MAJOR DEPRESSIVE DISORDER

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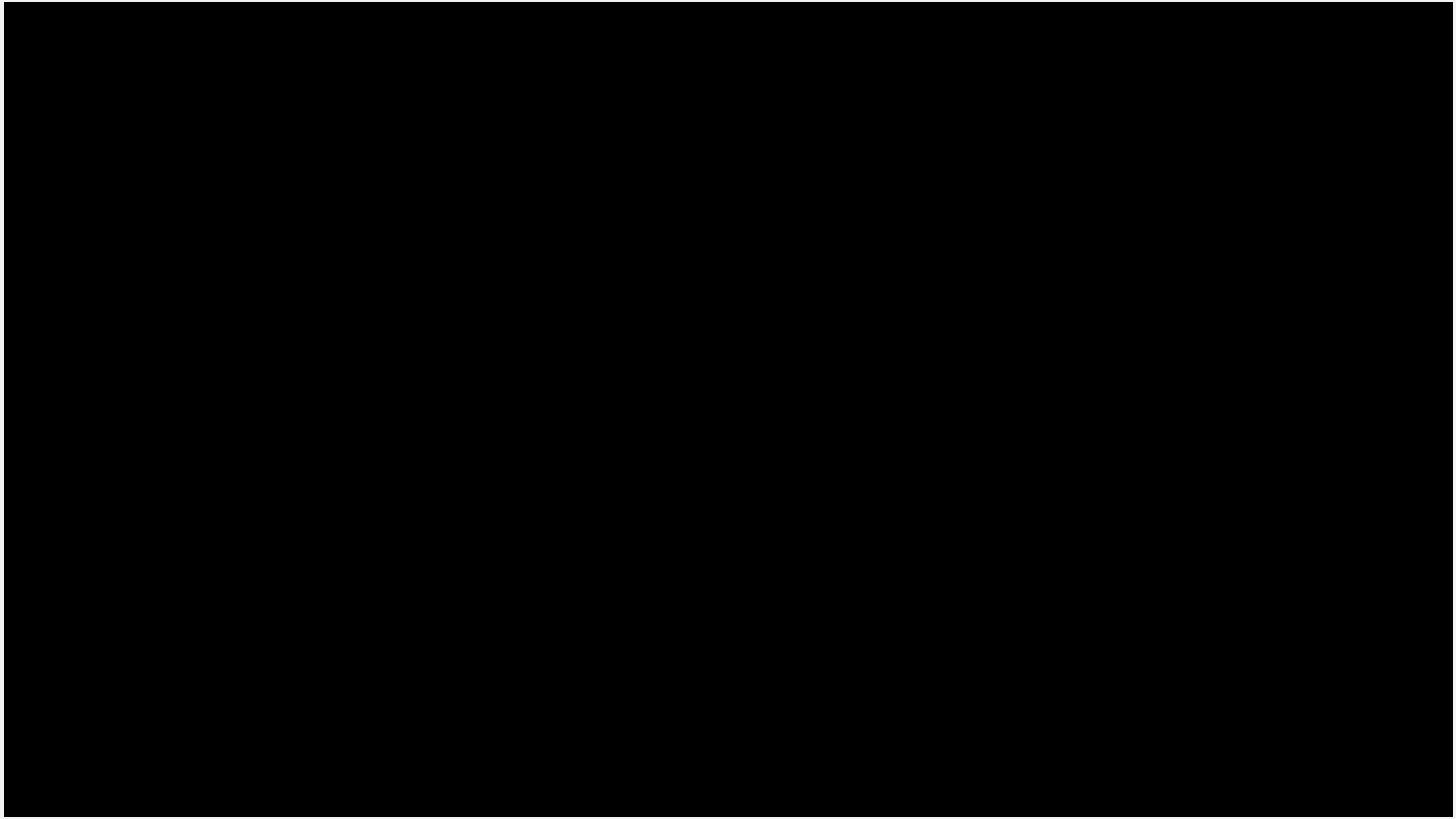
Clinical Psychology- PSX_002

