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Satellite symposia

SAT1 - Satellite symposium: TREATING SCHIZOPHRENIA WITHOUT SEDATING THE PATIENT: GOAL OR CHALLENGE Sponsored by Bristol-Myers-Squibb

SAT1.01

What does agitation mean in the acute setting?

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Agitation is a frequent symptom associated with schizophrenia, mainly in the acute or impending relapse phases, and can be defined as any inappropriate, excessive motor or verbal activity. Manifestations of agitation may include excitement, hostility, aggressive and destructive behaviours, verbal abuse and extreme personal distress. Agitation has an adverse effect on many aspects of psychiatric disorders, negatively impacting patient care, caregiver experience and society as a whole. In terms of patient care, the symptoms of agitation can hinder diagnosis and treatment of the psychiatric disorder. Delayed diagnosis and treatment and its associated effects can increase the duration of hospitalization for a patient. Agitation symptoms can heighten caregiver distress, as agitated individuals are generally perceived to be acting inappropriately. Among inpatients, agitation is a common warning signal that frequently precedes an act of violence and, therefore, is among the most fear-provoking aspects for caregivers. Potentially, this can lead to increased need for institutionalization, leading to societal implications due to the increased need for emergency care and the associated costs. Also, increased hospitalization further influences the patient experience - adversely affecting patient quality of life. Thus, addressing agitation as a symptom of schizophrenia is an important therapeutic target. Given the seriousness of these symptoms and their effects, together with the fact that patients with agitation associated with psychiatric disorders frequently present in the emergency department experiencing an acute psychiatric episode, rapid, effective intervention is key. The initial treatment period is critical for optimal patient outcomes, and an ideal treatment for a patient presenting with acute agitation would: calm the patient quickly, without excessive sedation; decrease the likelihood of harm to self or others; attenuate psychosis and associated symptoms; allow initiation of a therapeutic relationship between patient and physician; be easily and effectively administered; decrease the use of seclusion and restraint; and consider both short- and long-term treatment goals and patient health.

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SAT1.02

Sedation is not the opposite of agitation

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The induction of sleep was originally considered to be a desirable therapeutic endpoint for the rapid control of agitation associated with psychotic disorders. However, it has become clear that sleep is not essential for a decrease in agitation or for the rapid improvement in core psychotic symptoms. Indeed, although the initial calming effects of treatment may be considered useful, excessive sedation or 'oversedation' is not a desirable effect, as it can interfere with both the physician's ability to interview/evaluate the patient and establish an effective therapeutic alliance with them, and with the patient's ability to participate in their treatment (e.g., answer questions, hydrate themselves). Furthermore, oversedation has the potential to mask illnesses that show central nervous system depression as a symptom, which could lead to further morbidity or mortality. Thus, although sleep may be advantageous in certain circumstances, achieving control of agitation via rapid calming rather than sedation is becoming an important therapeutic goal. Management of acute agitation has traditionally involved the use of benzodiazepines, such as lorazepam; however, problems with oversedation have led to the increased use of intramuscular antipsychotics in place of, or in combination with, benzodiazepines. Although combination treatment, for example, with intramuscular haloperidol plus intramuscular lorazepam, may provide superior efficacy to treatment with either agent alone, the sedative effects are at least as great as with the use of benzodiazepines as monotherapy. Specific calming without excessive sedation is emerging as a significant clinical advantage of intramuscular formulations of atypical antipsychotics versus conventional treatments.

SAT1.03

Appropriate treatments for agitation associated with schizophrenia: Control of acute agitation and maintenance of efficacy

