Mental Health and Society
Week 5
Introduction to depression & mood disorders.

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Mood disorders are usually divided into unipolar disorders (depression) and bipolar disorders (manic depression) where periods of elation and depression alternate.

Definitions: Depression, according to DSMIV can involve:

i) A major depressive episode, where the depression is significantly disabling, lasts for two weeks or more, is characterised by at least five symptoms (see below) and is not caused by factors like drugs or alcohol. In addition a depressive episode may include delusions and hallucinations. A depressive episode may be described as single episode, recurrent, seasonal, catatonic (if there is excess motor activity or immobility) postpartum, or melancholic (early morning waking, no interest in pleasurable events, appetite loss, guilt). An untreated episode tends to last about 6 months. Half the people who have such an episode will have another within 2 years. People with recurrent depressive episodes have on average 7 in the course of a lifetime (Cancro, 1985).

In some cases of depression people may also experience delusions and hallucinations (Parker et al, 1997; Coryell et al, 1995; APA, 1994)

ii) Dysthymic disorder, where fewer symptoms are present. This lasts longer (2 years is specified) and periods of normal mood interrupt the depression. Sometimes dysthymic disorder leads on to a major depressive episode - this may be called 'double depression'.

Among the changes in DSM 5 are (i) premenstrual dysphoria may get its own category (ii) some symptoms of mania will be recognised as a component of depression - 'with mixed features'. (iii) inclusion of ratings for anxiety and suicide risk. (iv) dysthymia has now been replaced by 'persistent depressive disorder'. (v) there’s a new category ‘disruptive mood regulation disorder’.

Symptoms of depression can include:

i) Emotional symptoms: sadness, dejected feelings, depressed mood, little pleasure. Sometimes also anxiety, anger or agitation may occur. They may lose interest in affection with friends and relatives, and feel angry towards them.

ii) Motivational symptoms 'Paralysis of will' (Beck, 1967). Lack of drive, initiative and spontaneity. Sufferers may become indifferent to life and wish to die, or believe they deserve to. Between 7 and 15 percent of depressed people commit suicide (Coryell & Winokur, 1992; Tsuang, 1978).

iii) Behavioural symptoms. The sufferer may be less productive, spend more time alone, move and act slowly. Speech may be slow, quiet and monotonous. Posture may be hunched or bent and eye contact may be reduced.

iv) Cognitive symptoms include negative views of oneself, inadequate, undesirable, inferior, unattractive, repulsive. Usually a negative view of the future. May complain that their intellectual ability is deteriorating, may be confused and unable to remember things.

v) Somatic symptoms, e.g. headaches, indigestion, dizzy spells, tiredness, and disturbances of sleep. Some depressive problems may initially be diagnosed as medical problems (Simon & Katzelnick, 1995; Coyne et al, 1995)
A further typology is offered by Rutter (1986) who characterises depressed thoughts as follows: i) Feelings of unworthiness or self blame for what has happened. ii) Depressed people think they are unable to change the situation. iii) Pessimism about the future (Rutter, 1986).

**Reactive and endogenous depression**

*Reactive depression* occurs in response to a traumatic event e.g. bereavement, losing one’s job, home or romantic relationship, or in response to some failure, or sudden illness such as having a heart attack or other health problem. Reactive depression can also occur as part of e.g. Alzheimer's disease, withdrawal from e.g. alcohol, cocaine, amphetamines. In these cases depression is seen as a response to life stress or physical change and is not usually taken to indicate a mood disorder. *Endogenous depression* has no obvious precipitating cause.

**Mania & manic depression (Bipolar affective disorder).**

This has now got a separate category in DSM 5 ‘bipolar and related disorders’ and is not included in with depression as a ‘mood disorder’. Mania involves a flight of ideas, elevated mood, and increased psychomotor activity. Extreme so that normal social functioning is disrupted. Mania may also involve extreme and hostile overactivity. In addition hypomania is distinguished where people have some manic symptoms but social or job functioning is not disrupted. DSM distinguishes between *Bipolar disorder* - mania alternating with periods of depression; - *Bipolar II disorder* hypomania (elevated or volatile mood but not quite full ‘mania’) alternating with periods of depression; and *Cyclothymia* with hypomanic and mildly depressed episodes. Drugs based on lithium compounds the most popular treatment, relieves symptoms of about 2/3 of manic patients (Janicak and Boshes, 1987). A variety of other drugs have been tried including antipsychotics e.g. olanzapine & clozapine, and anticonvulsants, which are believed to be effective in rapid cycling forms. Seasonal variations – some patients seem to suffer from depression in the winter and mania in the spring and summer so phototherapy has been tried, where patients are exposed to bright light for 2-4 hours per day. Not known why it works (Wehr et al 1987). Some patients diagnosed with bipolar disorder are unwilling to continue treatment because of the pleasant cognitive and behavioural changes that come with elevated mood (Jamison et al, 1980) or the side effects of medication. There’s now a