Dr. Pavel Humpolíček- Autumn 2016



I FELT A FUNERAL IN MY BRAIN

EMILY DICKINSON

I felt a Funeral, in my Brain,

And Mourners to and fro

Kept treading - treading - till it seemed

That Sense was breaking through -

And when they all were seated,

A Service, like a Drum -

Kept beating - beating - till I thought

My mind was going numb -

And then I heard them lift a Box

And creak across my Soul

With those same Boots of Lead, again,

Then Space - began to toll,

As all the Heavens were a Bell,

And Being, but an Ear,

And I, and Silence, some strange Race,

Wrecked, solitary, here -

And then a Plank in Reason, broke,

And I dropped down, and down -

And hit a World, at every plunge,

And Finished knowing - then -

MAJOR DEPRESSIVE DISORDER: IN HISTORY

ANCIENT TIMES → EMIL KRAEPLIN

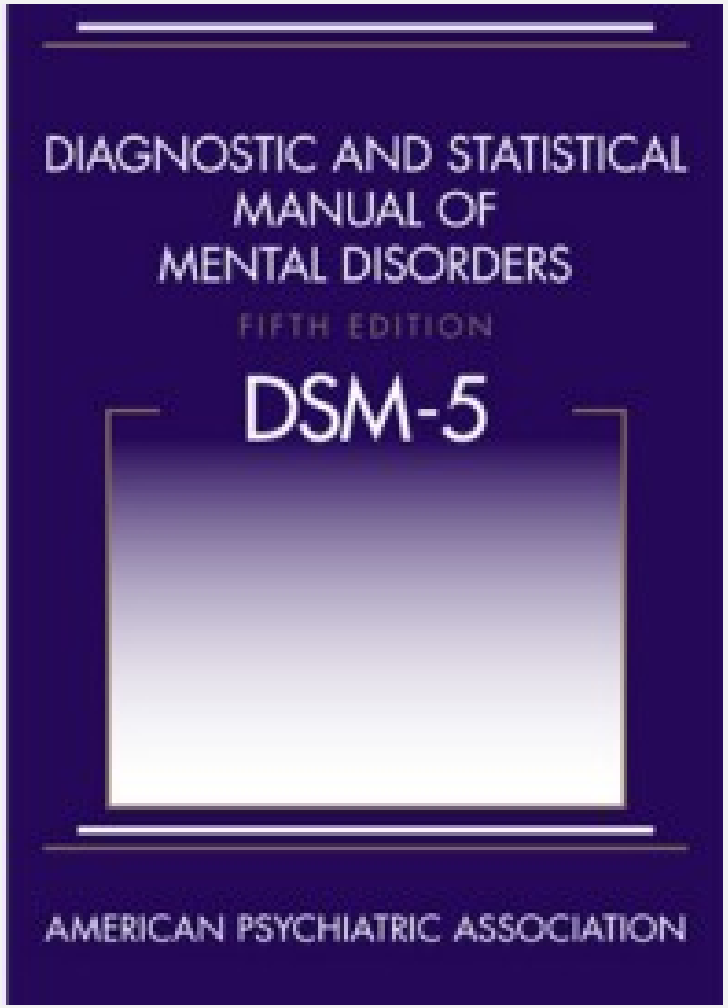
EMIL KRAEPLIN → SIGMUND FREUD

SIGMUND FREUD → THE LATE 19TH AND EARLY 20TH CENTURY

THE LATE 19TH AND EARLY 20TH CENTURY → DSM-I (1952) AND DSM-II (1968)

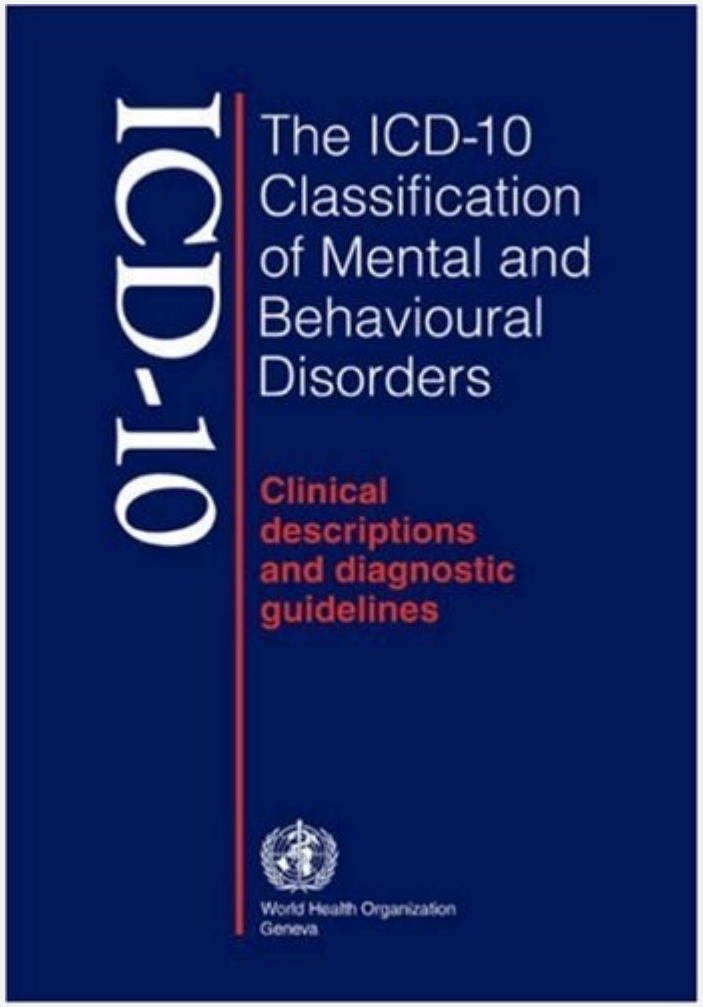
DSM-I (1952) AND DSM-II (1968) → 1980 – DSM-III