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The first challenge in the treatment of acute agitation associated with schizophrenia is to control agitation without excessively sedating the patient, while also treating the symptoms of schizophrenia. Although oral formulations of antipsychotics have shown efficacy in the treatment of agitation, some agitated patients may not be able to take oral drugs and it may be necessary to use an intramuscular form of medication. Intramuscular formulations of benziodiazepines, typical antipsychotics and, more recently, atypical antipsychotics, have all proved effective first-line therapies for the rapid control of agitation associated with psychotic disorders. Although widely used, intramuscular benzodiazepines have been associated with excessive sedation, and typical antipsychotics, such as intramuscular haloperidol, have a high propensity for causing acute extrapyramidal symptoms. Distressing side effects may adversely impact on patient acceptance of, and adherence to, future antipsychotic therapy. Intramuscular atypical antipsychotics may provide superior alternative treatments owing to improved safety and tolerability versus typical agents. Clinical studies have demonstrated the safety and efficacy of intramuscular formulations of aripiprazole, olanzapine and ziprasidone for the treatment of agitation associated with schizophrenia, and these agents have been approved for use in the USA and some European countries. Although rapid control of agitation is the primary goal, the longer-term effects of antipsychotic therapy also require consideration. Patients initially treated with an intramuscular antipsychotic will typically transition to oral therapy for the long-term management of their disorder. Therefore, the long-term safety and tolerability of oral therapy is important. For example, treatment-associated sedation can adversely affect patient quality of life and social integration during longer-term treatment, whereas treatment with antipsychotics that are associated with significant risk of weight gain, glucose dysregulation and dyslipidaemia may have serious implications for long-term patient health. Transferring from an intramuscular to an oral antipsychotic may impose a risk of the emergence of adverse effects, breakthrough symptoms and loss of therapeutic advantage, particularly if transitioning between intramuscular and oral formulations of different antipsychotics; ideally, continuation with the same agent would minimise this risk.

SAT2 - Lunch Satellite symposium: SEROTONIN, NORADRENALINE, DUAL -WHAT IS STATE OF THE ART? Sponsored by Lundbeck

SAT2.01

Pharmacological treatment of anxiety disorders - is there a state of the art?

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Selection of appropriate treatment for anxiety disorders is influenced by several considerations, including psychiatric comorbidity. Emerging data suggest that anxiety disorders have a chronic course and a high comorbidity with depression. Successful treatment can be facilitated by first establishing treatment goals, which include managing acute anxiety and following through to remission. Prevention of recurrence of anxiety disorders should be the ultimate objective.

Various treatment options exist for the treatment of anxiety disorders, including selective serotonin reuptake inhibitors (SSRIs), serotoninnoradrenaline reuptake inhibitors (SNRIs), tricyclic antidepressants, pregabalin, benzodiazepines, buspirone, and reversible and irreversible MAO inhibitors. Some SSRIs have been demonstrated efficacy in both acute and long-term trials. Regarding their risk-benefit ratio, they are established as first-line therapies. The combination of drug treatment with cognitive behaviour therapy (CBT) is also recommended.

The chronic nature of anxiety disorders, different treatment response among different anxiety disorders and the recognition of their frequent comorbidity with depression requires an informed and evidence based choice of the best pharmacological approach to the individual patient. The presentation will present the most recent data from randomised clinical trials of newer generation agents and put them into perspective, to help the physicians to appropriately diagnose anxiety disorders and achieve the goal of bringing patients to full remission.

SAT2.02

Requires severe depression a specific treatment?

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Depression is a disabling disorder associated with considerable comorbidity, risk of suicide and social consequences. Although antidepressants are among the most prescribed therapeutic agents, recent reviews highlight the significant percentage of depressed patients who fail to achieve a response or remission.

Although epidemiological and clinical data do not support severe depression as a separate illness category, and there is no consensus on the definition of "severe depression" regarding diagnostic scales, evidence suggest that the severity of depressive symptomatology may be associated with a worse prognosis and an increased mortality. Furthermore is there a perception that specific subpopulations of depressed patients e.g. melancholic patients or treatment resistant patients suffer of more severe forms of depression. The treatment of severely depressed patients is thus of major concern in view of the debilitating course of the disease.

Some early studies suggested that tricyclic antidepressants (TCAs) like clomipramine were more effective that selective serotonin reuptake inhibitors (SSRIs) paroxetine or citalopram in "endogenously" depressed patients. Other reviews report comparable efficacy of TCAs and SSRIs in patients with severe or melancholic depression, with SSRIs being better tolerated.

Recent data suggesting a surprisingly better differentiation of escitalopram, the active enantiomer of racemic citalopram, regarding effeicacy in more severely depressed patients (MADRS > 30 or > 35) versus SSRIs such as paroxetine and citalopram as well as versus the SNRI venlafaxine argue for a differentiated treatment approach, based on severity of symptoms.

SAT2.03

OCD quo vadis? The Cape Town consensus statement

H.G.M. Westenberg. Rudolf Magnus Institute of Neuroscience, Department of Psychiatry, University Medical Center Utrecht, Utrecht, The Netherlands

The perception of Obsessive Compulsive Disorder (OCD), once seen as a rare refractory condition, has changed significantly over the past two decades. Neuroimaging and genetic findings have advanced the understanding of the neurobiology of OCD and new treatment options have improved the outlook for patients.

