of recurrent MDD pa
teins the ability to inhibit was impaired compared to the
control group in the acute phase of illness (Hammar et
al., 2010). Interestingly, the results showed that this im
pairment was still present in the 9 month follow-up as
sessment, despite significant symptom reduction. Further,
the results showed that the patients showed an individu
ally stable level of performance on this task, both in the
acute phase of illness and after symptom reduction, indi
cating that an inability to inhibit may represent a stable
individual deficit across the time period from initial tes
ting to the follow-up assessment for the patient group
(Schmid et al., in revision) This pattern could be interpr
ted to represent a vulnerability trait for recurrent depres
sion. However, in order to fully understand this relation
ship, more studies are needed which longitudinally in
gestigate the cognitive function of inhibition in sub
groups of MDD patients.
The preliminary results regarding inhibition in the sub
group of first episode MDD patients showed that this pa
tient group was not impaired compared to the recurrent
group and the control group in the acute phase of illness.
However, this subgroup of MDD patients was not com
pared to an individually matched control group. For the
presentation, this subgroup was compared to the recurrent
group and this group’s corresponding control group, who
differ significantly from the first episode group on impor
tant confounding variables such as IQ and age. To gain
valuable information concerning cognitive functioning in
the first episode group, an individually matched control
group must be recruited. The present PhD-project will
continue the inclusion of a control group, and follow this
subgroup of patients in a one year follow-up assessment.
In addition, an expanded follow up assessment of the pa
tient and the control group is planned in the future.

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Subjects with major depressive disorders, bipolar disorder, schizophrenia and borderline personality disorder often have difficulties in cognitive-and emotional regulation, and research from Norway on these topics will be presented at the Symposium. Impairment in Executive Functioning (EF) in the acute phase of recurrent Major Depressive Disorder (MDD) is well documented. However, only few studies have investigated EF in MDD during the first depressive episode, and findings are inconclusive. Further, previous studies suggest that cognitive impairment worsens for every episode of depression. The aim of an ongoing study is to assess the role of diagnostic subtype on performance in EF in patients with first episode MDD (FE) and patients with recurrent MDD (RC). Marit Schmid presented preliminary results from the study “Executive functioning in patients with First episode Major Depression and in patients with Recurrent Major Depression”. Understanding how depression affects basic processing of emotional information.
is important in the understanding of acute depression and how it affects patients socially. Furthermore, it is important to obtain a better understanding of the recovery from depressive symptoms, and how patients’ basic processing of social cues in the surroundings changes in this phase.

Mari Strand presented data from the study “Emotional information processing in Major Depressive Disorder” and a summary of this presentation is included.

Carmen Simonsen discussed how neurocognition and psychosocial function varies across and within bipolar disorder and schizophrenia. Early-onset schizophrenia is rare, and the course of illness is characterised by an insidious onset, poorer cognitive function and poorer outcome compared with the adult-onset group. They are often less motivated and become easily tired during assessment. Thus, it is of importance to have a well-designed neuropsychological battery, and to select the right measures when assessing these patients. Aina Holmén focused on the characteristics of cognitive deficits in adolescents with schizophrenia. A summary of her presentation is included. Executive functioning has been identified to constitute a selective deficit in a neuropsychological profile analysis in patients with BPD. Vegard Øksendal Haaland focused on possible associations between emotional dysregulation and neuropsychological performance in patients with BPD. More specifically, associations between emotional dysregulation and working memory, executive functioning, social cognition, and autobiographical memory was discussed.

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1. Strand, Mari; PhD fellow, Psychologist, Marit Therese Schmid, PhD fellow, Bergen: Longitudinal studies of major depressive disorder (MDD) – cognitive functioning and emotional information processing.
2. Holmén, Aina; PhD fellow, psychologist, Oslo: Adolescents with schizophrenia-spectrum disorders: neuropsychological profile.
3. Haaland, Vegard Øksendal; PhD, neuropsychologist, Oslo: Possible associations between emotional dysregulation and neuropsychological functioning in borderline personality disorder.
4. Simonsen, Carmen; PhD fellow, psychologist, Marit Therese Schmid PhD fellow: Neurocognition and psychosocial function in bipolar disorder.
Frontotemporal dementia (FTD) is an umbrella term for a diverse group of neurodegenerative disorders that primarily affect the frontal and anterior temporal lobes of the brain. Most patients with FTD undergo dramatic changes in their personality and become socially inappropriate, impulsive or emotionally blunted, while others lose the ability to use and understand language. The symposium gave an introduction to different aspects of FTD.

AUTHORS:
1. Stokholm, Jette; Neuropsychologist, Copenhagen: From Pick’s disease to frontotemporal dementia.
2. Kipps, Christopher; MD,PhD: Clinical presentations of frontotemporal dementia.
3. Johannsen, Peter; MD, PhD, Copenhagen: Patoanatomical and genetic aspects of frontotemporal dementia.
4. Gade, Anders; Senior lecturer, Copenhagen: FTD-3: A Danish variant of frontotemporal dementia.
Multiple lines of evidence from animal and human research relate the neurotransmitter serotonin to aspects of emotions, personality and social behaviour, and this neurotransmitter system is targeted in drug treatment of mood and anxiety disorders. A major research program in Copenhagen (Cimbi) focuses on the neural bases - serotonin in particular - of personality dimensions that predispose individuals to affective and substance use disorders. This symposium dealt with highlights from both this research program and from a large SSRI treatment study, and preliminary analyses of social cognitive measures from both of these research programs were included as well. Projections from the raphe nuclei in the brainstem (figure 1) reach most of the brain, where serotonin acts to modulate neural processing. It is an exceedingly complex system, as there are more than a dozen subtypes of serotonin receptors, which seem to have separate functions. Early indicators of its role in social functions include studies in the vervet monkey (Raleigh et al., 1991, 1996). Treatment with enhancement or conversely reduction of serotonergic activity determined social status (when it was uncertain with the dominant male removed), and in this situation eventual acquisition of dominance was preceded by an early increase in prosocial behaviour. Observational studies indicated that prosocial behaviours are related to high 5-HT2A receptor density in orbitofrontal and medial prefrontal cortex, and in amygdala. These and related results from humans (e.g. Knutson et al., 1998) form part of the basis for current research and hypotheses (Gade).

The Cimbi studies have used many of the same measures across various clinical groups and their matched controls, including structural MRI-imaging, PET-studies of the serotonin transporter and a receptor (e.g., the 5-HT2A or 5-HT4 receptor), neuropsychological tests, personality inventories, and social cognitive measures. The ligand used for measurement of the 5-HT2A receptor is [18F]altanserin. An image based on this ligand is shown in figure 2, which shows that the receptor is found in widespread areas of cortex. We have not seen gender differences in 5-HT2A-binding, but there is a strong age effect with decreased binding with increasing age.

In amnestic mild cognitive impairment (MCI), we found a significant global reduction of 20-30% in 5-HT2A binding in most neocortical areas. This early reduction in 5-HT2A binding may contribute to the neuropsychiatric symptoms, in particular depression and anxiety, in early Alzheimer’s disease (Hasselbalch et al., 2008).

Putative measures of personality related to serotonin are moderately genetically determined, and it was thus of interest to study the heritability of measures of serotonergic activity, including the 5-HT2A receptor. In 24 subjects, six monozygotic and six dizygotic twin pairs, we found intraclass correlations of .87 in monozygotic and of .67 dizygotic twin pairs in regional binding potential values ( lumped over regions of interest). This is shown in figure 3, which also includes test-retest correlations for comparison.

In vivo cerebral 5-HT2A receptor binding was related to the outcome of personality assessment in a large group of normal subjects. We found a positive relation between 5-HT2A binding in fronto-limbic areas of the brain and the NEO-PI-R personality dimension of neuroticism (Frøkjær et al., 2008). This relation was strongest for the facets of vulnerability and anxiety. We have confirmed this relation in an independent sample of normal subjects with or without a high risk for depressive disorder (twins with a co-twin with or without depression). This study also indicated that familial risk and neuroticism interact in their relation to fronto-limbic 5-HT2A receptor binding (Frøkjær et al., 2010). (Moos Knudsen).
The SSri trial concerned healthy first-degree relatives of patients with depression. It was conducted as a triple-blinded controlled trial with a total of 80 participants randomised to either escitalopram 10mg/d or placebo for four weeks, and the study protocol was published prior to the conclusion of data acquisition (Knorr et al., 2009). The main outcome was the cortisol response in a combined dexamethasone corticotropine releasing hormone test. Neuropsychological and social cognitive tests as well as personality measures were also applied before and after treatment. This study is unique in being both the largest study to date of normal subjects treated for an extended period with SSri and in concerning normal subjects at risk of developing depressive disorders. The main outcome – HPA-axis results – was nonsignificant: no difference in plasma cortisol between placebo- and SSri-treated subjects. (There was, however, a negative correlation between escitalopram concentration in plasma and change in plasma cortisol). There was likewise no effect on neuropsychological measures, meaning that one month of treatment with escitalopram seems to be entirely without cognitive side effects, but also may not improve cognition. The only effect in personality measures found in analyses conducted so far was a greater change towards more agreeableness after escitalopram than after placebo (Knorr).