DIAGNOSIS



MAINLY USED IN USA

MAINLY USED IN EUROPE



DSM-V

CLUSTER A

Five (or more) of the following symptoms have been present consistently for more than two consecutive weeks, and represent a change in previous functioning:

- Depressed mood, indicated either by subjective report, or from the observations reported by others. In children and adolescents, this may also present itself as irritability.
- Decreased interest or pleasure in most or all activities.
- Significant weight loss or weight gain (a change of ± 5% over the period of a month). In children this may present itself as a failure to gain weight, rather than a change in mass.
- Change in sleep patterns, either insomnia or hypersomnia.
- Psychomotor agitation or retardation, observable by others.
- Fatigue or loss of energy.
- Feelings of worthlessness or excessive or inappropriate grief, which may be delusional.
- Diminished ability to think or concentrate, and/or more indecisiveness.

CLUSTER B

The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

CLUSTER C

These symptoms are not attributable to the physiological or psychological effects of a substance or other medical condition.

CLUSTER D

These symptoms must not be able to be better explained with a diagnosis of another disorder; particularly schizophrenic or schizophreniform disorders, delusional disorders or other psychotic disorders.

CLUSTER E

There has never been a manic or hypomanic episode that is not attributable to substance use.

ICD-10

The ICD diagnostic criteria for Major Depressive Disorder are similar in many ways to the criteria outlined in the DSM-V. There are a few variations, however:

The ICD, unlike the DSM, details further what constitutes MDD interrupting or changing a patients sleeping pattern. They are listed under 'somatic' symptoms, also known as 'melancholic' or 'endogenomorphic' symptoms. In addition to the symptoms listed in the DSM-V, the ICD states that symptoms of sleep change may also include waking in the morning 2 hours or more before the usual time.

On a similar note, the ICD also states that depression may be worse in the morning.

The 'somatic' symptoms a patient may experience also include a notable decrease in libido.

SUBTYPES OF MDD

Both the DSM and the ICD include further specifications for the subtypes or additional symptoms a patient may experience or exhibit. In the DSM, a practitioner may specify MDD existing alongside:

- Anxious Distress
- Mixed Features
- Melancholic Features
- Atypical Features
- Mood-Congruent Psychotic Features
- Mood-Incongruent Psychotic Features
- Catatonia
- Peri-Partum Onset
- Seasonal Pattern (Recurrent Only)