A consensus group at the International Anxiety Disorders Conference in Cape Town, South Africa in February 2006, felt it was timely and appropriate to revisit OCD, to identify key developments in the field of OCD and to examine how they might be translated into clinical practice.

The group reviewed the currently available data on symptomatology, diagnosis, assessment, psychobiology and treatment of OCD in order to provide an up-to-data summary of the literature and recommendations for the treating physician. Special attention was paid to the current controversies about the relationship of OCD to OCD spectrum disorders, and the subtyping of OCD. An important issue was also of whether OCD should be considered as a distinct disorder, separate from the anxiety disorders. Based on the available evidence, it was proposed to remove OCD from the anxiety disorders and place it within a separate category of OCDs. To do justice to the complex and heterogeneous presentation of OCDs it was also proposed to utilize a combination of categorical and dimensional approaches in the diagnostic process. The consensus was that this would enable not only the tailoring of treatment, but would also be helpful to studies on the neurobiology and endophenotyping of OCD.

Key issues in the neurobiology OCD, including the role of serotonin and dopamine, the cortico-striatal circuits and genetic factors, were addressed with respect to their relationship to special populations, such as treatment resistant patients, tic disorders and "schizoobsessive' patients, and the response to various treatments.

SAT2.04

Escitalopram - a new option in OCD treatment

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Substantial evidence from controlled studies demonstrate efficacy for clomipramine and SSRIs in the acute treatment of OCD across the lifespan. There have been fewer studies of long-term treatment and it remains less conclusively understood as to how well treatments that have been shown to be effective in short-term studies maintain their efficacy over the longer term, though placebo-referenced trials suggest efficacy for clomipramine, fluoxetine and sertraline up to twelve months. Most relapse prevention studies in acute responders revealed a significant advantage for remaining on active treatment (paroxetine, sertraline and fluoxetine at higher doses). For some of these studies methodological problems impaired their ability to discriminate active from placebo treatment on the chosen relapse criterion.

In a double-blind dose-finding study, 458 OCD patients were randomized to escitalopram (fixed at 10mg or 20mg), or 40mg paroxetine or placebo. At week 12 - the primary efficacy endpoint - 20 mg escitalopram showed a significant improvement in the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) compared to placebo (p<0.005). At week 24, escitalopram 10mg (p<0.05) and 20mg (p<0.005) showed significantly greater improvements in Y-BOCS total scores than placebo - as did paroxetine 40 mg (p<0.005). In a relapse prevention study, 320 patients (ITT) who had responded following 16 weeks of open treatment with escitalopram, were randomized to placebo or escitalopram for a further 24 weeks of double-blind treatment. The primary analysis (time to relapse) showed a significant advantage for escitalopram (Log-rank test p<0.001), and the risk of relapsing was 2.7 times higher for placebo compared to escitalopram. These results suggest that escitalopram is effective for acute and long-term treatment and relapse-prevention in OCD.

SAT3 - Satellite symposium: RESETTING THE INTERNAL CLOCK IN DEPRESSION: A NEW THERAPEUTIC APPROACH Sponsored by Servier

SAT3.01

Effective management of depressed mood with agomelatine, a melatonergic antidepressant

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Agomelatine is a new antidepressant with an unique pharmacological profile. It is a potent agonist of melatonin receptors (MT1 and MT2) and also an antagonist at 5-HT2C receptors Agomelatine's acute efficacy in treating MDD was seen in three placebo-controlled studies, including a dose-ranging study with paroxetine as active comparator.

The meta-analysis of these trials showed a significant difference between agomelatine and placebo in the main efficacy analysis, the HAMD score (= $2.86 \ 0.56$; P<0.001) and in the CGI scale (= $0.47 \ 0.10$; P<0.001).

Furthermore, evidence of agomelatine's efficacy in severe depression was illustrated by the meta-analysis of the patient subgroup with a baseline HAMD 25. Analysis of pooled data demonstrated an increase in the magnitude of the agomelatine-placebo difference with increasing severity at baseline.

The antidepressant efficacy of agomelatine was also evaluated in direct comparison to venlafaxine in 2 trials. Agomelatine showed at least comparable efficacy to venlafaxine in depressed patients after 6 and 12 weeks of treatment.