The social cognitive measures used in both Cimbi- and SSRI-studies studies include emotional face recognition, moral reasoning, understanding of social situations, and a test of emotional intelligence (MSCEIT, which is also the topic of symposium 9B). The analyses of both datasets are in a very preliminary stage. In the Cimbi-studies, 109 subjects were tested so far with both social cognitive measures and neuropsychological tests of cognition. Correlations are modest, indicating that social cognition is indeed a domain relatively independent of executive functions and other cognitive functions. We have related 5-HT2A-binding in specifically amygdala and medial frontal cortex (based on a priori hypotheses) to social cognitive functions in 30 normal subjects aged 20-40. Preliminary results include significant correlations to recognition of disgust (negative) and to moral behaviour (positive) as indicated in the Mendez et al. (2005) Moral Behavior Inventory. The latter result is substantial and highly significant for medial frontal cortex (Zornhagen).

We also study serotonergic contributions to social cognitive processes - specifically emotional face recognition and risk taking in decision making - in fMRI-experiments with pharmacological interventions that induce acute changes in the serotonergic system. Subjects in these experiments were scanned with fMRI in different ‘brain states’, i.e. with normal or pharmacologically modified serotonergic tone. The latter was obtained by an acute selective blockade of 5-HT2A receptors by the administration of ketanserin.

When people make risky decisions, they tend to be more sensitive to potential losses than gains of similar magnitude. Dopaminergic transmission in a ‘reward circuit’, including the ventral tegmental area, ventral striatum and medial prefrontal cortex, is generally regarded as critical for risky decision making. However, dopaminergic signaling may be influenced by serotonergic neurotransmission. We studied the modulatory effect of serotonin in 20 normal subjects by examining the effects of acute 5-HT2A blockade on regional activity and connectivity during a gambling task. Ketanserin induced changes in neural processing of negative outcomes, and the subjects became more loss avoidant. This was correlated with activity in the medial prefrontal cortex and a decreased coupling between ventral striatal areas and medial prefrontal cortex. In a similar study, we studied the response in fronto-limbic circuits to emotional (fearful and angry) faces compared to neutral facial expressions in a gender discrimination paradigm. This is a well-known procedure which reliably activates e.g. amygdala and orbitofrontal cortex. We hypothesized that blockade of 5-HT2A receptors would reduce the orbitofrontal response, since this area has a high receptor density and is known to be involved in the...
evaluation of socially relevant stimuli. This was indeed the case, whereas the amygdala activation was not reduced. However, we also found an interaction of the 5-HT2A binding potential (established in a separate PET-experiment) and the ketanserin occupancy with a positive correlation with amygdala activation and functional connectivity. Thus, these results point to a role of serotonin, particularly in the medial orbitofrontal cortex, in emotional processing of faces with negative valence (Siebner).

Iowa Gambling Task is a measure of decision making under ambiguity. This test first became known because it proved to be sensitive to ventromedial prefrontal lesions and formed part of the basis of the ‘somatic marker’ hypothesis of decision making (Gade, 1997). It has since been shown that it is sensitive to impulsivity in many other clinical conditions with symptoms indicative of poor decision making in personal and social situations. Such decisions may be made, it has been argued (see also the fMRI study above, and Øfsti, 2010), in a balance between an ‘impulsive’ reward-based dopaminergic system and a ‘reflective’ serotonergic system. We addressed this question in the final presentation of the symposium. The 80 participants in the SSRI trial (see above) also performed the Iowa Gambling Task before and after either escitalopram or placebo. Both groups performed well before and after the intervention, and there was no difference between change means of the two groups in advantageous and disadvantageous choices. (Øfsti).

In conclusion, serotonin is indeed involved in emotional processing and social behaviors, and this involvement seems to a large extent to depend on the 5-HT2A receptor in fronto-limbic areas, i.e. ‘the social brain’. Some of the effects of serotonin on emotions and social behaviour may seem subtle, consistent with a modulatory rather than a direct role for the behaviour in question. No general theory of serotonin function is agreed upon, and serotonin plays a role in many other functions besides those of the social brain. Yet for an understanding of its role in depression, anxiety and impulsivity disorders, further elucidation of serotonergic function in the social brain seems to be the way forward.

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2. Gitte Moos Knudsen, Copenhagen: Neuroimaging studies of the serotonergic transmitter system in the normal human brain and in neuropsychiatric disorders
3. Ulla Knorr, Copenhagen: SSRI-treatment of normal subjects at risk of depressive disorder: Clinical experiences and effects on HPA-reactivity, cognition and personality measures
4. Gry Zornhagen, Copenhagen: Social cognition in adults – measures and preliminary serotonergic correlations
5. Hartwig Siebner, Copenhagen: Mapping the contribution of 5-HT2A neurotransmission to risk avoidance and emotional processing (with Bettina Hornbøll and Julian Macoveanu).
6. Linn Øfsti, Oslo & Copenhagen. Decision making and the Iowa Gambling Task – are there effects of serotonergic downregulation (MDMA-abuse) or SSRI-treatment?

SYMPOSIUM 4

NEUROPSYCHOLOGICAL ASPECTS OF PAIN

Laura Petrini, organizer of the symposium

Traditionally, pain has been considered a pattern of convergent activity within part of the somatosensory cortex that deals with the exteroceptive sense of touch (Craig 2003). Pain is mostly caused by a direct activation of the nociceptive system, which is relayed through this part of the somatosensory cortex. However, this view is contradicted by the fact that pain can also occur without any excitation of nociceptors (i.e.: such as phantom pain) and the excitation of nociceptors does not always result in the sensation of pain (i.e.: emphatic responses to pain). Converging evidence shows that human pain experience is a multidimensional experience manifesting sensory-discriminative, cognitive-evaluative, and affective-motivational components, resulting in the pain system being one of the most complex human perception systems. As Craig (2003) recently underlined, pain is an enigmatic feeling that can be distinct from all other senses since it is multifaceted. Pain perception is highly subjective and its presence is modulated by a series of psychological factors (attention, distraction, anxiety, catastrophizing, coping etc.). Pain must be understood not only as physiological phenomenon but also as psychological phenomenon. Specifically, pain represents a perceptual process associated with conscious awareness, selective abstraction, ascribed meaning, appraisal, and learning (Melzack and Casey, 1968). Emotional and motivational states are central to understand its nature (Price 2000).

The International Association for the Study of Pain (IASP) provides the following definition: “Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage”. However, no defi-
nition of pain has ever encompassed all its manifestations or been scientifically complete (Tiengo 2003).

The present symposium addresses how both the interoceptive (the sense of physiological condition of the body) and the exteroceptive (cutaneous nocireception and proprioception) components of pain are combined together in the unitary perception of pain. The research and the experimental studies presented in this symposium are all conducted at the Center for Sensory-Motor Interaction, Aalborg University. These studies focus on three phenomena: (a) graphesthesia; (b) mirror and (c) thermal grill illusions.