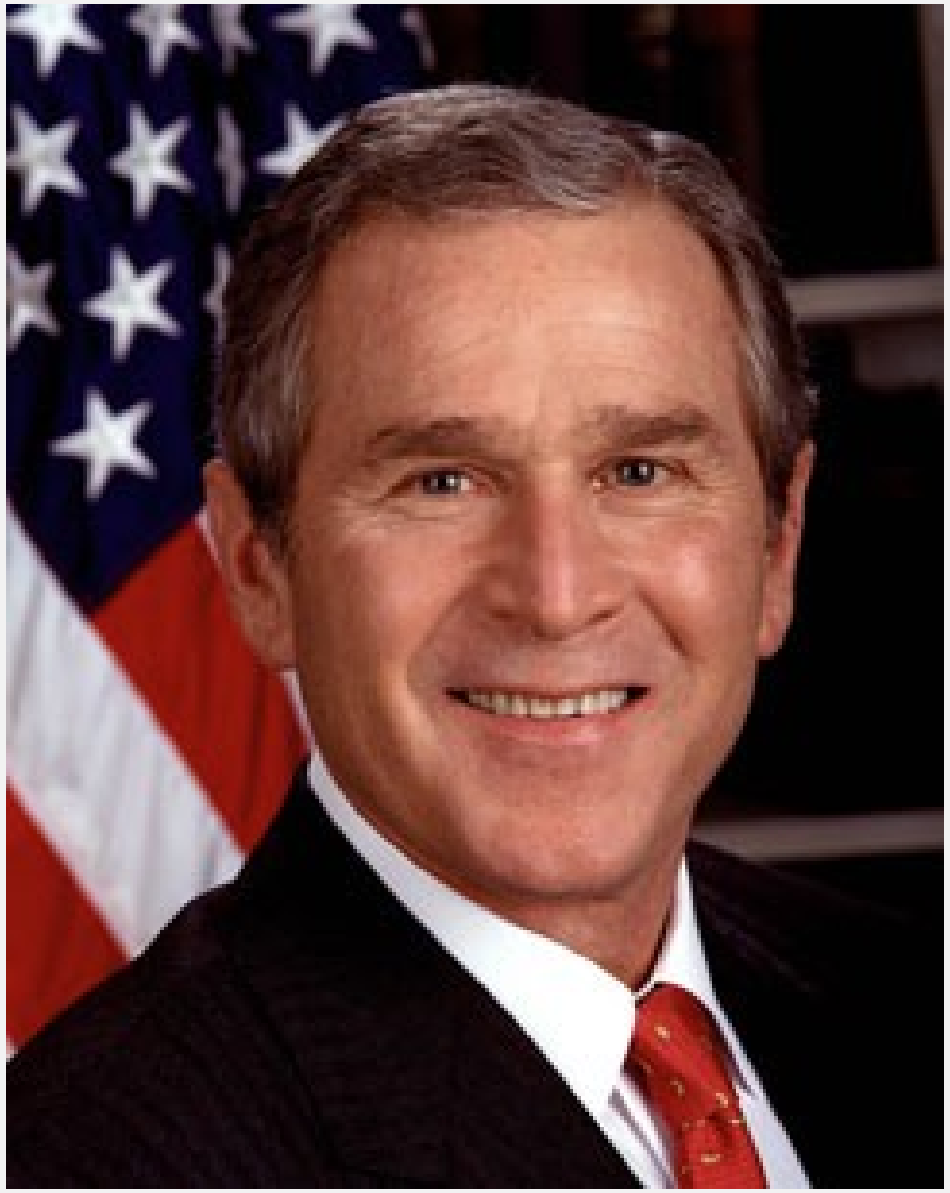
The diagnostic criteria of MDD in the ICD-10 include the above subtypes, but also include the following subtypes (MDD with):

- Recurrent Depressive Reactions
- Endogenous Depression
- Reactive Depression
- Vital Depression

MAJOR DEPRESSIVE DISORDER: IN SOCIETY

Stigma is one of the many barriers that sufferers must overcome when pursuing treatment, and is one of the primary reasons that treatment is not sought out (Corrigan, 2004). If treatment is begun, the impact of social stigma can result in a discontinuation in treatment that is of extreme importance, given the effect that depression can exert on a patient's life (Sirey et al., 2001).

Blumner and Marcus (2009), in a 10 year follow to a study completed in 1996, found that there was a shift in the beliefs about what caused depression, with 88% of participants surveyed believing that clinical depression (also known as Major Depressive Disorder) was caused by a biological imbalance, and 60% of participants prioritising a biological focus for treatments, such as medication.



RISK FACTORS

STRESS

- Andrews and Wilding (2004) in a study of British university students found that the impact of significant financial and other stressors led to 9% of a previously symptom free sample developing depressive symptoms at a clinically significant level.
- Wong, Cheung, Chan, Ma and Tang (2009)- in a sample of over 7000 students attending a Hong Kong university, 21% were found to exhibit depressive symptoms of above moderate severity, and 27% reported significant levels of stress, and after investigation, it was found that there were high levels of psychological co-morbidity for these two

- Lee and Kim (2006) studied over 400 nurses, and found that perceived stress was a predictor of depressive symptoms, with perceived stress and depression having a positive correlation.
- Ayerst (1999) found too that stress and depression have a positive correlation.

- Webster-Stratton and Hammond (1988) found that depressed mothers were significantly more likely to have suffered a significant stressor prior to birth, such as childhood or spousal abuse, than mothers who were not depressed.