Agomelatine did not show the typical side effects found with selective serotonin reuptake inhibitors (SSRIs) (ie, gastrointestinal disorders, weight gain, serotonergic syndrome, and insomnia).

Moreover, agomelatine was shown to lack discontinuation symptoms compared with placebo in a study showing significant discontinuation symptoms with paroxetine.

In conclusion, the experience with agomelatine across a wide range of clinical trials suggests that agomelatine offers an important alternative for the treatment of depression, combining efficacy, even in the most severely depressed patients, with a favourable side-effect profile.

SAT3.02

A new pharmacological step: The melatonergic approach

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A breakthrough has recently been made in antidepressant research with the development of agomelatine. Agomelatine has a distinct pharmacological profile compared with all other classes of clinically available antidepressants.

Agomelatine is a high-affinity agonist at both the melatonergic MT1 and MT2 receptor types, and, in addition, blocks 5-HT2C receptors. Agomelatine did not significantly bind to any other site studied. In accordance with this profile, agomelatine resynchronized circadian rhythms and elicited a dose-dependent elevation in extracellular levels of noradrenaline and dopamine in the frontal cortex of freely moving rats while exerting no effect upon serotonin levels. The antidepressant actions of agomelatine have been described in several validated animal models: learned helplessness, forced swim, chronic mild stress, mice with impaired glucocorticoid receptors, isolated aggressive mice, and the marble burying test, with antidepressant-like effects being shown in all behavioral paradigms examined. Based on these results, the nocturnal sleep pattern of psychosocially stressed male tree shrews (a valid animal model for depression) was investigated: agomelatine resynchronized disrupted circadian rhythms and antagonized the effect of stress on the total amount of rapid eye movement (REM) sleep and on the fragmented sleep pattern. In conclusion, the antidepressant efficacy of agomelatine may be due to its receptor profile, and it is hypothesized that melatonergic and 5-HT2C receptors may be acting in synergy, thus representing a novel approach to treating depression.

SAT3.03

How the internal clock interacts with mood and depression

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In all life forms, circadian rhythms are defined by a period of approximately 24 hours. The daily light/dark cycle governs rhythmic changes in behavior and physiological and mental functions, ie, in activity, core body temperature, hormones, sleep-wake cycle. All circadian rhythms are driven and controlled by the biological clock, which in mammals is located in the suprachiasmatic nucleus (SCN) of the anterior hypothalamus.

Disruption of circadian organization is a characteristic of a variety of affective disorders, especially major depression, and, circadian abnormalities may constitute a core component of the pathophysiology of depression and may also determine the treatment response.

Depressed patients have documented abnormalities in mood, body-temperature, neuroendocrine secretion, and, most importantly and disabling, in sleep (approximately 90% of patients complain about their sleep). The sleep alterations are mainly related to poor sleep quality and maintenance and to difficulties in maintaining alertness during the day. Polysomnographic recordings show disruption of sleep continuity with prolonged sleep latency, increased wake time during the night, increased early morning wake time, decreased slow-wave sleep, and disinhibition of REM sleep. Most antidepressants can influence the architecture of sleep: SSRIs, SNRIs, and some TCAs (clomipramine) have "alerting" effects whereas others, among them, mirtazapine or trazodone, are sleep promoting often also causing sedation and daytime sleepiness. An important clinical goal in the treatment of major depression would therefore include antidepressants that improve both mood and quality of sleep without impairing daytime alertness.

SAT3.04

Beyond efficacy on the core symptoms of depression: Sex and sleep benefits

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The outcome of depression can be affected after chronic use of antidepressants, because of the spectrum of side effects affecting compliance and quality of life. Among the most disabling side effects are sleep disturbances and sexual dysfunction.

Agomelatine, with its unique pharmacological profile acting as an agonist at melatonergic receptors and as an antagonist at 5-HT2C receptors, improves sleep and does not affect sexual functioning in major depressive disorder. In one study, agomelatine 25 mg, increased slow-wave sleep and normalized its distribution throughout the night (P<0.05) without altering REM sleep. In another study, agomelatine 25-50mg, compared with venlafaxine 75-150 mg, showed similar antidepressant efficacy and demonstrated significant sleep improvement (LSEQ questionnaire) as early as from the first week of treatment (P=0.007 for getting off to sleep and P=0.015 for quality of sleep). This improvement was maintained throughout the entire 6-week treatment period, with a parallel improvement in daytime alertness.