THE PHENOMENON OF GRAPHESTHESIA IN THE NOCICEPTIVE SYSTEM

The phenomenon of graphesthesia in the nociceptive system was investigated in order to understand the extent of the exteroceptive properties of the pain system. In fact, the pain system is often thought of as being crude and inaccurate compared to the tactile system. It has even been argued that pain is based on internal perceptions like our mood and to less extent on activation of sensory pathways. There are however several reports failing to show differences in the accuracy of painful and tactile sensory tasks, such as localizing a single stimulation point on the skin. A more complex sensory ability is the recognition of numbers being “written” on the skin; an ability known as graphesthesia. In this study, we tested graphesthesia in the pain system by heating the skin with a moving infrared laser beam and compared it to graphesthesia in the tactile system. Numbers were “written” on the forearm and the abdomen of healthy young volunteers. The aim of the study was to compare the exteroceptive capabilities of the tactile and nociceptive systems. We found that subjects performed the painful graphesthesia task better than chance, but not as well as they performed the tactile graphesthesia task. The tactile graphesthesia tasks were performed with 89% (82–97%) correct responses on the forearm and 86% (79–94%) correct responses on the abdomen. Tactile graphesthesia tasks were significantly better than painful heat graphesthesia tasks that were performed with 31% (23–40%) and 44% (37–51%) correct responses on the forearm and abdomen, respectively. This could in part be explained by our finding of wider two-point discrimination in the pain system. It does however indicate that the painful information changing over time and place when writing a number remain intact from the periphery to the centers in the brain recognizing the numbers even though the painful and tactile information ascends through different pathways (Dahl-Mørch et al. 2010).

PSYCHOLOGICAL FACTORS INVOLVED IN THE MIRROR BOX ILLUSION

Mirror box therapy is a relatively new treatment, which has been successfully used in both upper and lower limb patients to help reduce pain (e.g. chronic regional pain syndrome, and phantom limb pain), and visual hemineglect. In the “mirror box” paradigm, the affected limb is placed on one side of the mirror, out of view, and the healthy limb is placed so that the subject can see its reflection (Ramachandran et al. 1995; 2009; Flor 2008). By observing the reflection of the healthy limb moving in the mirror, a visual illusion is created and the patient experiences having two healthy limbs. The illusion can be so strong, that the subjects have reported being able to “see through” the mirror’s surface, in which case the subjects feels as if the mirrored image is part of his/her own body. In this respect, the strength of the illusion may be connected with the sense of ownership to the mirrored limb (Tsakiris et al., 2006). Mechanisms involved in the alleviation of symptoms caused by the mirror illusion are not well understood, and very limited explanation can be given when trying to understand individual differences in treatment outcome and in how authentic the illusion feels, despite patients having similar injuries. This suggests that there’s not only a need for a better understanding of how the body and the brain interact, but also a better understanding of how personality factors contribute to the susceptibility to the mirror box illusion, and finally how the illusion affects different patient groups, so that we might come closer to understanding who will benefit and who would not. In the present study, we investigate tactile acuity, body ownership and inner representations of the body in a group of healthy volunteers before, during and after being exposed to a mirror illusion that distorted the image of the left hand by enlarging or decreasing its size. The illusion was induced using an augmented reality technology. The aim was to optimize the methodology of the mirror illusion, to study the possible relationship between the sense of ownership, the limb size and the strength of the illusion. Our results show that cognitive representations of the body can be modulated strongly by the visual modality. Furthermore, the augmented reality design appears more effective than the classical mirror design.
THE ROLE OF SENSORY AND PERCEPTUAL INTEGRATION IN THE THERMAL GRILL ILLUSION

Thermal Grill Illusion (TGI) is an illusion introduced for the first time by a physiologist Thunberg in 1896 showing that spatially interlaced warm (40°C) but no-painful and cold (20°C) but no-painful stimuli induce a paradoxical sensation of strong heat comparable to the burning sensation that commonly accompanies cold pain. Despite the studies on the topic (Craig and Bushnell, 1994; 1996; Green 2002; Fruhstorfer et al., 2003; Bouhassira et al., 2005; Defrin et al., 2008; Li et al., 2009), the underlying neurophysiological mechanisms of TGI are not completely understood. Neither if the illusion produces a painful burning perception. In fact, contradictory and not uniform results emerge from attentive analyses of the literature (Craig and Bushnell, 1994; 1996; Bouhassira et al., 2005; Fruhstorfer et al., 2003). Nevertheless, the thermal grill illusion has an important relevance in the study of pain since it can provide a unique tool to study the central basis of pain perception. It has a physiological relevance since it underlines the interactions between pain and thermal integrations. Additionally, it has also a neuropsychological relevance; if pain sensation can be produced by innocuous stimuli then pain experience involves more than the stimulation of the receptors in the skin, consequently pain is an active product of the mind and the result of a multidimensional experience.

Our research has investigated the phenomenon of TGI on healthy volunteers with the aim to study the neuropsychological differences observed in the experience of pain illusion. For the first time we record electroencephalography (EEG) during the experience of thermal grill illusion. We then compared the brain responses of those who experience painful illusion (called responders) versus those who do not experience painful illusion (called non-responders). The presence of painful TGI can be observed by changes in the brain oscillations over the contralateral and central frontal areas. Moreover, the difference of baseline EEG power in Alpha2 frequency band (10-12 Hz) between responders and non-responders to thermal grill stimulation suggested that psychological and cognitive factors such as the anxiety level or...
attention and anticipation to pain experience may be among the influential factors that mediate painful thermal grill illusion.

**CONCLUSION**

In conclusion, pain is a complex phenomenon, distinct from the classical senses because it is multifaceted and it is influenced by a number of factors. In the pain response converge both interoceptive (the sense of physiological condition of the body) and exteroceptive (cutaneous mechanoreception and proprioception) components. Illusory phenomena might shed light on some of the neuropsychological mechanisms that influence pain perception.

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Schizophrenia is a very complex mental illness with different symptoms such as specific psychotic symptoms (e.g. third person hallucinations and delusions), negative symptoms (e.g. anhedonia and reduced verbal fluency), formal thought disorders (e.g. neologisms and reduced ability for abstract thinking) and specific neurocognitive deficits (e.g. reduced attention, working memory and executive functions).

In addition patients with schizophrenia also have comprehensive deficits in social cognition compared to healthy people, and to a larger extent than most patients with other mental disorders.

There is a large degree of heterogeneity among patients in the severity of all of the abovementioned symptoms and deficits.

There is growing evidence that aspects of social cognition, primarily theory of mind, may serve as a mediator between neurocognition and functional outcome in schizophrenia (Fett et al., 2010).

In 2008, the CNTRICS-initiative (Cognitive Neuroscience Treatment Research to Improve Cognition in Schizophrenia, from the National Institute of Mental Health, USA) specified the following areas of social cognition to be of particular interest for research in schizophrenia: Theory of mind, social perception, social knowledge, attributional biases and emotion processing (Green & Leitman, 2008; Green et al., 2008). These social cognitive deficits are stable regardless of the clinical state of patients. In fact, a variety of social cognitive deficits and biases are thought to partially underlie some of the psychotic symptoms, e.g. paranoia. In current clinical practice, social cognitive deficits are not routinely assessed in a valid way, and therefore these deficits are not taken into account when planning treatment strategies for patients.

There is a fairly long tradition of doing cognitive behavioral therapy (CBT) in psychosis (Morrison & Barratt, 2010). The basic assumption is that people with schizophrenia are simply trying to find an explanation of the weird things they are experiencing due to their symptoms. This causes them to create bizarre beliefs, e.g. delusions, about themselves, other people and the world in general.

CBT focuses on modification of these problematic beliefs and thus changing the distressing emotional components of the symptoms.

CBT is often combined with psychoeducation (which
involves teaching patients about the symptoms of schizophrenia. Traditionally, psychoeducation and CBT focus primarily on positive symptoms, negative symptoms and to a smaller degree neurocognitive deficits, without including the social cognitive deficits. There is a need for this to be changed in the future so that both patients and therapists achieve a better understanding of the patients’ specific social cognitive difficulties and can take these into account when performing CBT.

In CBT the aim is to reduce and normalize a patient’s symptoms via a case conceptualization which includes important aspects of the patient’s past experience (e.g. psychiatric vulnerability, onset of symptoms and important life events). Hypotheses are then made of how these core features resulted in specific conditioned and unconditioned beliefs about the patient himself and other people, and how these contributed to the patient’s current coping strategies. In the future CBT should also focus on the quality of the patient’s former interpersonal relations trying to detect the abovementioned social cognitive deficits to get a more realistic view of the patient’s interpersonal abilities. This knowledge should influence the rest of the therapy as to whether it is a matter of social cognitive deficits which acquires development of compensatory strategies or whether it is a matter of problems which could be changed and modified via cognitive reframing. In cognitive reframing the patient’s problematic life events are analyzed via cognitive restructuring of the patient’s negative automatic thoughts trying to help the patient generate more adaptive and neutral ways of thinking of the specific event. However it is a well known fact that paranoid patients in particular have severe difficulties with metacognition. In the future CBT should combine cognitive restructuring with psychoeducation of the three specific attributional errors that paranoid patients tend to use when analyzing life events: 1) jumping to conclusions, 2) bias against disconfirmatory evidence, and 3) personalizing bias (blaming others rather than situations for negative events) (Penn, Sanna, & Roberts, 2008).