**The incidence and prevalence of depression**

The incidence of depression has been the subject of a variety of surveys and epidemiological studies. Helgason (1979) studied Icelanders born between 1895 and 1897 and found that there was a 1 in 8 chance that Icelanders would develop an affective disorder before the age of 75. Holden (1986) estimates 6% of US population experiences depression in any 6 month period. About 20% of depressed people receive treatment. Other estimates come from the National Co-Morbidity Survey (Kessler et al, 2005) where as many as 21% of their
sample of over 9,000 Americans had suffered a mood disorder at some time in their lives. Moreno et al (2011) estimate that 25% of Facebook profiles suggest evidence of depression on the part of their authors. A number of studies suggest that the incidence of depression is increasing (Weismann et al, 1992, Klerman, 1985; Klerman & Weismann, 1989). The risk of depression has increased with each generation since 1915. Efforts were made in these studies to ensure that the methods used to identify problems were comparable for each generation studied.

**Risk factors for depression** can include *stressful life events* in the period leading up to the depressive episode. Paykel (e.g. 1982; 1983, Paykel and Cooper, 1992) has noted that depressed people often have a greater number of these events in the year leading up to the disorder with a particular concentration in the few months before onset. Suija et al (2011) found that episodes of depression were more likely to recur in people who had drug or drink problems, had experienced discrimination and had abusive experiences in childhood.

*Gender* Women are about twice as likely to have a major depressive episode than men (Nolan Hoeksema, 1987). This is true of both treatment seeking rates and incidence rates. The maximum period of risk seems to be in the mid to late twenties. In the UK some researchers have found that if you are a woman having three young children at home, lacking a close confidant not being employed outside the home, losing one's mother before the age of 11 this increases the likelihood of depression (Alloway and Bebbington, 1987; Brown, 1988; Brown et al, 1973).

**Demographic factors and depression**

*Marital status and place of residence.* According to Weismann et al (1991) if you are separated or divorced you are more likely to be depressed than married, widowed or never married people. Place of residence is associated with being depressed - 16% of nursing home residents in the US suffer a major depressive disorder, compared to 9.2% in prisons, 4.4% in mental health facilities and 2.7% in private households.

Leaf et al (1986) and Bromet et al (2011) note that being separated or divorced is a risk factor, and that married people showed lower rates of depression. Weismann (1987) identified risk factors that included a pileup of stressful events, having a family history of depression, being a young child with a depressed parent, all of which increased the likelihood of being diagnosed depressed.

*Social class, gender and children* 
In the UK the social and demographic correlates of depression have been investigated most fully in the Camberwell Study (e.g. Brown and Harris, 1978, Brown et al, 1995). The study has now been taking place for over 30 years. This study highlights the importance of factors such as poverty and the loss of one’s mother before the age of 11. In addition, they also identified the stress of child rearing, and the presence or absence of a close relationship with another adult. In addition they noted that working class women were more likely to experience
severe negative events. Thus, the Brown and Harris research highlights the problems of material adversity a lack of direct economic power and not having access to the labour market. They also found some interactions, such that childless women from all socio-economic groups had similar rates of depression, whereas those with children were more likely to be depressed if they were from a lower SES. The link between poverty and depression is also promoted by Groh (2007). Finally, Brown et al (1995) argue that the crucial factor is a sense of entrapment or humiliation, rather than loss or threatened loss per se. In the US, in work by Mirowsky and his colleagues (Mirowsky, 1990; Ross and Mirowsky, 1988) a similar picture emerges. Amongst employed married women with children in their studies, they found that depression was related to the degree of responsibility for childcare. If childcare was shared between both partners then women were no more likely than the men to be depressed, whereas the likelihood of depression increased with higher childcare responsibilities.

Alternative formulations of depression

The nature of the construct of depression reflects cultural values and ways of thinking about persons. The concept of depression individualises a social transaction (Wiener & Marcus, 1994: 225): 'helplessness, powerlessness and worthlessness do not occur in social vacua'. Allwood (1995) notes how depression serves a number of social functions, in that it urges people to see life events as matters of the mind rather than public domain, encouraging internal self-regulation, especially on the part of women. In the form of liberal humanist therapeutic theory, we are encouraged to see our life-course as a burdensome personal responsibility rather than a need for social change.