A comparison of sexual functioning in depressed patients treated with agomelatine or venlafaxine indicated that agomelatine 50 mg had a better sexual profile than venlafaxine XR 150 mg in remitted patients after 12 weeks of treatment on both orgasm and preorgasm measures; both treatments showed comparable antidepressant efficacy. To confirm the favourable effects of agomelatine on sexual functions, a study in healthy volunteers has been carried out and these results will be discussed.

In conclusion, agomelatine is a novel antidepressant that ameliorates disturbed sleep and leaves sexual functioning unaffected, thus improving both depressive symptoms and quality of life of depressed patients.

SAT4 - Satellite symposium: THE INTEGRATED MANAGEMENT OF LONG-TERM PSYCHIATRIC AND MEDICAL NEEDS IN PATIENTS WITH SEVERE MENTAL ILLNESS Sponsored by pfizer

SAT4.01

Impact of medical comorbidities on patients with severe mental illness

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Patients with schizophrenia and bipolar disorder carry a heavy burden of medical comorbidities. Patients with schizophrenia or bipolar disorder have a life expectancy that is 15 years less than that of the general population. This increased mortality is partly associated with factors inherent to the patients' psychopathology. For example, the risk of suicide is about 20 times higher than that of the general population. However, despite increased psychiatric mortality, cardiovascular disease is the primary cause of death in patients with schizophrenia. While some of this morbidity is the acknowledged result of long-term antipsychotic medication, not all can be explained by pharmacotherapy-for example, patient lifestyle choices may account for at least part of this elevated risk. Smoking, for example, is much more common among patients with schizophrenia than the general population. However, psychotic patients often have undetected general health problems despite a higher than average physician consultation rate, suggesting that there is inadequate monitoring and treatment of the physical health of individuals with mental health problems. This may reflect the fact that mental healthcare is separated from physical healthcare in many countries and access to primary healthcare is often limited for individuals with mental illness.

SAT4.02

Considerations in the treatment of severe mental illness: Differential profiles of antipsychotics

J.W. Newcomer. Departments of Psychiatry, Psychology and Medicine, Washington University School of Medicine, St. Louis, MO, USA Second-generation antipsychotic drugs (eg, olanzapine, quetiapine, risperidone, aripiprazole, and ziprasidone) have a reduced incidence of extrapyramidal side effects compared with first-generation neuroleptics, leading to increased use in psychiatric practice. However, some second-generation antipsychotic drugs can increase cardiometabolic risk by increasing risk for weight gain, dyslipidemia, and insulin resistance. Growing evidence, including baseline metabolic data from the CATIE study, indicates that patients with schizophrenia have an increased prevalence of metabolic syndrome (obesity, hypertension, hyperglycemia, dyslipidemia, and hyperglycemia). In CATIE Phase 1 and 2, treatment with different antipsychotic medications is associated with different effects on weight, plasma lipids and risk of hyperglycemia, ranging from clinically significant increases to decreases in metabolic risk. While mortality related to cardiovascular disease is elevated in this patient population, cardiovascular disease risk is under-monitored and undertreated. Current public health efforts aim to increase attention to this at-risk population. Long-term treatment strategies in persons with mental illness should aim to address psychiatric illness as well as key medical comorbidities.

SAT4.03

Toward the reintegration of psychiatry and medicine in patients with mental illness

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Treatment goals in schizophrenia and bipolar disorder are no longer simply the reduction of psychosis and manic or depressive episodes. Today's treatment goals encompass a broader improvement of quality of life and, as much as possible, the return of patients to premorbid levels of functioning. To achieve these wider-reaching goals, patient care must simultaneously address not only patients' psychiatric illness but also their medical problems. In addition to reducing mortality, there are good psychiatric reasons for addressing the physical well-being of patients: the presence of a comorbid physical illness worsens the prognosis of the mental disorder and vice versa. General medical monitoring should form as much a part of the routine management of patients with long-term mental illness as should psychiatric reviews, and any barriers between diagnosis and treatment in these patients should be examined. The care team needs to be expanded beyond the core psychiatric team, and patient access to primary medical care needs to be improved to ensure parity of medical treatment with the general population. As patient function improves, patients and their families can become more involved in self-management and feel empowered to affect their own outcomes.

