WEEK 1 - Application of the stress-vulnerability model in the context of schizophrenia and bipolar affective disorder:

Aetiological models –

- Theories relating to the aetiology of the mental illness can be explored from the basis of the
  - 1/5 16-85 yrs old
  - 250000 : schizophrenia bipolar
  - biomedical model
  - psychological model
  - sociological model

1. biomedical model;

- Proposes that behaviour is influenced by physiology, with normal behaviour occurring when the body is in a state of equilibrium, and abnormal behaviour being a consequence of physical pathology
- Cerebrum – cognition conscious
- Limbus – emotion, behaviour
- Cerebellum / basal ganglia – movement
- Brain stem – physiology ie BP

2. Psychological model;

- Psychoanalytic theory
  - Asserts that behaviour is driven by unconscious processes, and influenced by childhood/developmental conflicts that have either been resolved or remain unresolved
- Behavioural psychology
  - Presents the view that behaviour is influenced by factors external to the individual (learned)
- Cognitive psychology
  - Acknowledges the role of perception and thoughts about oneself, one's individual experience and the environment in influencing behaviour
- Humanistic psychology
  - Focuses on the development of a concept of self and the striving of the individual to achieve personal goals

3. Sociological model;

- Shifts the emphasis from the individual to the broader social forces that influence people. This model challenges the notion of individual pathology

Stress vulnerability model – stress diathesis model; a conceptual framework for understanding the relationship between stress and vulnerability

- Vulnerability
- Ambient and life event stressors
- Protective factors

- Vulnerability: can be described as the disposition of the person to manifesting symptoms of mental illness
  - What determines the natural vulnerability threshold for an individual? Is this static?
  - How does the degree of vulnerability vary across mental illnesses?

- Innate (genetically determined)
- Acquired (specific disease, perinatal complications, previous life events)

What percentage of genes is shared in successive generations of the same family?

Relationship	**Amount shared**
Monozygotic (identical) twin	100%
Dizygotic (non-identical) twin	50%
First-degree relative (parent, son/daughter, sibling)	50%
Second-degree relative (grandparent, uncle/aunt)	25%
Third-degree relative (great grandparent, great uncle/aunt, cousin)	12.5%

What is the relative risk of developing schizophrenia based on relationship to someone who has schizophrenia (the proband)?

Relationship to proband	**Risk of developing schizophrenia**
First-degree relative	6-17%
Second-degree relative	2-16%
Third-degree relative	2%
General population	0.85-1%

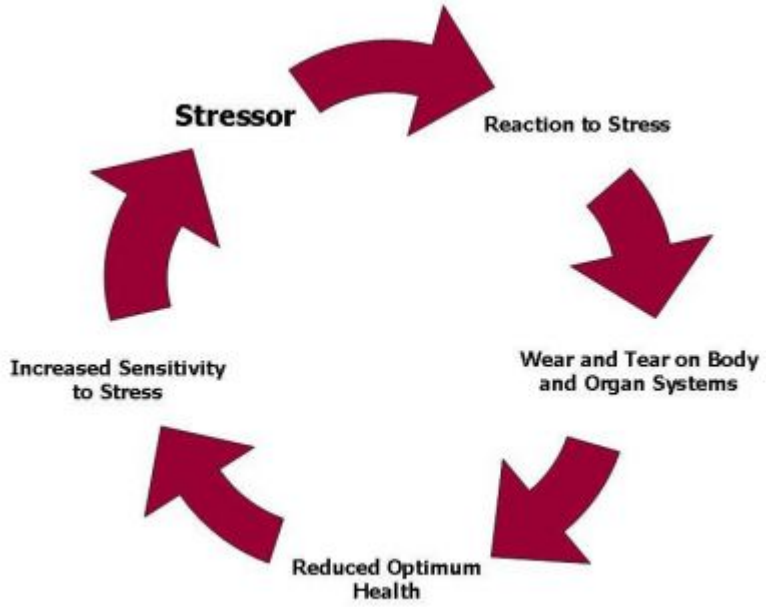
What is the relative risk of developing schizophrenia for a twin of someone who has schizophrenia (the proband)?

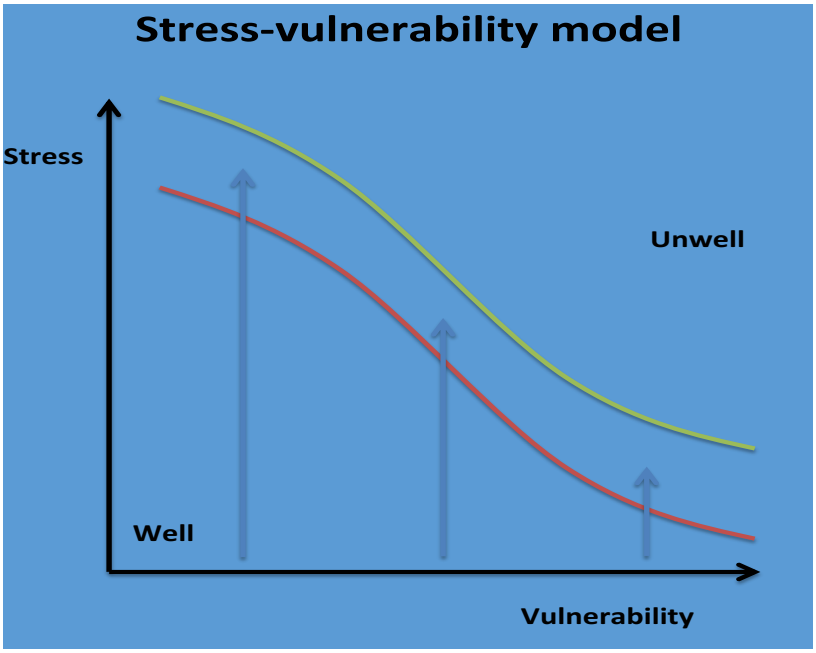
Relationship to proband	**Risk of developing schizophrenia**
Monozygotic twin	30-40%
Dizygotic twin	10-15%

