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# HKCPMA Newsletter

Official newsletter of the Hong Kong Community Psychological Medicine Association



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## Message from the Chairman

On Wednesday, 11 November 2009, Dr Aaron Lee, Dr Charles Yan, Dr Eunice Wong and Dr Leo Chan, represented the Hong Kong Community Psychological Medicine Association (HKCPMA) in a meeting with Mr Ian Wylie, the Senior Manager of Service Planning & Development of Hospital Authority (HA), at HA headquarter, Argyle Street, Kowloon.

Mr Wylie explained that service planning by the HA in the upcoming three-years, will focus on mental health of the general public. At present, there is a huge burden of mental disorders on the HA. In the United Kingdom, the majority of mental health care is managed by primary care professionals. However, in Hong Kong the role of general physicians is often overlooked. Currently, he is trying to find ways to improve and develop the mental health service in Hong Kong. Accordingly, his department is proposing plans to change the mental health system.

A big meeting is to be held at the end of November, 2009 and editorial work is currently underway to prepare a draft mental health plan for submission. The formulated plan is proposed to be finalized by January 2010, for publication in April 2010. The Health Secretary is responsible for developing health policy for Hong Kong, while the HA is responsible for providing services.

Recently, I made a presentation to introduce the annual course in community psychological medicine, organized by The University of Hong Kong (HKU). The graduates of Postgraduate Diploma in Community Psychological Medicine (PGDCPM) of HKU were confident in providing front-line mental health care to their patients. There is a need for these graduates to be enlisted in the Family Medicine Unit (FMU) website, so that patients can choose to seek consultation from general practitioners, psychiatrists, or doctors in the HA. We assured Mr Wylie that graduates of PGDCPM have an "add-on" value to the existing mental health system at a community level in Hong Kong.

How can we meet the challenges of mental health problems?

Here are a few of my suggestions for a better mental health care in Hong Kong:

- Implementing health promotion interventions by the HA, to encourage appropriate help-seeking behaviour. For example, coupons may be offered to patients with mental health problems on first consultation to a general practitioner or psychiatrist.
- Organizing a proactive psychiatric collaborative community network. This would involve hospital/community psychiatrists to work with and provide leadership to a multidisciplinary team of general practitioners, social workers, community psychiatric nurses.
- Reviewing the clinical practice of general practitioners and psychiatrists on a regular basis.
- Establishing an open dialogue between general practitioners and psychiatrists, to expand the community mental health team for improved patient care.

I would also like to take this opportunity to thank you for the continued support for our Association. We hope you continue to enjoy reading our newsletter and we look forward to seeing you in forthcoming HKCMPA activities!

Dr Aaron Lee Fook Kay  
Chairman, HKCMPA



# The Annual General Meeting of Hong Kong Community Psychological Medicine Association 2009

A joint HKCMPA luncheon symposium and Annual General Meeting 2009 was held on Friday, 30 October 2009, at the Mira Hotel in Tsim Sha Tsui. Guests from Hong Kong Medical Association (HKMA) graciously came to support the meeting. Professor David Dunner, Professor Emeritus of the Department of Psychiatry, University of Washington, delivered an insightful presentation on, "The power of SNRI in achieving and sustaining true remission". This was followed by the Annual General Meeting, in which new council members were elected. Dr Aaron Lee was re-elected as the chairman for one more year. HKCMPA would like to take the opportunity to thank Pfizer Corporation Hong Kong Limited for their generous sponsorship of the event.



Photograph taken at the Annual General Meeting of HKCPMA 2009 on Saturday, 10 October 2009 at the Mira Hotel, Tsim Sha Tsui, Kowloon.

From left to right: Dr Chan Yee Shing, Alvin (Vice-Chairman of HKMA); Dr Lee Fook Kay, Aaron (Chairman of HKCPMA); Dr Choi Kin, Gabriel (Immediate-Past President of HKMA); Miss Yvonne Yan (representing formerly Wyeth, now Pfizer).