BIRTH

- A meta-analysis of studies detailing the prevalence of women affected by peri-partum depression estimated that worldwide approximately 13% of women will experience this disorder (O'hara and Swain, 1996).

- Gulseren et al. (2006) found that in a sample of 125 pregnant Turkish women, the prevalence of depression was highest during the 36th and 38th week of pregnancy, with 21.6% of women reporting depressive symptoms. This level fell to 9.6% at 20-26 weeks postpartum.
- . Dubey, Gupta, Bhasin, Muthal and Arora (2011) found a similar prevalence rate, of 6%, for women in India.
- Cooper et al. (1999) found that the rates for this disorder were far higher in developing countries, with a study in rural South Africa finding a prevalence rate of 34.7%.

PRE-EXISTING PHYSICAL ILLNESS

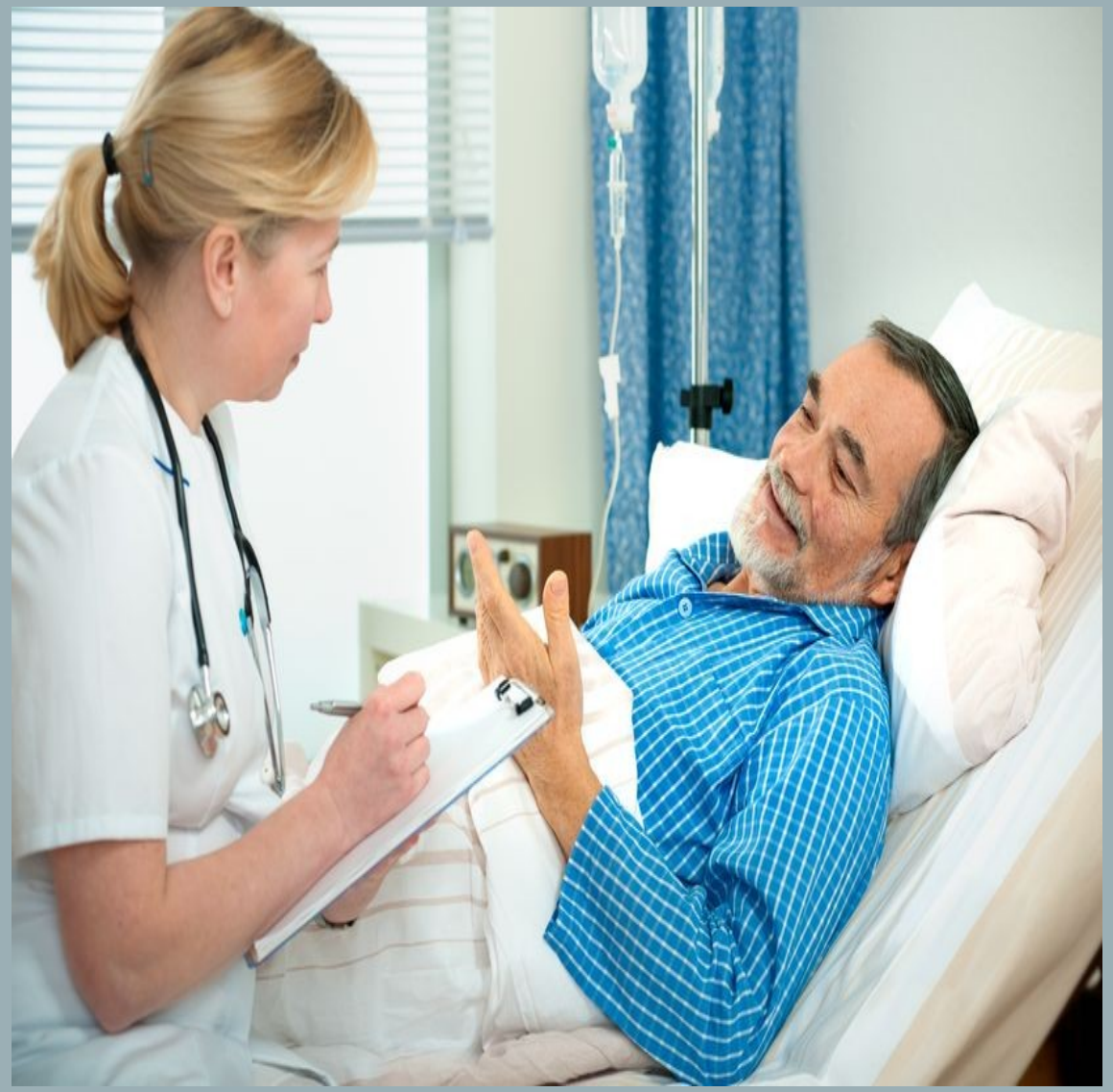
MDD accounts for approximately 50% of the psychopathology of medically ill patients (Creed and Dickens, 2006), making physical illness, particularly those that are debilitating or terminal, a significant risk factor for the development of MDD.