What is the incidence of schizophrenia among biological relatives versus adoptive relatives in people who have been adopted?

Relationship to proband	**Risk of developing schizophrenia**
Biological relative	5-32%
Adoptive relative	0-18%

- Stress: a variable that influences the manifestation of symptoms
  - Ambient stress (day-to-day stress – e.g. unpaid bill)
  - Life event stress (specific, high levels of stress – e.g. death of a close family member)





what is mental illness?

- Mental illness is a general term for a group of illnesses that affect the mind or brain: and then inturn effect, mood, behaviour and thought.

Recent developments:
The Roadmap for National Mental Health Reform 2012 - 2022:

- Promotes person centred approaches
- Improve mental health and social and emotional wellbeing of all
- Prevention of mental illness
- Early identification and intervention
- Quality services
- Social and economic participation for people with mental illness

NSW Mental Health Act 2007:

- The NSW Mental Health Act 2007 is an Act of Parliament that governs the care, treatment and control of people in NSW who experience a mental illness or Mental Disorder.

WEEK 2 – SCHIZOPHRENIA

- A syndrome
- Psychosis is characterised y fundamental distortions of thinking, perception and emotional response
- The most common of these disorders is schizophrenia
- 47% of all psychoses

minimum, psychosis means delusions and hallucinations. It generally also includes symptoms such as disorganised speech, disorganised behaviour, and gross distortions of reality testing. Therefore, psychosis can be considered to be a set of symptoms in which a

person's mental capacity, affective response, and capacity to recognise reality, communicate and relate to others are impaired. Psychotic disorders have psychotic symptoms as their defining features and include illnesses such as schizophrenia

Epidemiology:

- Three in every 100 people (3%) will experience a psychotic episode.
- One in every 100 people (1%) will develop schizophrenia. This represents approximately 224,000 Australians.
- Symptoms of schizophrenia usually become apparent during adolescence or early adulthood. Age of 1st onset is usually 15 to 25 for men and 25 to 35 for women.
- More males than females are affected.
- Schizophrenia exists in all cultures in all countries. The symptoms are similar around the globe.
- It is estimated that schizophrenia costs the community at least $2.5 billion per year in direct costs.
- Approximately 10% of people diagnosed with schizophrenia will end their own lives.

Neurodevelopmental hypothesis of schizophrenia;

- Abnormal brain development
  - One theory is foetal brain development during the early stages of neuronal selection and migration
  - Usually not evident to later stages in late teens
- Obstetric complications
  - Increased in those with a foetal history of obstetric complications ranging from viral infections to starvation to autoimmune processes and other such problems in the pregnant mother
  - Insult to the brain in early foetal development
  - 'die cast' much earlier – this is abnormal degenerative process may be 'turned on' genetically very early in foetal brain development
  - symptoms do not occur until the brain extensively revises its synapses in adolescence
  - possible that the degenerative process continues during the symptomatic phase of schizophrenia.
  - Reducing nerve growth factors and stimulating certain noxious processes that kill off critical neurones such as viral infections, hypoxia, trauma, starvation, or stress
  - The results count be either structural abnormalities or more subtle problems such as, including selection of the wrong neurones to survive in the foetal brain, neurone migration to the wrong places, neurone innervation of the wrong targets, and mix-up of the nurturing signals so that what innervates these neurones is also mixed up.
  - Problems with proteins involved in the structural matrix of synapses may occur in schizophrenia, leading to reduced numbers of synaptic vesicles, aberrant synapse formation, and delays or reduction in synapse formation.
- Genetics
  - Twin studies – higher concordance for monozygotic twins than for dizgotic twins

- Monozygotic twins' offspring have a morbidity rate of 10-15% regardless of whether the parent had schizophrenia or not.
- dizygotic twins, the morbidity rate was 10-15% for the offspring of the parents with the disorder, whilst the offspring of the unaffected twins had a morbidity rate of only 2%.
- the data indicates having a parent who is genetically vulnerable to schizophrenia is a significant risk factor, and that it makes no difference whether the parent actually does or does not develop the disorder.
- multiple genetic abnormalities are likely to each contribute in complex ways to a vulnerability to schizophrenia and other psychotic illnesses, perhaps only when other critical environmental inputs are also present.