Cognitive therapists should also integrate other aspects of social cognitive training in therapy in order to help the patient compensate for recurrent interpersonal stressors. Relevant topics in this sense could be: 1) the point of view of other people may be different from your own; 2) people do not necessary literally mean what they are saying (examples are irony, humor, and sarcasm); 3) interpretation of social interaction involves the tone of voice, situational context, etc. Also of importance is to integrate knowledge of specific deficits in emotional processing such as: 1) problems regarding recognizing sadness and fear in other people; 2) difficulties in expressing one’s own feelings; 3) difficulties understanding and handling the feelings of other people.

Besides the above mentioned implications for future CBT treatment in first episode schizophrenia, the specific social cognitive deficits in schizophrenia also have implications for therapists getting more realistic expectations regarding the quality of the therapeutic relationship.

But it also raises a more general question since CBT therapy often is group based. Many patients are not able to participate in these groups or tend to participate on a very irregular basis. Perhaps it would be more realistic to focus on specialised individual treatment. The patient’s social cognitive deficits might mean that therapeutic groups are far too stressful for many of the patients.

In practice, social skills training often focuses on social problem solving and behavioural training based on a philosophy such as “fake it till you make it”. However it seems that we have been far too optimistic and need to start psychoeducating and training patients at a much more basic level.

Therefore, knowledge about the patients’ social cognitive deficits should influence almost all aspects of future cognitive treatments of schizophrenia.

REFERENCES
The Social Cognition and Interaction Training – in short SCiT - is a psychosocial treatment to address functional impairments in patients with schizophrenia. The SCiT was developed by David Roberts and David Penn as a group training for patients with schizophrenia (Roberts & Penn, 2006). It focuses on active social cognitive processes of daily life, such as the effective use of social cues in real-world situations. This comprises the domains of emotion perception, theory of mind (ToM), and attributional style in ambiguous situations. The training was developed to improve both social cognition and social functioning in real life of people with schizophrenia.

In a pilot study, Penn, Roberts and colleagues (Penn et al., 2007) demonstrated in a group of forensic patients with schizophrenia that SCiT was associated with improved emotion perception, theory of mind, and attributional style in ambiguous situations. The training was developed to improve both social cognition and social functioning in real life of people with schizophrenia.

In another study comparing SCiT and treatment as usual (TAU) to TAU alone in outpatients (Roberts & Penn, 2009), individuals receiving SCiT and TAU again improved significantly in emotion perception relative to TAU at a medium-effect size level. However, stable ToM improvements could not be found in this sample. A large effect size instead was found for the patients’ social skill performance when belonging to the SCiT group, suggesting strong improvement in social competence. In a first uncontrolled study in a community setting (Roberts et al., 2010), results revealed significant improvements in emotion perception and theory of mind corresponding to effect sizes in the small to medium range, but no effects on attributional biases.

In a first uncontrolled study in a community setting (Roberts et al., 2010), results revealed significant improvements in emotion perception and theory of mind corresponding to effect sizes in the small to medium range, but no effects on attributional biases.

A large randomized controlled trial with a longitudinal design is still missing as well as further effectiveness studies. Another question not answered yet is whether or not there is a direct effect on social functioning, which has been measured only indirectly in the previous studies. Beyond, floor or ceiling effects of measurements concerning ToM and attributional bias may have prevented sensitive evaluation of change.

Nevertheless, these data show that SCiT is promising in routine settings of in- and outpatients with psychotic disorders who seek to improve their social functioning.

SCiT is appropriate for individuals who are at least 18 years of age, suffering from a psychotic illness, and who have interpersonal difficulties as a result...
of their illness. SCIT is designed for individuals in the non-acute phase of the illness, although symptom exacerbations may occur. The focus on cognitive and emotional aspects of interpersonal functioning is especially important. SCIT is particularly appropriate for individuals with symptoms of suspiciousness and paranoia. It is less useful for individuals with profound cognitive impairments or for individuals with serious substance abuse or dependence problems.

The group therapy is administered over 20-24 sessions at least once per week, dependent on the needs of the clients. The SCIT consists of three phases: (1) emotion training, (2) analysis of interpersonal situations, and (3) integration in daily situations of real life.

The initial two sessions are spent building group alliance and introducing clients to the SCIT group and the concept of social cognition. After this introduction, the remainder of Phase I is spent defining basic emotions, discussing the relationship between emotions and social situations, and identifying facial expressions of emotion. In Phase II, the primary goals are learning to recognize jumping to conclusions and attributional biases with an emphasis on perceived hostile intents towards other people. In addition, it is aimed at searching for alternative explanations of negative events, perspective taking, gathering guesses by tolerating ambiguity, and distinguishing guesses from social facts. The final phase of SCIT is a consolidation of skills learned up to this point, and application of these skills to clients’ own lives. It is the most important section of the manual and the most challenging for both clients and therapists. Clients are encouraged to bring in problematic interpersonal situations and events from their lives. The group then analyzes the situations and develops behavioral strategies based on SCIT skills.

In a first pilot study using a German translation of the SCIT manual, we intend to examine the efficacy of the SCIT in outpatients’ daily clinical routine on health related outcome parameters such as psychosocial functioning. Specific attention will be given to patients with first-episode schizophrenia in comparison to chronic patients with schizophrenia. The study will be carried out using a randomized 2-group pre-post longitudinal design (including 6-month and 1-year follow-ups). Patients will receive either SCIT twice a week over a 10-week period or will get CogPack (Marker, 2007) as control condition focusing on verbal, intellectual and occupationally-related cognitive skills. It is expected that SCIT is superior to CogPack in terms of primary outcomes like theory of mind and emotion perception and secondary outcomes like psychosocial functioning and quality of life.

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Individuals with schizophrenia are characterized by deficits in social cognition when compared to healthy control persons. Social cognition is an umbrella term covering domains such as emotion perception, theory of mind, attributional bias, and social perception. Social cognition has been shown to be related to everyday life functioning in schizophrenia. Because social cognition is central to the disorder and impacts on functioning, several new treatment programs targeting social cognition have been developed during the last years.

This symposium reviewed the literature of the mediating role of social cognition on the relationship between neuropsychology and functioning in schizophrenia. Three different social cognitive treatment programs were described. Training in Affect Recognition (TAR) was the first to be developed. Its method and rationale was presented along with data showing that social cognitive deficits are remediable through the use of TAR. Cognitive behavioral therapy (CBT) in schizophrenia has traditionally focused on reducing the emotional component of symptoms through psychoeducation and modification of dysfunctional beliefs.

The symposium demonstrated how social cognitive deficits can be integrated when doing case conceptualizations and cognitive reframing as part of CBT. Social Cognition and Interaction Training (SCIT) is a treatment program that was developed to improve both social cognition and social functioning among persons with schizophrenia. The training program was described and the outline of a new study was presented.

Summaries of Daniela Schaub’s and Vibeke Fuglsang Bliksted’s contributions are included here. See also a summary of professor Brüne’s talk in the current issue.

AUTHORS:
1. Vaskinn, Anja; Oslo (organizer): Neurocognition, social cognition, and functioning in schizophrenia: A mediation model
2. Frommann, Nicole; Düsseldorf: Training of affect recognition: An approach to remediate social cognitive dysfunction in schizophrenia
4. Schaub, Daniela; PhD student, psychologist, Bochum: Social cognition and interacting training (SCIT) for patients with schizophrenia.
Looking back only a couple of decades, neuropsychology was puzzled by – if not altogether agnostic towards – subcortical structures, and found the functions of the frontal lobes “enigmatic” (Fellows, 2007). Neuropsychology at this time was primarily a study of behavioural and mental aberrations resulting from cortical injury and degeneration. In the time until today, multidisciplinary studies of decision making in health and disease have led to new advances in our understanding of the brain bases of decision making. For example, our understanding of subcortical structures such as the striatum has moved from a focus on motor control functions, to a more comprehensive view that now includes emotional responses, outcome expectation and behavioural regulation (Horvitz 2009; Rushworth, Mars, Summerfield 2009; Schultz, Tremblay, Hollerman 2003).