DIABETES

Ali, Stone, Peters, Davies and Khunti (2006)- prevalence rates of comorbid depression were found to affect an average of 17.6% of patients diagnosed with diabetes, higher than participants without, and females were more likely to suffer from depression than males (23.8% vs. 12.8% respectively), in a meta-analysis that encompassed over 51,000 participants.

A meta-analysis searching for prevalence rates of diabetes and MDD found that diabetics were twice as likely to develop depression than their non-diabetic control groups, and that diabetic women were more likely to develop depression than diabetic men, with prevalence rates of 28% and 18% respectively (Anderson, Freedland, Clouse and Lustman, 2001).

The relationship between diabetes and depression is a reciprocal one, as not only have patients suffering from diabetes been found to be more likely to develop depression than participants who do not, but patients suffering from depression early in life are also more likely to be diagnosed with diabetes later on (Lloyd et al., 2011).



HEART DISEASE

A patient having heart disease with co-morbid depression makes it less likely that they will engage in treatments, both in hospital but later in rehabilitation, and therefore, alarmingly, increases the morbidity rates for those patients who are unfortunate enough to suffer from both conditions (Shapiro, Lidagoster and Glassman, 1997; Roose and Spatz, 1998; Nemeroff, Musselman and Evans, 1998; Jiang, Krishnan and O'Connor, 2002).

Depression has been found to be co-morbid with other serious disorders, such as cancer and stroke patients (Kang et al., 2015), and has been shown to increase functional impairments, lower quality of life, and increase likelihood of mortality for these patients.

GENETICS

- Levinson (2006) identified that cases of patients with an early onset of depressive symptoms, and those who have a high rate of reoccurrence, or 'relapse' in their symptoms, present the highest risk to their future offspring.
- Feder, Nestler and Charney (2009) found that genetic mechanisms can play a role in the way a person responds to stress, and the resilience of their physical and mental health in response to external stressors.

- Figueiredo et al. (2015)- in a systematic review of women experiencing depressive symptoms post-partum, that genetic influences left women more open to the development of MDD, and that these genetic differences also had a role in the time period in which MDD developed; the late stages of pregnancy and the early months' post-partum were identified as the most vulnerable period for genetically vulnerable women.

- Lerman et al. (1998)- a genetic difference in the dopamine receptors in depressed patients brains also made them more likely to self-medicate with smoking that depressed patients who did not have this genetic difference.