# Treatment of Major Depressive Disorder Future Prospects

## Misdiagnosis common in MDD

Depression is a serious medical condition that is common and prevalent across many ethnic groups and socio-economic classes.<sup>1</sup> Major depressive disorder (MDD) is one of the most frequently seen psychiatric illnesses in primary care setting.<sup>2</sup> Often under-recognized and misdiagnosed,<sup>3</sup> a diagnosis of MDD relies on evidence gathered from psychological and physical evaluation. The disorder is characterized by a combination of symptoms such as sadness, loss of interest, a decrease in energy, sleep and appetite disruptions; along with painful and non-painful somatic symptoms.<sup>3</sup> Somatic symptoms often mask the diagnosis of depression, with physicians attributing unexplained somatic causes to other factors. It has been reported that primary care physicians misdiagnose more than 78% of patients with MDD who present with somatic symptoms.<sup>3</sup>

**“Primary care physicians misdiagnose more than 78% of patients with MDD who present with somatic symptoms.”**

## Neurobiology of depression

MDD is an illness attributed to the dysregulation of certain neuronal circuits in several areas in the brain involved in the regulation of mood and stress response, particularly the prefrontal and limbic structures.<sup>4</sup> Low levels of dopamine, serotonin and noradrenaline at critical synapses is associated with various symptoms of MDD.<sup>4-6</sup>

The role of serotonin and noradrenaline in the pathophysiology of MDD is more related to their role in modulation of, and being modulated by, other neurobiological circuits that together mediate the symptoms associated with MDD.<sup>6,7</sup> Evidence shows that increasing circulating levels of serotonin, noradrenaline or both can reduce or eliminate MDD symptoms.<sup>4,6,8</sup> Serotonin and noradrenaline both modulate response to painful stimuli and mood in the brain.<sup>9</sup>

## Treatment options: Role of SNRI therapy in MDD recovery

There are various pharmacotherapies available for MDD (Table).<sup>1,10</sup> However, according to the National Institute of Mental Health's Sequenced Treatment Alternatives to Relieve Depression (STAR\*D) trial, 70% of patients do not achieve remission after first-line treatment with a selective serotonin reuptake inhibitor (SSRI).<sup>11</sup> Furthermore, MDD patients treated with antidepressants often experience residual symptoms that prevent them from attaining complete remission; over 73% of patients have at least one residual symptom after 2 months of treatment with fluoxetine.<sup>6,12</sup> This implies that not all circuits are successfully targeted by treatment.

Serotonin and noradrenaline reuptake inhibitors (SNRIs) inhibit the presynaptic reuptake of both serotonin and noradrenaline, and produce higher rates of response and remission from MDD than SSRIs.<sup>4,8,13-15</sup> Studies show that SNRIs are superior to SSRIs in

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improving depression.<sup>8,14,15</sup> By targeting both serotonergic and noradrenergic systems, an SNRI such as venlafaxine extended-release (XR), improves not only the core features of MDD, but also somatic symptoms.<sup>4</sup>

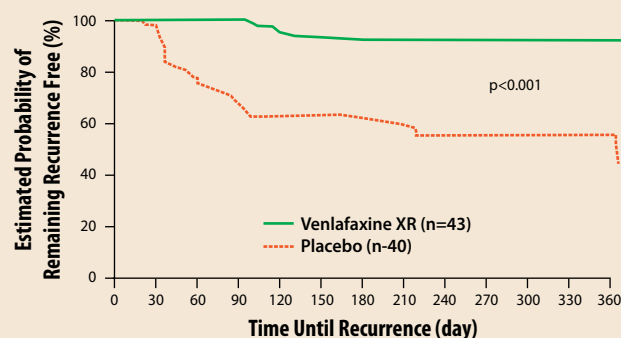
Patients with chronic pain and MDD can have many symptoms also found in generalized anxiety disorder (GAD) including, sleep disturbance.<sup>16</sup> Data shows that venlafaxine XR improves somatic symptoms including, cardiovascular, insomnia, respiratory, gastrointestinal and autonomic symptoms, over 8 week treatment in GAD patients.<sup>17</sup> Symptoms were assessed using the 14-item Hamilton Scale for Anxiety (HAM-A).