Bonnie Burstow (1992) in 'Radical feminist therapy' identifies women's depression as a kind of protest 'depression paradoxically is often the strongest protest that people can muster in a dehumanising situation.' (p 63). Horsfall, (1998, p. 228) says that 'culture and gender relations structure vulnerabilities to extreme distress and disturbance which may be construed as psychiatric. Jacobs (1994) sees the bulk of psychopathology as resulting from deprivation and abusive backgrounds and argues that clinical and research literature has systematically ignored this in favour of biomedical notions of pathology. Gilbert (2000) sees depression as being to do with a loss of status or social rank. Scheff (2001) sees it as being about shame. A number of commentators, including Pilgrim and Bentall (1999) speak of what they call 'the medicalisation of misery' – that sufferers and health professionals are apt to give a medical-sounding title to their distress more commonly nowadays than in the past.

Depression as a problem of social relationships – the “contagion” of depression. Rosenquist et al (2011) looked at data from the Framingham study, a longitudinal study of over 12,000 people originally set up to look at variables such as heart disease, Type A behaviour and stress. However, it has been possible to use the study to examine patterns of mood disorder over time too. Having friends and neighbours with depression increases a person’s own chance of having depression. Female friends are especially potent in the spread of depression.
Depression around the world

Nationality can affect the likelihood of depression. In a cross national study by Bromet et al (2011) lifetime rates of major depressive episode ranged from 21% (France) and 19% (US) to 6.6% (Japan) and 6.5% (China).

A good deal of research on depression (and other problems) is conducted from a European or American point of view, such that many researchers tend to see other cultures’ problems as masked versions of their own (Patel and Winston, 1994). For example Ndetei and Muhangi (1979) reported that anxiety and depression were the commonest problems at a rural clinic in Kenya. Yet they say that 'none of the patients complained of subjective symptoms of either apprehension or fearfulness in the case of anxiety' (p.270). Likewise, there was a lack of 'sadness, guilt or nihilism in the case of depression' (p.270). Even 'direct enquiry about these feeling states also failed to elicit positive responses' (p.270). It would appear that the power of the language of Western diagnostic systems enabled even the absence of key features of anxiety and depression to be glossed over or ignored in the eagerness to find a diagnosis.

Some further evidence about the prevalence of depression comes from Kleinman, (1988) who claimed that depression was rare in India, Africa and other non western cultures. Kleinman believes that depression rates are elevated in response to the pressures of modernisation and industrialisation. In Taiwan, claims Kleinman, in two studies by Lin et al done 15 years apart in the late 1940s and the 1960s a significant increase in depression and anxiety disorders was noted. In China, says Kleinman, most of the research done from the 1950s onwards does not detect depression until after about 1980.

Some authors argue that people express their distress through more somatic symptoms in non-western countries, for example by sleep disturbance, feelings of fatigue, weakness and weight loss (Manson & Good, 1993; Marsella, 1980)

Theories of the causes of depression

Psychodynamic models of depression
In the early years of the 20th century Freud (e.g. 1917) conceptualised depression as a reaction to a loss. The similarities between depression and grieving have been established recently too (Beutel et al, 1995; Stroebe et al, 1992). According to this model people respond to losses by regressing to the oral stage and introjecting the lost person or object. Whereas this passes in most cases some people become preoccupied with the sense of loss and become angry with their lost love object for deserting them. Because the love object has been introjected, the anger becomes anger at the self. Some later object relations thinkers in the same tradition that the threat of loss is also a significant factor (e.g. Kernberg, 1997) such that people who are insecure in their relationships with others will become depressed.

Research evidence suggests that there may be some relationship between early losses and depression later in life. Bowlby (1980; 1969) coined the term anaclitic depression for this phenomenon. A study of 1,250 medical patients by Barnes and Prosen (1985) indicated that those whose fathers had died in their childhood were at greater risk of developing depression. This finding is supported by other
studies comparing people who have lost parents early in life with those who have not (Crook & Eliot, 1980). Childhood experience of parenting has been addressed retrospectively by e.g. Parker, (1992). People who report a childhood featuring ‘affectionless control’ are more likely to be depressed later (Parker et al, 1997). The view of the self in dreams has been investigated by Hauri et al (1974) and suggests that depressed people show higher levels of hostility and masochism.