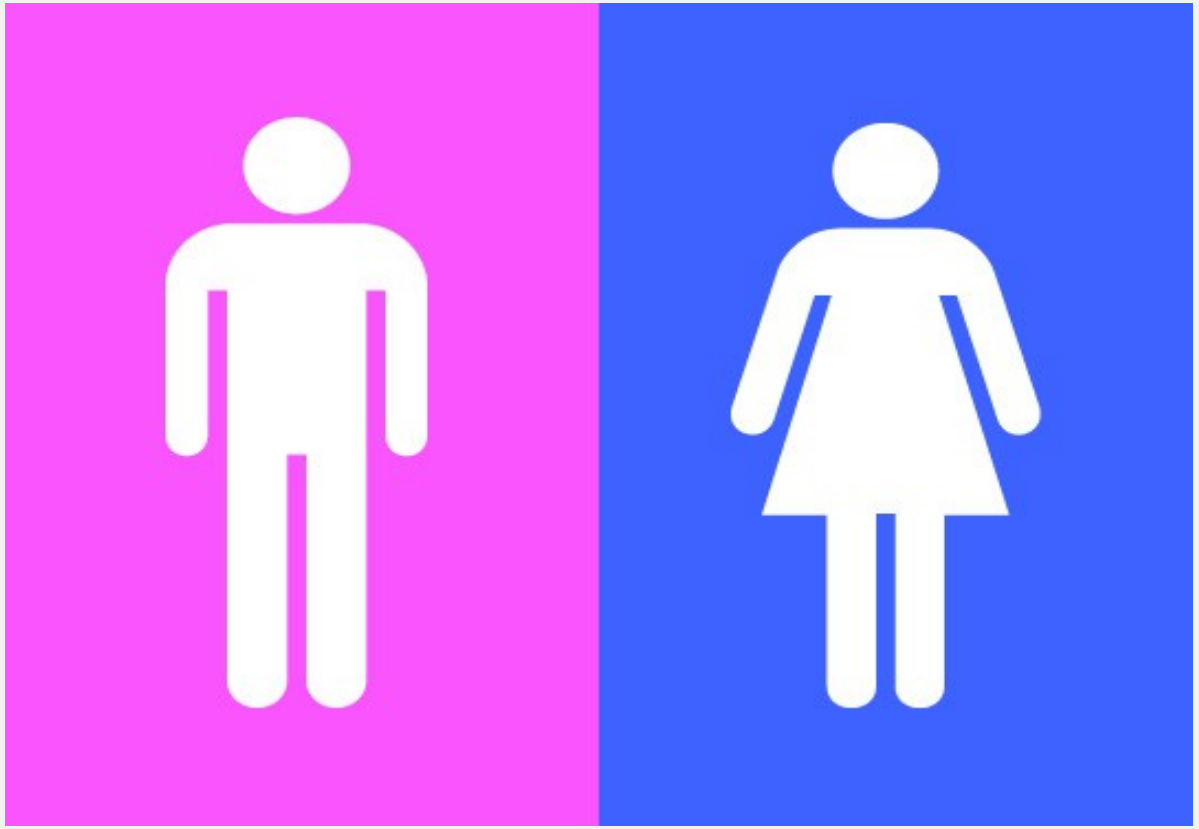
- Kato (2007) in a meta-analysis of all molecular genetic papers published on MDD found that the results published regarding the genetic influence in a patient's response to stress was unsubstantiated for some genetic codes that had previously been found to be influential. It was, however, found that a patient's genetic makeup could alter how they responded to medication

- McGuffin and Katz (1989) found, that the genetic risk is solely a risk for the development of endogenous depression, and that other forms of depression are much more heavily influenced by the familial environment.

GENDER

- There is little to no gender difference in the development of MDD is pre-adolescent children, but after the age of 15, pubescent girls and women become twice as likely to develop MDD than boys and men (Nolen-
- Hankin et al (1998)- 10-year longitudinal study on gender differences in the development of MDD, found that there was a 'critical period' for the development of MDD, during which time the gender differences became apparent. There were small gender differences identified between 13 and 15 years of age, but this sensitive time period was identified as the years between 15 and 18 years of age, when a significant gender difference in the development of MDD became most apparent.
- Ustün (2000)- found that depression is 1.5 to 3 times more likely to be diagnosed in women than men.

- Nolen-Hoeksema (2001) investigated this gender difference, and found that it was caused by the gender differences in response to stress and stressful events.



CO-MORBIDITY

ADHD

PTSD

GENERALISED ANXIETY DISORDER

OCD

Other disorders that can present as having MDD as a co-morbid disorder include Schizophrenia and Schizoaffective disorder, eating disorders such as Anorexia Nervosa and Bulimia, personality disorders such as Borderline Personality Disorder, and Dissociative Disorders such as Dissociative Identity Disorder.

TREATMENTS- SSRIS

Wagner et al. (2003) found that usage of the SSRI Sertraline with children and adolescents diagnosed with MDD led them to experience a significant improvement in all depressive symptoms associated with MDD.

Whittington et al. (2004) found that SSRIs were significantly beneficial for young people and adolescents with a diagnosis of MDD.

Gorman, Korotzer and Su (2002) found that the SSRIs Citalopram and Escitalopram were both more effective than placebo drugs in the reduction of the symptoms patients diagnosed with MDD experienced.

Burke, Gergel and Bose (2002) study on the same two SSRIs, with Escitalopram being both quicker acting and, importantly, requiring a quarter of the dosage of Citalopram and having the same effects.

Keller et al. (2001) found in a comparative study of placebos, tricyclic antidepressants and the SSRI Paroxetine, that Paroxetine demonstrated significantly greater improvement in symptoms when used with a sample of adolescents diagnosed with MDD.