**“By targeting both serotonin and noradrenaline systems, a SNRI such as venlafaxine extended-release (XR), improves not only the core features of MDD, but also somatic symptoms.”**

## Efficacy of a dual-acting treatment for MDD

Venlafaxine XR is a bicyclic antidepressant that has been shown to be effective and well tolerated in the treatment of MDD.<sup>18</sup> The prevention of recurrent episodes with venlafaxine for two years (PREVENT) study, a 2-year combined maintenance phase study, demonstrated that venlafaxine XR was associated with significantly longer time to recurrence of MDD than placebo (Figure 1).<sup>19</sup> At the second 12-month maintenance phase, the rate of remission was significantly higher in the venlafaxine XR group (93%) than the

**Figure 1. Time to recurrence, primary definitions (maintenance phase B of the PREVENT study)<sup>19,a</sup>**



<sup>a</sup> Recurrence defined as HAM-D17 score >12 and reduction in HAM-D17 score from acute phase baseline that was not more than 50% at 2 consecutive visits or at the last valid visit prior to discontinuation. XR=Extended release, HAM-D17=17-item Hamilton Rating Scale for Depression, PREVENT=Prevention of Recurrent Episodes of Depression with Venlafaxine for Two Years

placebo group (63%; $p=0.002$ ). The incidence of adverse events in recipients of venlafaxine XR is similar to well established SSRIs.<sup>18</sup>

In addition, data from a pooled analysis of five placebo-controlled studies demonstrate that venlafaxine XR produced higher rates of sustained remission than SSRI (fluoxetine/paroxetine). Remission was also achieved earlier with venlafaxine XR.<sup>20</sup>

The dual actions of venlafaxine XR benefit patients who remain severely depressed and unresponsive to SSRI therapy.<sup>11,21</sup> In one study of patients unresponsive to SSRI treatment, 6–8 weeks' treatment with venlafaxine formulations resulted in a responder (25-item Hamilton Depression [HAM-D25] or 21-item Patient Global Improvement [PGI-21] criteria) rate of 94.2% and remission rate of 87%.<sup>21</sup>

Furthermore, in placebo-controlled clinical trials, duloxetine has been shown to be superior to placebo in improving core depressive symptoms and painful somatic symptoms. Data from the analysis of pooled data showed that duloxetine reduced pain severity by 22–41% compared with a 5–18% reduction in placebo.<sup>22</sup>

## Conclusion

MDD is an illness with significant neurobiological aspects mediated by different malfunctioning neuronal circuits. A neurobiological understanding of somatic symptoms in MDD may guide more effective treatment strategies. SNRI therapy is proven to be effective in controlling both psychic and painful or non-painful symptoms of MDD. An SNRI, like venlafaxine XR, could be one of the best treatment options for patients with MDD.

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Table. Pharmacotherapy for major depressive disorder <sup>1,10</sup>	
Drug class	Mechanism of action
<b>SSRIs</b> Escitalopram Sertraline Fluoxetine	Inhibits the CNS uptake of serotonin, increasing the amount of neurotransmitter at the synapse
<b>SNRIs</b> Venlafaxine XR Duloxetine Desvenlafaxine (approved by US FDA and available in United States)	Inhibits the noradrenaline and serotonin reuptake as well as dopamine to a lesser degree
<b>NDRIs</b> Bupropion	Inhibits the noradrenaline, serotonin, and dopamine reuptake systems, increasing the amount of neurotransmitter at the synapse
<b>TCA's</b> Imipramine	Inhibits the reuptake of noradrenaline and serotonin in the CNS, increasing the available amount of neurotransmitter in the brain
<b>MAOIs</b> Phenelzine	Increases the levels of noradrenaline, dopamine, and serotonin through irreversible inhibition of monoamine oxidase

SSRI=Selective serotonin reuptake inhibitor; SNRI=Serotonin and noradrenaline reuptake inhibitors; NDRI=noradrenaline and dopamine reuptake inhibitor; TCA=Tricyclic antidepressants; MAOIs=Monoamine oxidase inhibitors; XR=Extended release; US FDA=United States Food and Drugs Administration.