However, not all depressed people have undergone a loss, and only about 10% of people who have experienced a loss experience a major depressive episode (Paykel & Cooper, 1982). Equally some studies have not found a link between childhood loss and later depression, (Parker, 1982) and others have not found a link between hostility to self or others and depression (Klerman, 1984)

**Biological aspects;**

**Hereditary** Twin studies, kinship studies are taken to suggest heredity's role. Weismann et al (1984) discovered that the younger a person is when s/he first experiences depression then the more likely relatives are to be diagnosed depressed.

**Neurotransmitters** have been implicated in depression, particularly the monoamine group, the most important of which are norepinephrine (noradrenaline) NE, Dopamine (DA) and the indoleamine serotonin (5HT). Catecholamine theory of depression - that depression results from a decrease in NE and DA in the brain. This theory is based on the effects of drugs such as reserpin depletes NE and DA and causes depression in some patients. Monoamine oxidase (MAO) inhibitors and tricyclic antidepressants increase the availability of catecholamines in the brain, prevent the breakdown of NE by MAO, or by blocking the uptake of amines and allowing the neurotransmitters to continue to work.

Indolamine theory of depression - serotonin. Traskmann et al (1981) decreased level of serotonin metabolite 5-HIAA in people who’d made suicide attempts, suggesting that depression is associated with low levels of serotonin. Similar theory about acetylcholine (ACh) - increased brain ACh levels were associated with depression and decreased levels with mania (Gershon and Shaw, 1961).

Neurotransmitter regulation failure theory. Siever and Davis (1985) neurotransmitter activity may be highly variable and may be inappropriate to the situation experienced.

It is difficult to be sure about the mechanisms which accompany depression. As France et al (2007) state, contemporary accounts of ‘biochemical imbalances’ are not easy to pin down to decisive scientific findings and appear to simplify and overstate the case. As many authors including Dean (2010) have pointed out, a whole variety of drugs have been observed to improve depressive symptoms, including antipsychotics and anti-anxiety drugs, which are believed to influence neurotransmitter activity in different ways so there is little consistency about the presumed modes of action. See also Barker and Barker (2012).

**Treatments based on biological theories include** antidepressant drugs, tricyclics and MAO inhibitors which increase NE availability, or SSRIs which increase serotonin availability. Tricyclics slow down the reabsorption of NE by
transmitter neurone and MAO inhibitors inhibit the enzyme which breaks it up. SSRI’s or ‘second generation antidepressant’ drugs such as fluoxetine and paroxetine which increase the availability of serotonin by inhibiting its reuptake by the secreting cell. Lithium compounds are used in treating bipolar mood disorders, which may alter the balance of body fluids by replacing calcium, magnesium, potassium, sodium, or may slow down the release or increase the absorption of NE.

Whether or not these treatments work is still somewhat debatable. Irving Kirsch and his colleagues (Kirsch et al, 2008) performed a meta-analysis of a large group of studies submitted to the US Food and Drug Administration and found that overall the benefits of antidepressant drugs were modest, and little different from placebo. They appeared to be more effective in severely depressed people but that was because severely depressed participants responded less favourably to the placebo. The idea of chemical imbalances seems to owe more to drug company advertising than to the primary scientific literature (Lacasse & Leo, 2005)

**Attributional models of depression.**

Attributional models of depression are based on the causes people assign to things. Depressed people tend to blame themselves when things go wrong and luck when things go right. Nondepressed the opposite. Depressed people feel helpless to control their lives and their environment. Seligman (e.g. 1974) and learned helplessness. The helplessness people feel depends on how they interpret the situation. Depression more likely when people attribute negative qualities to themselves as a result of having experienced situations in which they felt themselves to be helpless (Abramson et al, 1978). 3 dimensions to the feeling of helplessness. i) Whether the person sees the problem as internal or external ii) whether the situation is seen as global or specific - totally helpless or just helpless in this situation. iii) Whether the situation is viewed as stable - persistent (chronic) or unstable - transitory (acute). Research indicates that depression can be produced by either stable or global attributions. Internal attributions can produce depression when they are connected with stable and global components (Robins 1988). However, attributional style is not consistent for all people in all situations. Indications are that attributional style is implicated only in a subgroup of depressed people.

**Accuracy** - There’s an assumption in psychology that positive self appraisal, internal locus of control etc. is good. But most nondepressed people have: i) Unrealistically positive views of themselves. ii) Exaggerated perceptions of how much control they have over events. iii) Unrealistic optimism about the future. (Taylor & Brown, 1988). People overestimate their degree of control over events that are largely determined by chance, overestimate their role in making a desired outcome happen (Miller & Ross, 1975). Depressed individuals are less vulnerable to the illusion of control (Greenberg & Alloy, 1989).

**References**


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