Theories now focus on the interplay between cortical regions such as the ventromedial and dorsolateral prefrontal cortex, and subcortical regions including the striatum and amygdala, in the integration and regulation of emotional behaviour. Today, the realisation that this network is engaged in value-based decision making has spurred whole new fields of scientific endeavour, such as neuroeconomics (Schaefer 2009, Sanfey & Chang 2008) and decision neuroscience (Jessup & O’Doherty 2010; Huettel 2010). Other multidisciplinary areas, such as neuromarketing (Lee, Broderick, Chamberlain 2007), neuroaesthetics (Cinzia & Vittorio 2009) and consumer neuroscience (Kenning & Plassmann 2008), now suggest that we may understand the changes seen in brain injured patients’ daily activities.

From economics and other studies of human decision making, it has been realised that we deviate from optimal and “rational” choices. Such deviations relate to the engagement of the brain’s emotional circuitries. Avoiding financial risks are found to involve regions such as the amygdala and insula (Dreher 2007), while being risk seeking involves stronger activation in the ventral part of the striatum, often referred to as the nucleus accumbens (Kuhnen & Knutson 2005). Recent studies now suggest a distinction between a motivational “wanting” system driven by incentives and often operating under the limen of awareness, and a second “liking” system denoting the experience of pleasure, or lack thereof (Berridge, Robinson, Aldridge 2009). Those two systems are also thought to be based on different neural systems, including the ventral striatum and orbitofrontal cortex, respectively.

In this workshop, introduced by Thomas Z. Ramsøy, researchers from the Decision Neuroscience Research Group (DNRG) presented some of the latest insights into the different neural, behavioural and contextual factors that underlie decision making behaviours in many different contexts. The DNRG is a collaboration between the Copenhagen Business School and Danish Research Centre for Magnetic Resonance (DRCMR) at Copenhagen University Hospital Hvidovre, both in Denmark. Through this collaboration, researchers seek an improved understanding of the neurobiological bases of preference formation and decision making in everyday settings.

In his talk, Hartwig Siebner discussed how repetitive transcranial magnetic stimulation (rTMS) can be used to shape brain networks involved in the updating of pre-planned actions. He presented the results of study which has been recently published in Journal of Neuroscience (Ward et al. 2010). In this study, the excitability of the left rostral dorsal premotor cortex (rPMd) was transiently suppressed by 1 Hz rTMS. After this, subjects underwent functional magnetic resonance imaging while making spatially congruent button presses with the right or left index finger in response to a left- or right-sided target. Subjects were asked to covertly prepare motor responses as indicated by a directional cue presented one second before the target. On 20% of trials, the cue was invalid, requiring subjects to readjust their motor plan according to the target location. Compared with sham rTMS, real rTMS increased the number of correct responses in invalidly cued trials. After real rTMS, task-related activity of the stimulated left rPMd showed increased task-related...
coupling with activity in the ipsilateral supramarginal gyrus (SMG) and the adjacent anterior intraparietal area (AIP). Individuals who showed a stronger increase in left-hemispheric premotor–parietal connectivity also made fewer errors on invalidly cued trials after rTMS. These results suggested that rTMS over left rPMd improved the ability to dynamically adjust visuospatial response mapping by strengthening left-hemispheric connectivity between rPMd and the SMG–AIP region. The results further supported the notion that left rPMd and SMG–AIP contribute toward dynamic control of actions and demonstrate that low-frequency rTMS can enhance functional coupling between task-relevant brain regions and improve some aspects of motor performance.

Pathological gambling is a progressive and chronic disorder that encompasses an unrelenting failure to resist impulses to gamble. It is a maladaptive behavior that disrupts or damages personal, family or vocational pursuits, and is now diagnosed as one of several formal impulse control disorders (ICD) by international classifications (DSM-IV-TR). Sofie Gelskov, a PhD student at DRCMR and DNRG reviewed the existing literature on neuroimaging of pathological gamblers. Through this, she suggested that these studies have mainly focused on the sensitivity to reward and punishment, attentional bias, cue reactivity, impulsivity and executive functioning. Only a few imaging studies have directly investigated decision making in pathological gamblers. fMRI studies on pathological gamblers have revealed differential activity in frontal brain areas such as the vmPFC, DLPFC and ACC as well as striatal areas. These are areas related to diverse processes, such as conflict-solving, evaluation, emotional processing and reward processing. Nevertheless, the role of areas processing aversive events, like the amygdala and insula, has not yet been investigated directly, and could play a pivotal role in the development of pathological gambling. As these regions are recently shown to be a part of the emotion-based decision making apparatus (Rushworth, Mars, Summerfield 2009) current research is focusing on the role of such structures in pathological gambling, as well as gambling behavior in general.

It has become increasingly clear that aesthetic emotions also motivate behaviour. In his talk, Martin Skov demonstrated how aesthetic cues can be thought of as sensory information that give rise to aesthetic valuation, including the feeling of like or dislike, signalling a valuation of targets in their environment. For instance, when the target of evaluation is a potential mate, research has shown that heterosexual males respond to cues that signal reproductive fitness, such as body symmetry, facial attractiveness, and healthy skin and hair (Gangestad & Scheyd, 2005; Rhodes, 2006). Critically, such cues influence behavior, as evidenced by patterns of mate preference and choice in heterosexual males (Johnson, 2005). In other social interactions aesthetic cues play a role as well. For instance, more attractive children are less punished (Dion, Berscheid, & Walster, 1972), attractive students are judged more favorable (Ritts, Patterson & Tubbs, 1992), and in mock court cases attractive defendants are found less guilty (Parry, 2008). In a series of experiments facial attractiveness has been shown to bias how people are treated in workplace situations, influencing hiring, pay and promotions (Hamermesh & Biddle, 1994; Mobius & Rosenblat, 2006: Judge, Hurst & Simon, 2009). Indeed, in studies using economic games to directly investigate social interaction levels of attractiveness have been shown to modulate trusting behavior (Wilson & Eckel, 2006; Andreoni & Petrie, 2008), and influence how generous people act towards others (Hancock & DeBruine, 2003; Van den Bergh & Dewitte, 2006).
The question raised by these diverse strands of research is how aesthetic cues work to influence decision-making? In my talk I propose that recent neuroimaging research point to a possible answer. Studies investigating the neural correlates of aesthetic evaluation in different domains suggest that aesthetic values are computed primarily by the human reward system (e.g., Blood, Zatorre, Bermudez & Evans, 1999; Blood & Zatorre, 2001; Aharon, Etcoff, Ariely, Chabris, O’Connor & Breiter, 2001; Erk, Spitzer, Wunderlich, Galley & Walter, 2002; Vartanian & Goel, 2004; Kawabata & Zeki, 2004; Menon & Levitin, 2005; Yue, Vessel & Biederman, 2007; Winston, O’Doherty, Kilner, Perrett & Dolan, 2007; Kirk, Skov, Hulme, Christensen & Zeki, 2009; Platek & Singh, 2010). Specifically, fMRI studies have confirmed that attractiveness judgments are correlated with increased neural activity in structures known to play a role in evaluative judgment and reward processing, such as the nucleus accumbens, the ventral striatum, and the orbitofrontal cortex (Haber & Knutson, 2010). Furthermore, the human brain is also responsive to evaluative judgments involving political leaders, such as verbal statements of the form “I like George W. Bush: yes/no” (Zysset, Huber, Ferstl & von Cramon, 2002). In fact, it has been suggested that a system involving orbitofrontal and striatal neurons may underlie valuation of rewards irrespective of the modality giving rise to the rewarding stimuli, and that a second system involving the rostrolateral prefrontal cortex may underlie self-relevant evaluations across a wide range of situations. These findings locate processes underlying the generation of aesthetic values within the neural system for value-based decision-making (Rangel, Camerer & Montague; Kable & Glimcher, 2009). Further research must determine exactly how aesthetic cues activate the reward system in order to motivate behavior.

In the literature, social decision-making is a wide concept. Susanne Henningsson, a post.doc researcher at DRCMR and DNRG, presented the case of social decision-making. This is defined as the decision making in social contexts, in which one’s decisions may affect other persons. This category of social decision-making includes economic games such as the ultimatum game and the trust game. In the ultimatum game proposers choose how fair they should be when dividing a sum of money between themselves and a responder, whereas the responders choose whether to accept or reject these offers. In the trust game, investors choose whether to trust a trustee, whereas the trustees choose to repay or betray after the money has been multiplied. In addition, the investors sometimes get the possibility to choose whether to punish the trustees for their behaviour or not. Choosing between social stimuli, such as faces, is called social decision-making, and, also, making judgments about how much of a property (e.g. trustworthiness or intelligence) that a face expresses is called social decision-making.