TREATMENTS-SNRIS

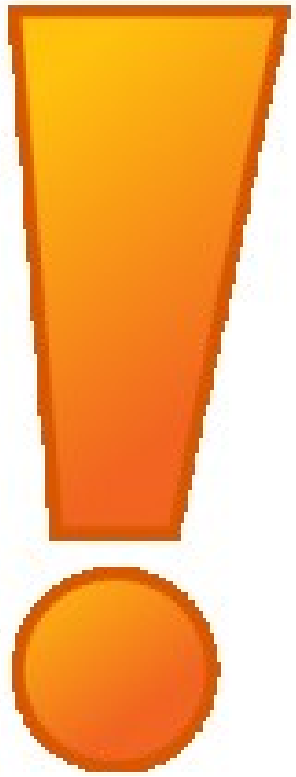
Entsuah, Huang and Thase (2001) contrasted the first and most commonly used, Venlafaxine, with SSRIs and placebo drugs in the treatment of MDD.
Thase et al. (2007) found concurrent results, with a comparison study of two types of SSRI, one SNRI and one placebo showing that though in patients with mild depressive symptoms both active treatments were equally effective, SNRI was far superior in the treatment of moderate- to severe-MDD, and resulted in higher rates of remission.
Nemeroff et al. (2002) found that the SNRI Duloxetine was significantly superior in clinical trials than a placebo in the reduction of both the depressive symptoms of MDD, and the physical symptoms that often plague sufferers of MDD.
Levomilnacipran, another SNRI was also found to cause a significant improvement in patients' MDD symptoms post-treatment in a study of over 700 participants (Asnis, Bose, Gommoll, Chen and Greenberg, 2013).
Doyle et al. (2001) found that SNRIs were more cost effective in almost every country surveyed than SSRIs or tricyclic antidepressants, within both inpatient and outpatient settings.

TREATMENT- TETRA- AND TRICYCLIC ANTIDEPRESSANTS

- Anderson (1998)- in a comparative meta-analysis of studies of tricyclic antidepressants and SSRIs; tricyclic antidepressants were significantly more effective than SSRIs in the reduction of the symptoms of MDD.
- MacGillivray (2003) in a meta-analysis of studies comparing the same two drug groups did not replicate these results, finding no significant difference in the efficacy of SSRIs and tricyclic antidepressants.
- Anderson and Tomenson (1994) and Arroll et al. (2005) who found equal efficacy in treatment for SSRIs and tricyclic antidepressants.

In a study of 186 participants, Jabbari, Bryan, Marsh and Gunderson (1985) found that 15.6% of patients receiving treatment with tetracyclic antidepressants developed seizures after treatment,

Burckhardt et al. (1978) and Glassman and Bigger Jr. (1981) both found that in high doses tricyclic and tetracyclic antidepressants can have extremely detrimental cardiovascular side effects, including a marked increase in heart rate after only three weeks of drug therapy.



NOVEL TREATMENTS

In 2010, the European Union approved the use of the antidepressant Agomelatine in the treatment of MDD. Hale et al. (2010) found that, when compared with the more traditional treatment Fluoxetine, Agomelatine was significantly more effective in the treatment of MDD in assessments both during and post-treatment.

Lemoine, Guilleminault and Alvarez (2007),- found that not only was Agomelatine effective in the treatment of the symptoms of MDD, but also assisted in the reduction of sleep difficulties experienced by sufferers of MDD and other depressive disorders that current medications often fail to alter.

Ghose, Winter, McCarson, Tamminga and Enna (2011) found after dissection of the brains of laboratory rats that the GABA system played a key part in the mediation of symptoms of MDD and the way that a brain may react to antidepressant therapy.

Researchers at Penn State University in a report published in 2016, have detailed how this information has been used to create the effect of anti-depressants in the brains of laboratory mice.

Biochemical changes in brain tissue were also found, and these changes mimicked those found in the brains of mice who had been given antidepressants in separate clinical trials. Treatments such as these show obvious promise in the field of antidepressant therapies, but more trials are needed to confirm their efficacy. Trials with human subjects are also necessary.

THERAPUTIC TECHNIQUES

Casacalenda, Perry and Looper (2002) in a comparative meta-analysis of psychotherapy and pharmacological treatments for MDD, found that both approaches are equally effective in the treatment of mild to moderate MDD.

Thase et al. (1997) concur, with their results showing that though it is necessary in the treatment of severe to combine a course of psychotherapeutic treatments with medication, for mild to moderate MDD there are no statistical benefits for combined medication and psychotherapy over psychotherapy alone.

WHY WE CHOOSE SUICIDE

MARK HENICK

DEPRESSION, THE SECRET WE SHARE

ANDREW SOLOMON