# Understanding and treating somatic symptoms in depression: An expert interview with Dr Dominic Tak Shing Lee

Dr Lee is Specialist in Psychiatry and Adjunct Professor of the Centre of Research and Promotion of Women's Health at the Chinese University of Hong Kong.

Like any other mood disorder, major depressive disorder (MDD) is diagnosed according to patient symptoms, following a psychological examination. There are no diagnostic laboratory tests available for the diagnosis of this illness. Currently, the Diagnostic and Statistical Manual of Mental Disorders — Fourth Edition (DSM-IV) and the International Classification of Diseases (ICD-10) are used for the clinical diagnosis of the disorder.

Dr Lee points out that in the DSM-IV classification, of the nine diagnostic criteria for MDD, only three are physical in nature — fatigue, insomnia and appetite change. It is important that general practitioners (GPs) do not focus solely on patients' psychological troubles but also what troubles them somatically.

In Dr Lee's view, GPs' clinical experience is more important than adhering to the DSM-IV or ICD-10 diagnostic criteria. Many GPs are aware that the majority of somatic symptoms presented are related to the mind. He added, "Patients' somatic symptoms are the centre of their depression experience." Therefore, patients expect treatment that will help alleviate all aspects of their symptoms, not just the psychological ones.

In clinical practice, patients present not only psychological symptoms, but also somatic symptoms. These bodily symptoms range from dizziness, tinnitus, shortness of breath, back pain, irritable bowel syndrome, and weakness of the limb. "Somatic symptoms related to the heart are very common because the Chinese culture believes that the mind/spirit is located here," said Dr Lee. Moreover, there is a close inter-relationship between somatic and mood symptoms. The greater the severity of the mood symptom, the more likely the patient will report of somatic symptoms.

Patients will often seek a physical explanation for somatic symptoms from a GP or specialists. It is often difficult for patients to understand that these bodily symptoms may be secondary to MDD, and there is a tendency to attribute them to non-psychiatric causes. For this reason, depression may be under-diagnosed. However, Dr Lee highlighted that, "somatic symptoms are valid indicators of depression and it doesn't make depression any less real".

Different approaches are adopted for symptomatic or curative treatment for MDD. Selective serotonin reuptake inhibitors (SSRIs) and selective noradrenaline reuptake inhibitors (SNRIs) are among the first-line agents for curative treatment of MDD. Moreover, Dr



Lee highlighted that somatic symptom relief is of central importance to patients, which influences treatment strategy. In patients presenting with somatic symptoms, a benzodiazepine should be given SSRIs in conjunction with/or SNRIs.

## Case study: SNRI effectively relieves shortness of breath related to depression

A 48-year-old, married woman working in middle management level of sales and marketing, started to become increasingly stressed in the past year, due to the economic downturn. She began to experience sudden attacks of shortness of breath, dizziness and a gripping sensation in the heart. She was concerned and went to the hospital for a full body health check-up, but no abnormalities were found. The doctor who carried out the check-up suggested it might be anxiety-related.

Three days later, she experienced a panic attack while commuting on the MTR. After 15 minutes of rest, she felt better. The next day, she went to see a GP. The GP discovered that her somatic symptoms were accompanied with insomnia (waking at 5am), impaired concentration, forgetfulness, social isolation and loss of interest in activities. She required the use of tranquilizers to decrease anxiety levels when delivering presentations at work.

The GP diagnosed her with depression. She was prescribed with a SNRI, benzodiazepine and sleeping pills. After one week of treatment, she showed good response, with 80% relief of somatic symptoms and a return to a satisfactory sleeping pattern. A further three weeks later, her interests in daily activities increased and she had higher productive levels of functioning. By week 11, she was in full remission and was able to return to work. The dose of benzodiazepine was reduced from week six onwards. All treatment, with the exception of SNRI, was discontinued by week 11. SNRI treatment was continued for a further six months. She was educated on the purpose of the continued treatment of SNRI, to minimize the possibility of a relapse. After the six months, the SNRI treatment was gradually tapered off over a two month period. She has since been free from anxiety attacks, depressive and above all somatic symptoms.