Emotions play a big role in social decision-making, since there are several emotions such as envy that only are present in relation to other people. Even if there are many possible interpretations of the same actions in the social economic games, valuable insights about how social emotions and decisions are processed has been attained by studying these games with fMRI, TMS and drug challenges: When responders are proposed unfair offers in the ultimatum game, activity in the amygdala and insula is higher and also predict a future rejection of the offer (Sanfey 2003, Gospic 2010, submitted), a finding that is specific for the social situation since this is not the case when the proposer is a non-social machine. Behaviour in this game has been shown to be influenced by serotonergic transmission (Crocket 2008), inactivation of the DLPFC (Knoch 2006) and to be altered in subjects with vmPFC lesions (Koenigs & Tranel 2007). When it comes to the trust game, caudate nucleus activity correlates with the satisfaction of punishing a trustee for not repaying (de Quervain 2004). Oxytocin has been shown to make investors continue trusting the trustee also after they have been betrayed (Baumgartner 2008).

Other findings in the social decision-making field are that comparisons of options appear to be processed in similar ways when the nature of the options is social (people) as when it is non-social (numbers) (Kedia, submitted). In addition, the same region of the vmPFC has been shown to correlate with choice difficulty in social (choosing whether to punish the trustee) and non-social (choosing between foods from a menu) settings (Arana 2003, de Quervain 2004).
Taken together, this session on decision neuroscience demonstrated a variegated list of projects that all seek to understand different aspects of decision making behaviour in humans. While much of this research is focusing on decision making in healthy subjects, such insights may be employed to improve our understanding of decision making deficits in neuropsychology. As our understanding of these processes now stress subcortical structures, and the role of emotional functions in “rational” decisions, researchers and practitioners alike within neuropsychology should focus on these aspects in future work.

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**AUTHORS:**

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2. Martin Skov: Aesthetic cues in social decision-making.
4. Sofie Gelskov: Neuroimaging findings related to social decision making.
5. Susanne Henningsson: Neuroimaging findings related to social decision making.

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**Cologne, Bangor University, (2010) HBM poster**


The symposium started with a description of brain development and the neural networks that are crucial for the optimal maturation and the process for self-regulation. Implications to the development of cognitive, emotional and social skills was outlined.

Neuropsychiatric disorders include a wide variety of syndromes that have a brain-derived background. The symptoms, causing difficulties in reciprocal and interactive communication, are based on the un-equilibrium of self-regulation as well as in motivational, cognitive and emotional balancing. A case example of a neuropsychotherapeutic rehabilitation process with a teenager with Asperger syndrome character was described.

Motivational regulation includes cognitive and emotional knowledge about the state of mind and body functions. The information processing between different integration areas in the brain is mediated via five frontal-subcortical circuits. The specific areas of the frontal cortex are connected to certain lower brain structures, which are inevitable in motivational and behaviour regulation. The system also integrates the somatic and autonomous reflexes, the physiological machinery into the regulation network. The same brain structures are involved in both physiological, cognitive and emotional regulation. The motivational network in the brain underlies human emotion. A case example of a neuropsychotherapeutic rehabilitation process with an adult, who got TBI, was described.

In order to have realistic tools and goals for the neuropsychotherapeutic intervention, a wide and well-structured neuropsychological investigation is needed. Understanding the underlying theories and concepts in neurosciences and functional integrity of brain-behavior systems and self-regulation helps the clinician to plan the assessment methods. The investigation itself should be seen as a therapeutic and emotionally meaningful process for the client. The wiring and tuning of the motivational machinery is the first priority for reciprocal and emotionally curing therapeutic co-operation. Neuropsychotherapeutic investigation was illustrated by a few clinical syndromes.

**AUTHORS:**

2. Paavola, Liisa: The neuropsychotherapeutic process with a teenager with Asperger Syndrome characters: Diagnostic and rehabilitational perspectives.
3. Loukkola, Jukka: Motivational regulation and its effect on mental processing in neuropsychiatric disorders: A case example of an adult client with TBI.
4. Ylikoski, Raija: Neuropsychological assessment as a tool for understanding the neural networks and planning neuropsychotherapy.
5. Laaksonen, Ritva (Discussant)
It is increasingly clear that many clinical conditions are characterized also by impairments or alterations in social cognition, and that working through social cognition may be part of a possible therapeutic intervention. The interacting minds project at Aarhus University examines links between the human capacity for minds to interact and the putative biological substrate, which enables this to happen. In this mini symposium, we present four studies where this perspective may throw new light on a clinical condition.

SOCIAL COGNITIVE DEFICITS IN FIRST-EpISODE SCHIZOPHRENIA

Patients with schizophrenia have comprehensive deficits in social cognition compared to healthy people, to a larger extent than most patients with other mental disorders. There is growing evidence that aspects of social cognition, primarily social perception, may serve as a mediator between neurocognition and functional outcome in schizophrenia. Vibeke Bliksted presented preliminary results from a study of social cognitive deficits in patients with first-episode schizophrenia. The results suggest that, while general cognitive deficits are indeed typical of this group, deficits in social cognition are particularly severe and can occur in the absence of other cognitive problems. In particular, these patients have great difficulty in distinguishing between sincere and sarcastic speech. These results suggest that future therapeutic interventions should differentiate between different social cognitive “profiles” and that specific training in social cognition may be essential.
WHAT’S LEFT TO LEARN ABOUT RIGHT HEMISPHERE DAMAGE, PRAGMATIC IMPAIRMENT, AND SOCIAL COGNITION?

After two decades of research on pragmatic impairment following right hemisphere damage (RHD), we are left with more questions than answers. There is general agreement that social cognition is important for pragmatic function, and that damage to right hemisphere areas can result in impairment of both social cognition and pragmatic function, but there is as yet no consensus about how or why. Ethan Weed presented behavioural and physiological (EEG) data from patients with right hemisphere damage and healthy controls confirming a role for the right hemisphere in inferring intentions from low level motion cues, as well as in recognizing emotions from facial expressions and speech prosody. He showed that the right hemisphere also has a role in resolving discrepancies between these social cues. It is from this combination of factors that the role of the right hemisphere in pragmatics emerges.

EMPIRICAL INVESTIGATIONS OF PAIN MODULATION FROM RELIGIOUS PRACTICES

It is commonly known that religious patients often turn to religion as a source for coping when dealing with illness or other negative life events. But it has never been clinically tested how religious coping strategies such as personal prayer modulate pain intensity and pain unpleasantness or how the subjective assessment of this activity correlates with autonomic functions in healthy subjects. Else-Marie E. Jegindø presented experimental studies demonstrating how expectations, such as those associated with prayer, can modify both the experience and the physiological responses associated with pain. She also presented video footage from religious rituals demonstrating the extraordinary extent to which the experience of pain can be modified by expectations.

CAPGRAS DELUSION EXPLAINED BY FUNCTIONAL ASYMMETRY OF THE FUSIFORM FACE AREA

Tony Jack presented some striking new observations on the neural basis of Capgras Syndrome. The Capgras Delusion is a disorder in which a loved one is believed to be replaced by an identical looking impostor. It is prevalent in 13.3% of patients with Alzheimer’s disease (AD). Capgras sheds light on the nature of human attachment, and can be dissociated from another facial recognition disorder, prosopagnosia. Together these disorders suggest dual routes to facial recognition, with one route for visual recognition, via the fusiform face area, and a second route for emotional recognition via the amygdala. New fMRI evidence from a patient with Capgras Delusion, alongside data from controls (with and without AD) suggest that there is a functional asymmetry between the left and right fusiform face area which underpins the appearance of this disorder.

AUTHORS:

1. Andreas Roepstorff, Department of Social Anthropology and, Centre for Functionally Integrative Neuroscience (CFIN)
2. Vibeke Fuglsang Bliksted (PhD Candidate, Aarhus University)
3. Ethan Weed (PhD Candidate, Aarhus University)
4. Else-Marie E. Jegindø (PhD Candidate, Aarhus University)
5. Tony Jack (Assistant Professor, Brain, Mind and Consciousness Lab, Case Western University, Cleveland, Ohio)
The focus of the two presenters was upon psychopathy as a general construct and the association between psychopathy and violence from a mentalization-based perspective. Psychopathy continues to be one of the most discussed and debated “constructs” in psychiatry. It is not a (formal) diagnosis, it cannot (easily) be treated, the psychopath himself is most often not suffering from his “condition” – but his surroundings are as psychopathy is closely linked to violence, and the forensic system often deals with both psychopaths and their victims.