SAT5 - Satellite symposium: DOPAMINE TRANSPORTER SPECT IN THE DIFFERENTIAL DIAGNOSIS OF DEMENTIA - A NEW CLINICAL TOOL Sponsored by GE Healthcare

SAT5.01

Dementia with Lewy bodies: A comparison of clinical diagnosis, DaTSCAN imaging and neuropathological diagnosis

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Background: Dementia with Lewy bodies (DLB) is a common form of dementia. The presence of Alzheimer's disease (AD) pathology modifies the clinical features of DLB, making it harder to distinguish DLB from AD clinically during life. Our aim was to determine, in a series of patients with dementia in whom autopsy confirmation of diagnosis is available, whether functional imaging of the nigrostriatal pathway improves the accuracy of diagnosis compared to diagnosis by means of clinical criteria alone.

Methods: A SPECT scan was carried out with a dopaminergic pre-synaptic ligand [123I]-2beta-carbometoxy-3beta- (4-iodo-phenyl)-N- (3-fluoropropyl) nortropane (FP-CIT) on a group of patients with a clinical diagnosis of DLB or other dementia. An abnormal scan was defined as one in which right and left posterior putamen binding, measured semi-quantitatively, was more than 2 standard deviations below the mean of the controls.

Results: Over a ten year period it has been possible to collect twenty patients who have been followed from the time of first assessment and time of scan through to death and subsequent detailed neuropathological autopsy. Eight patients fulfilled neuropathological diagnostic criteria for DLB. Nine patients had AD, mostly with coexisting cerebrovascular disease. Three patients had other diagnoses. The sensitivity of the FP-CIT scan for the diagnosis.

Conclusions: FP-CIT SPET scans substantially enhanced the accuracy of diagnosis of DLB by comparison with clinical criteria alone.

SAT5.02

Results of a multi-centre study of DaTSCAN in dementia with Lewy bodies

I. McKeith. United Kingdom

Clinically based diagnostic criteria for DLB have limited accuracy. The availability of a biomarker to assist with diagnosis would be a major advance. Severe nigro-striatal degeneration and dopamine loss occurs in DLB but not in most other dementia subtypes offering a potential system for a biological marker. In the PDT-301 study, 326 patients with dementia with clinical diagnoses of probable or possible DLB, or non-DLB dementia established by a Consensus panel, had a FP-CIT SPECT brain scan labelling the dopamine transporter (DAT) reuptake site in the striatum. Three readers, blinded to clinical diagnosis, classified the images as normal or abnormal by visual inspection. This study which was conducted across 40 European sites, confirms the high correlation between abnormal (low uptake) DAT activity measured using FP-CIT SPECT and a clinical diagnosis of probable DLB. The diagnostic accuracy is sufficiently high for this to be clinically useful in distinguishing DLB from AD.

SAT5.03

The impact of DATscan can have on dementia patients: Case studies

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Although good epidemiological data do not yet exist, Dementia with Lewy bodies (DLB) is increasingly recognized as one of the most common causes of dementia after Alzheimer's disease (AD). The identification of DLB has important implications in terms of prognosis and patient management. These patients frequently develop motor, psychiatric, and sleep-related disturbances in addition to the dementia syndrome, and information and re-assuring of patients and caregivers are important. In addition, drug treatment of patients with DLB is different from that of AD patients. DLB patients may respond better to cholinergic and dopaminergic agents, but are more likely to develop severe side-effects when treated with anti-psychotic agents, including the atypical ones.

However, diagnosing DLB may be difficult. Several studies have demonstrated a low diagnostic sensitivity compared to AD when using international consensus criteria for a clinical diagnosis of DLB. Thus diagnostic markers are needed to improve diagnostic accuracy. Although emerging data indicate that neuro-imaging techniques such as structural MRI and perfusion SPECT may differentiate AD and DLB at group level, there is too much overlap for these techniques to be useful in the diagnosis of individual patients. Accordingly, the finding that Dat scan can reliably distinguish patients with DLB from those with AD, even DLB patients without parkinsonism, can improve patient management. The most important situation is a patient fulfilling DLB criteria but without parkinsonism, where Dat scan ascertain involvement of the nigrostriatal system typical for DLB. Another potentially important situation is a patient with dementia and parkinsonis and psychosis treated with antipsychotic, where it is unclear whether parkinsonism is secondary to the antipsychotic drug treatment or part of the dementia syndrome. Cases illustrating these clinical dilemmas will be presented and discussed.

