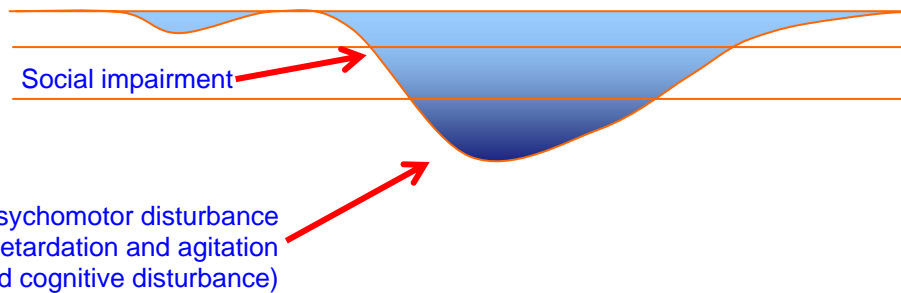




About Melancholic Depression

Melancholic depression is a very distressing condition. It may have a unipolar or bipolar course.



Bipolar depression (previously called manic depression) is a condition which is under-diagnosed in general practice. At some stage in their lives, between 1-5% of people living in Australia (i.e. approximately half a million Australians) are affected by bipolar disorder.

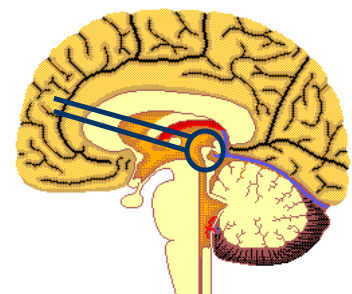
Symptoms of Melancholic Depression

- Anhedonia
- Non-reactive mood
- Mood and energy worse in the morning
- Profound and uncharacteristic inanition – ‘emptiness and inactivity’ (eg. unable to ‘fire-up’ and get out of bed and have a shower).
- **Observable psychomotor disturbance** (see *Glossary of Clinical Terms*) is a very important and specific diagnostic feature of melancholic depression> It includes **cognitive processing problems** (poor concentration, inattention) and **motor signs**: Retardation and agitation affecting the face, speech and body
- *Usually worse in the mornings: Signs tend to fluctuate during the course of the day. This is best observed first hand by the general practitioner. Family and friends may report change in behaviour but not be aware of the significance of this feature.

Mechanisms and Age of Onset

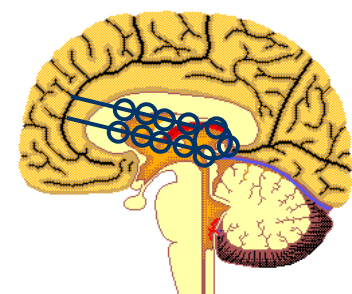
(a) Functional Melancholia

- Younger onset (eg <60 years), ? genetic predisposition
- Often strong family history of depression
- Structural abnormalities rare on imaging
- Good response to broad spectrum antidepressants, ECT
- Mechanism: *Functional* shut-down of circuits linking basal ganglia and pre-frontal cortex.



(b) Structural Melancholia

- Older onset (eg > 60 years), ? vascular predisposition
- Family history of depression less common, but cerebrovascular disease more common
- Structural abnormalities on imaging
- Poorer response to antidepressants & ECT, risk of delirium
- Mechanism: *Structural* disruption of circuits linking basal ganglia and pre-frontal circuits, preceding full dementia in months or years.

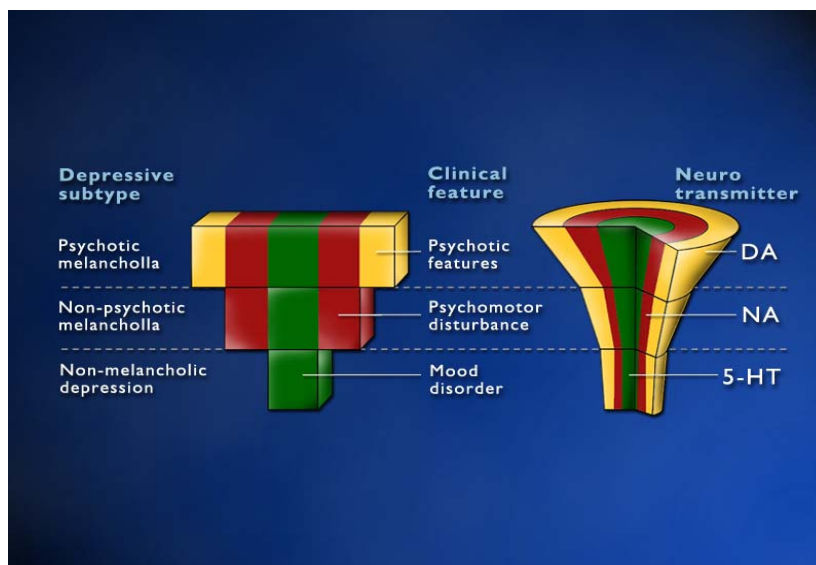


Our Hierarchical Model

The Black Dog Institute model of depressive disorders first identifies melancholic and psychotic depression and then the residual depressions are termed non-melancholic.

Responses to stress and individual personality factors are more important in onset of depression but play a much greater role in the various types of non-melancholic depression.

The model illustrated here suggests the non-melancholic depressions are principally underpinned by serotonergic dysfunction (affecting sleep, appetite, anxiety, irritability, and mood). For the specific features (such as psychomotor change) evident in melancholic depression and the psychotic experiences in psychotic depression, there are additional noradrenergic and dopaminergic contributions. This model aids treatment decisions.



Observational Assessment

- Individuals with good social skills or psychosis may underplay inner distress and despair
- When observing the patient, consider factors such as voice tone, whether the 'light in their eyes' is lost
- Individuals may fluctuate over the day, usually worst in the mornings. It is best for the GP to see them during the part of the day when they report being slower, more hopeless. This is usually in the morning.
- Some useful questions which can help to determine if a patient is suffering from melancholic depression include:
 - Do you still read the newspaper... watch TV?
 - What do you do all day – what would you normally do?
 - What do you still enjoy...hobbies...children/grandchildren...sunrise?
 - Do you feel worse in the morning or the evening?
 - How do you sleep? Do you wake early in the morning?
 - Can you be cheered up? What lifts your mood?

Response to Treatment

- ECT is highly effective. Broader spectrum antidepressants are more effective than 'narrow spectrum', that is TCAs, MAOIs are better than > SNRIs (venlafaxine, mirtazapine, duloxetine) > SSRI and other single-action drugs. The superiority of TCA over SSRI antidepressants increases with age.
- If antidepressant alone fails, brief augmentation of antipsychotic may 'kick-start' response.