Psychopathy is generally assessed in adults, but over the past few decades an increasing awareness among clinicians and researchers has given attention to psychopathy in adolescents.

Converging evidence exists to suggest a robust association between psychopathy and violence. Still, however, little is known about the underlying bio-psycho-social mechanisms mediating this association. In Mickey Kongerslev’s presentation, a review of studies demonstrating such links was presented. Then the findings were discussed from a mentalization-based perspective. Mentalization, defined as the processes by which human beings understand themselves and others as psychological agents and beings, is a multidimensional social-cognitive-affective construct. It was argued, that partial dysfunctions in mentalizing might be some the core psychological mechanisms underlying psychopathy, and mediating/explaining its links with violence and other antisocial tendencies.

AUTHORS:
1. Wøbbe, Tine; Retspsykiatrisk Afd., Psykiatrisk Center Sct Hans Roskilde, Denmark
2. Kongerslev, Mickey Toftkjær; Ungdomspsykiatrien i Region Sjælland, og Det Sundhedsvidenskabelige Fakultet ved Københavns Universitet
Psychopathy tends to intrigue and capture the interest of researchers, professionals and the everyday man. It is not a (formal) diagnosis, it cannot (easily) be treated, and the psychopath himself does not suffer from the ‘condition’, although his surroundings usually do. Psychopathy is closely linked to violence, and the forensic system often deals with both psychopaths and their victims.

The first aim of the symposium was to outline the diagnostic issues and problems concerning the ‘construct’ of psychopathy. Neither one of the two major diagnostic systems (i.e. DSM-IV & ICD-10) fully comprise the core of what is generally perceived to be psychopathy. It leaves the clinician (as well as the researcher) with unanswered questions such as (i) whether psychopathy is a specific personality disorder? (ii) An adaptive reproductive strategy? (iii) Or a clinical construct traditionally defined by a cluster of personality traits and behaviours?

Professor Robert Hare, in the search of a more specific conceptualisation of psychopathy, developed the Psychopathy Check List (PCL-R). This is based upon four features traditionally associated with psychopathy, i.e., (i) problems regarding interpersonal relations, (ii) affective difficulties, (iii) life style problems, and (iv) antisocial behaviour, and consists of a 20 item scale and scores 0 – 40. It may give a ‘psychopathy cut-off’ (the extent to which an individual matches the prototypical psychopath) at around 30 (PCL-R mean community sample: 6.6).

Hare estimates that about 1% of the general population meet the criteria for psychopathy - as opposed to 10-20% in a male prison population (Hare, 1985, 2003; Hart & Hare 1997). This leads to one of the major problems with the PCL-R: it was developed and based upon a prison population. Critics have raised the question of whether it might be a specific ‘type’ of psychopath that the PCL-R assesses and describes (what about ‘white collar psychopaths’ for example?). Factor analyses (Cooke et al., 2001, 2004, 2007) have indicated a three-factor structure (with items strictly relating to antisocial behaviour removed from the final model): (i) Arrogant and deceitful interpersonal style, (ii) deficient affective experience, and (iii) impulsive and irresponsible behavioural style. Research into and debate about the construct of psychopathy continues, aiding clinicians in the difficult task of trying to treat psychopathy as well as providing new data for helping us to understand the origin of psychopathy.

Much evidence points to strong links between psychopathy (as assessed by the PCL-R), violent crime and an elevated risk of re-offending. The risk is higher for instrumental violence than for reactive violence. This means that adults with psychopathic characteristics present both the criminal justice system and the mental health care system with a major therapeutic challenge. Hence more research and knowledge is needed, both concerning treatment options, the psychological mechanisms contributing to (or causing?) psychopathy as well as the ‘core’ biological mechanisms that contribute.

Blair (2003, 2007) and his co-workers (2005) created a bimodal model of aggression and violence, dividing violence into instrumental (proactive/premeditated) and reactive (impulsive/affective). Meloy (2006) found no specific neuropsychological dysfunctions in the group that exhibited instrumental violence, whereas problems in executive functioning (as well as a negative correlation with verbal skills) were found in the group that exhibited reactive violence. Generally, research demonstrates two different populations of violent individuals: those who only show reactive aggression (e.g. with lesions to the frontal orbital cortex/BPD/bipolar) and those who show both types (individuals with psychopathy). Psychopathy is the only psychiatric condition to confer heightened risk of instrumental aggression, and this emphasizes the importance of understanding the neural and cognitive underpinnings of the condition. The research of Blair and his co-workers (e.g., 2005) as well as similar research by others has shown elevated responsivity in the amygdala in individuals with reactive violence, but a reduced responsiveness of the amygdala in individuals who execute instrumental violence. High PCL-R scores have been associated with reduced amygdaloid volume. An fMRI study presen-
Mentalization is a new word for an old concept related to normal adaptive human behavior and interactions. It has been used by Fonagy and Bateman (e.g., Allen et al., 2008) in order to understand and treat Borderline Personality Disorder. Mentalization develops in the attachment relationship and in relation to psychopathy it has been found to have elements of both intact mentalizing and partial mind blindness (e.g., failure of imaginative empathy, failure to identify with victims’ distress, a relative unimpaired cognitive understanding of self and other - but very impaired affective understanding). But so far, the more exact relationship between mentalization and psychopathy lacks specific empirical evidence. Focus upon the role of attachment and the interaction between biology, psychology, behavior and social components in the development (or reinforcing of existing) psychopathy is warranted.

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“It is not just IQ, but emotional intelligence that matters”. This quotation is from the popular science bestseller Emotional Intelligence (Goleman, 1995) which made the term emotional intelligence familiar to almost everyone, yet left many psychologists sceptical of the concept both in terms of its validity as an independent construct and whether it could be measured. Goleman also hyped his message, and as shown by Schmidt & Hunter (1998), IQ and other measures of ‘general mental ability’ have clearly greater predictive validity for success in terms of job performance than any other psychological measurement. However, this meta-analysis of 85 years of research findings also demonstrated interesting runners-up: integrity tests and conscientiousness tests. These were independent of IQ, and their modern successors are emotional intelligence tests. Efforts to develop such tests and to measure and apply emotional intelligence have flourished in the last 15 years. Some emotional intelligence tests rely on self reports, and others are performance based (Stough et al., 2009; Mayer et al., 2008).

The aim of the symposium was to present and discuss what may be the most serious – in both theoretical underpinnings and empirical research – performance or ability measure of emotional intelligence: the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCeIT). This test consists of eight subtests in four “branches” (i) Perceiving Emotions, which are two versions of the familiar emotional recognition tasks; (ii) Using Emotions, which examines a person’s ability to use emotions to facilitate cognitive processes; (iii) Understanding Emotions, which purports to measure the ability to understand the relationships between different emotions and how they may progress, change, and combine, as well as how emotions may arise from and facilitate social situations; (iv) Managing Emotions, which concerns the control of both one’s own emotions and those of others so that they can be used effectively.

We gave a description of the MSCeIT and its structure and psychometric properties based on both published data and our own data from two studies of normal subjects, including the standardization sample (N: 1094) in Denmark. Scoring of items is ‘normative’ in the sense that answers are not coded as ‘right’ or ‘wrong’, but scored with a weight based on the percentage of the answers given by the standardization population. While this scoring has been criticized for favouring the average rather than the ‘correct’, it is also highly consistent with expert rating. Potential participants for the standardization sample were randomly drawn from the CPR (population register), but predictably volunteers were younger and better educated than average. Age had minimal influence on results, whereas gender and education were of some importance. For the construction of norms (mean 100; SD 15) the sample was weighted based on the Danish census. Details of this and of reliability estimates have been published after the conference (Mayer et al., 2010). Reliability was predictably better for the composite scores (.73-.93 in Cronbach’s alpha coefficients) than for subtest scores. The factorial structure was tested in confirmatory factor analysis and found to be generally in accordance with the theoretical model outlined above.

As many would expect, gender was of some importance: about 5 EIQ points in total EIQ and 2-5 EIQ across subtest and composite scores. And yes, females scored higher on average. Differences between the highest and the lowest educational categories were moderate, e.g. 9 total EIQ points. A small curvilinear age effect was also seen in the data, with 30-60 year olds scoring a few points higher than those either younger or older.