SAT6 - Satellite symposium: MENTAL AND PHYSICAL HEALTH ARE INTERCONNECTED: THE NEED FOR INTEGRATED HEALTHCARE Sponsored by Bristol-Myers-Squibb

SAT6.01

Schizophrenia and overweight/obesity: Pathophysiology and medical consequences

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Overweight/obesity is a growing concern throughout the general population. The prevalence of overweight and obesity in schizophrenia is high, compounding the burden of an already devastating illness. This is making overweight/obesity an important consideration if the physical health of patients is to be improved. Ultimately, overweight/obesity results from an imbalance between food intake and energy expenditure over several years. However, the pathophysiology of overweight/obesity is complex and involves interactions between environmental, behavioural and genetic factors. There is compelling evidence that patients with schizophrenia are more prone to weight increase than the general population. Although a number of social factors contribute to the increased risk of overweight/obesity and metabolic disturbances in patients with psychiatric disorders, there is also evidence that genetic factors may play a role. Additionally, many psychiatric medications are known to be associated with weight gain. It is thought that weight gain may be related to neurotransmitter-receptor affinity, which can have multiple effects on energy homeostasis. For example, histamine H1 receptor affinity has been shown to predict short-term weight gain with both typical and atypical antipsychotics.

Obesity, especially abdominal obesity, is associated with a number of adverse health consequences. These include an increased risk of glucose intolerance, insulin resistance, Type 2 diabetes, dyslipidaemia, hypertension and cardiovascular disease. In addition, the presence of metabolic syndrome, a cluster of metabolic disturbances, significantly increases the risk of cardiovascular morbidity and mortality. It can therefore be seen that there is an urgent need to start identifying schizophrenia patients who are at risk to help improve long-term health outcomes.

SAT6.02

Impact of antipsychotic treatment on physical health

M. De Hert. UC St. Jozef, Kortenberg, Belgium

Metabolic abnormalities have consistently been identified as a part of schizophrenic illness, but with the introduction of second-generation antipsychotics and their possible association with metabolic abnormalities, the interest in this topic has been renewed. Many studies have now provided convincing evidence for a high risk of obesity, diabetes and other glucose abnormalities, the metabolic syndrome, and mortality due to elevated cardiovascular risk in patients with schizophrenia. These metabolic abnormalities are of major clinical concern, not only because of their direct, somatic effects on morbidity and mortality, but also because of their association with psychiatric outcome, such as a higher prevalence of psychotic and depressive symptoms, a lower functional outcome, a worse perceived physical health and lower adherence to medication. The reasons that underlie the high prevalence of these metabolic abnormalities are much debated, especially when considering the possible role of second-generation, 'atypical' antipsychotics in the occurrence of these abnormalities. Many studies have suggested a role of (certain) atypical antipsychotics in the occurrence of metabolic abnormalities; case reports, cross-sectional or retrospective studies and prospective studies. Different consensus groups have proposed guidelines for screening, monitoring and management of metabolic abnormalities for people treated with antipsychotic agents.

SAT6.03

Psychosocial consequences of physical health impairment in schizophrenia

P. Thomas. France

As well as the obvious medical consequences associated with physical health problems in individuals with schizophrenia, physical health problems lead to a number of psychosocial consequences - further contributing to the existing burden of schizophrenia itself. Weight gain is one that may have repercussions on both psychosocial and economic parameters. Weight gain can seriously impair quality of life through decreased functioning, social stigmatization, discrimination, and potential financial consequences. Obesity and being overweight appear to have the same impact on the self-esteem and well-being in people with schizophrenia as those in the general population, and it has been shown that patients who experienced recent weight gain had lower psychosocial adjustment and self-esteem compared with patients without weight change. However, patients with schizophrenia may be less capable of managing their weight via exercise and dietary interventions compared with the general population, and

may, therefore, be more prone to non-compliance with a medication that induces weight gain. This may lead to exacerbation of the mental illness. Patients with schizophrenia may also experience other physical health problems owing to the tolerability profile of antipsychotic therapies, such as sexual dysfunction or extrapyramidal symptoms; such adverse effects also have the potential to impact on patient well-being, quality of life, functioning and compliance with therapy. Patients' perceptions and experience of antipsychotic treatments are also important as these can further impact on overall patient wellbeing. It is hoped that by increasing awareness of the psychosocial consequences of physical health impairment, we can move towards reducing the burden that patients face.