We also used the MSCeIT in a study of 108 normal subjects also tested with personality inventories and traditional neuropsychological measures of cognitive domains such as memory, executive functions, language, and mental speed. These data allow a discussion of whether the MSCeIT is indeed a unique and distinct measure of emotional intelligence, or whether its correlations to IQ, executive and other cognitive measures, or personality are so high as to threaten this purported uniqueness. We found a moderate (r=.4) correlation with an IQ measure (Danish Adult Reading Test, DART), and low to no correlations with most measures of cognitive functions. One exception to this is slightly worrying: both MSCeIT total...
EIQ and composite measures correlated moderately $(r=0.3-0.51)$ with Boston Naming Test, a test of verbal semantic knowledge. This may indicate that the MSCeiT is quite demanding in terms of verbal understanding. This is also our informal observation, and indeed many items contain rather infrequent words and subtle linguistic distinctions. Thus, validity of the MSCeiT may be better in subjects with some advanced education.

Lately many scientific groups have shown an interest in self control and emotion regulation (e.g. Hassin et al., 2010; Gross, 2007), and the MSCeiT branch score ‘Managing Emotions’ has some promise in the study of individual differences in this important construct. This was also the sole test in the MATRICS initiative selected to represent social cognition (Nuechterlein et al., 2008). Studies of the neurobiology of single elements of emotional intelligence are at hand (cf. this meeting on “the social brain”), but studies on the neural bases of individual differences in emotional intelligence as measured by the MSCeiT or other emotional intelligence tests are scarce. So are intervention studies. For the study of neurobiological correlates more specific measures may also be more informative, and tests with more straightforward scoring may also be preferred.

It seems obvious that the MSCeiT may have an important role in e.g. personnel selection, but can and should it be used in clinical neuropsychological assessment? We envisage a limited use with patients suspected of frontal lobe lesions, without significant cognitive deficits, and some indication of impairment in social comportment and understanding. Indeed, few relevant tests for such patients exist, and studies of the ability of the MSCeiT to fill this void should be undertaken.

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FAMILY MATTERS: INCLUDING THE FAMILY IN BRAIN INJURY REHABILITATION

Rikke Kieffer-Kristensen (organizer)

Acquired Brain Injury (ABI) is a chronic condition that is associated with changes in the patients’ physical, cognitive and behavioural functioning and may have serious consequences on patients’ quality of life. Family stress following brain injury has in the recent years been well documented and the impact of brain injury on the family is often immense. ABI can be seen as a negative stressful life event that is likely to cause long-term strains, uncertainties and ambivalent feelings within the family (Lezak, 1988; Thomsen, 1974). Family members often have to redefine the relationships and roles.

PARENTAL ABI
In her presentation Kieffer-Kristensen presented the results of her investigation of the effect of parental brain injury. Two-parent families with dependent children seem to be particularly vulnerable since the brain injury creates practical and emotional demands on the parent, spouse and child simultaneously (Armistead et al., 1995). Parental ABI may thus have a distressing impact on the close relationships within the family. Dependent children seem particularly vulnerable since their caregivers become less available as attention shifts from the children to the brain-injured parent (Pessar et al., 1993).
This study is to our knowledge the first to examine post-traumatic stress symptoms in children with a parent with an acquired brain injury. The participants were 35 patients with acquired brain injury, their spouses and children aged 7-14 years recruited from outpatients Brain Injury Rehabilitation Units across Denmark. A matched control group consisted of 20 children of parents suffering from diabetes, which is another chronic illness, and were recruited from the National Danish Diabetes Register. In this symposium the results from the children’s questionnaires were presented.

Significant high levels of post traumatic symptoms in children with a parent with ABI compared to children in the control group were found (Kieffer-Kristensen et al., 2010). Nearly half of the children in the ABI group, 46%, had scores above cut-off when screening for posttraumatic stress disorder, compared to 10% in the control group. These findings make what we believe to be an important point for daily work in clinical practice. The treatment and rehabilitation of ABI in parents with dependent children must acknowledge that the need for support can be diverse as differences in trauma exposure among family members often occur. This study underscores the need for a more family orientated treatment with a child-centred approach, as parental ABI can have a profound effect on the children’s proximal and distal development.

PAEDIATRIC ABI

Another important topic is within the treatment of paediatric brain injury. Pediatric brain injury has been found to be highly distressing, especially for parents and siblings (Hawley et al., 2003; Wade et al., 2006). In her presentation From recognised the need for family support that exceeds the traditional definition of relatives, by including not only parents and siblings, but also grandparents, friends, and others. Based on her clinical practice within the paediatric brain injury rehabilitation setting From presented an overview of the counselling offered to the close relatives at the Children’s Rehabilitation Centre. The service addresses a wide variety of cognitive and emotional issues that often present barriers to recovery and community reintegration and to optimal family functioning. The aim of the counselling is to prevent isolation for any involved person and to present a better understanding of the nature of the brain injury. This type of support tends to lead to a better adjustment and to more realistic personal expectations for all involved parties.

Conclusion

In conclusion this symposium highlights the need for family intervention following ABI. Furthermore, a greater understanding of family relational functioning after ABI is required to improve and develop effective prevention approaches for the whole family in the rehabilitation process. The effect of ABI on the child-parent relationship is for that reason of great importance and most not be underestimated. Finally more child-centred interventions are needed.

REFERENCES


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From the reception at Aalborg Congress and Culture Centre

Malene Stoltze Rasmussen, Head of Organising Committee

Lene Sahlholdt
Co-chair of Scientific Committee

Jens Østergaard Riis
Head of Local Committee
The 10th Nordic Meeting in Neuropsychology
"The social brain – development and dysfunction”
Aalborg, Denmark, 15-18 August 2010

Kildeparken by Aalborg Congress and Culture Centre, venue of the meeting

Charlie and the Pearson brain

Conference dinner in Rold Skov

More photographs (including a link to a Picasa Webalbum collection):
http://gade.psy.ku.dk/Aalborg1.htm
INNHOLD

EDITOR
The 10th nordic meeting in neuropsychology in Aalborg

INVITED SPEAKERS
C. NEIL MACRAE:
The social brain: the face and person perception

SARAH-JAYNE BLAKEMORE:
The social brain in adolescence

UTA FRITH:
Autism: theory of mind revisited

FRANCESCA HAPPE:
Central coherence revisited

MARTIN BRÜNE:
Social cognition in Schizophrenia

PAUL BURGESS:
Executive functions of the frontal lobe

ESSI VIDING:
Development of the psychopathic brain

SIMONE G. SHAMAY-TSOORY:
The neural bases of competitive emotions

MICHAEL TRIMBLE:
Music, belief and the social brain

KRISTINE B. WALHOVD:
Neuroimaging of the developing brain

MORTEN KRINGELBACH:
Finding pleasure in the brain

CHRISTOPHER KIPPS:
Impairment in social cognitions is the core of frontotemporal dementia

TORKEL KLINGBERG:
Training plasticity of working memory

SHIHUI HAN:
Cultural selves, encultured brains

CHRIS FRITH:
The social brain: summing up and looking ahead

SYMPOSIA
AINA HOLMÉN:
Neurocognitive profile in early-onset schizophrenia

MARI STRAND:
Emotional information processing during recovery from MDD

MARIT SCHMID:
Inhibition in major depressive disorder- in recurrent- and first treated episode patients

MERETE GLENNE ØIE AND ERIK HESSEN:
Neurocognition and emotional processing in psychiatric disorders

JETTE STOKHOLM:
Frontotemporal dementia

ANDERS GADE:
Serotonin, emotions and the social brain

LAURA PETRINI:
Neuropsychological aspects of pain

VIBEKE BLIKSTED:
Social cognitive deficits in first-episode schizophrenia: implications for cognitive behavioral therapy

DANIELA SCHAUB:
Social cognition and interaction training (scit) for patients with schizophrenia

ANJA VASKINN:
Social cognition in schizophrenia

THOMAS Z. RAMSOY:
Decision neuroscience and neuropsychology

JUKKA LOUKKOLA:
The ever-lasting process of selfregulation throughout the life span: the alliance between affective neurosciences, neuropsychological rehabilitation and neuropsychiatry

ANDRÉAS ROEPSTORFF, CHRIS FRITH & UTA FRITH:
Interacting minds – clinical perspectives

TINE WØBBE:
Assessment and aspects of psychopathy

ANDERS GADE:
Emotional intelligence: focus on the MSCEIT

RIKKE KIEFFER-KRISTENSEN:
Family matters: including the family in brain injury rehabilitation

Closing ceremony
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