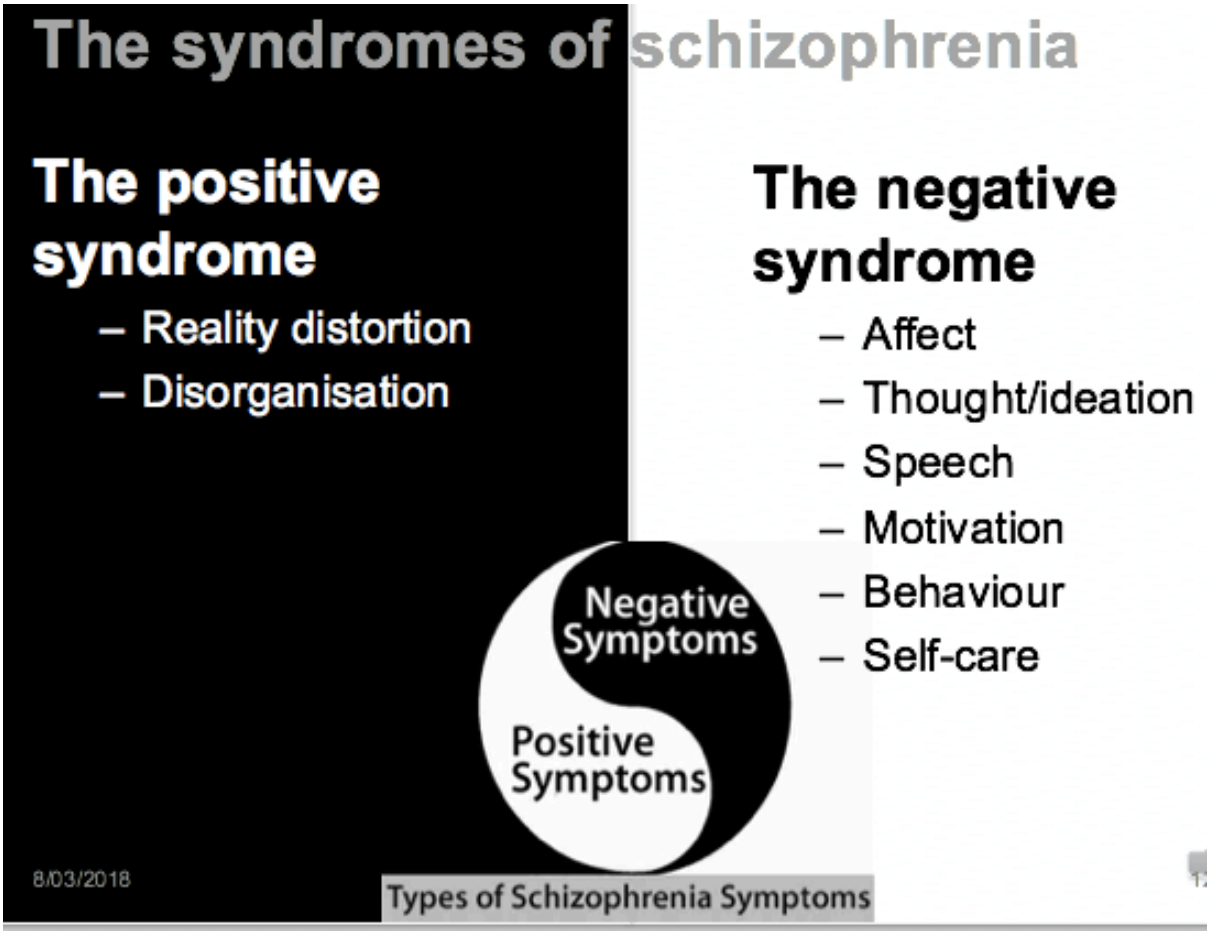
Causative theories:

- Biological theories
  - Neuroanatomical abnormalities
  - Genetic predisposition
- Biochemical theories
  - Dopamine hypothesis
- The diathesis-stress model
  - Stress vulnerability model

Neuroanatomical abnormalities:

- "Commonly, but not conclusively, modern imaging techniques reveal lower brain tissue volume and higher cerebrospinal volume in people with schizophrenia".
- "How and why this occurs is unknown. Did the changes occur due to genetics or environmental factors during gestation or are the brain changes the result of the illness? More research required".

Neurodegenerative hypothesis:
Course of illness –
Prodromal – low – grade psychosis, initial markers often develop in early childhood
Acute – including exacerbation, relapse, subacute or convalescence
Chronic – includes adaptive or stable plateau



Affect

The patient shows decreased emotional responsiveness to their surroundings and thoughts and to what people say or do to them. Affect may be flat, vacant and blunted or coarsened, or there may be a cold, aloof self-absorption. All forms of affective negative disorder result in difficulty in establishing rapport with the patient. Social disability follows, as support networks are lost because of the indifference of the patient towards relationships.

Thought/ideation

An impoverishment of thinking is discernible. The circle of ideas seems narrowed; in conversation themes are reiterated in a stereotypical and repetitive fashion without the usual elaboration. Additional cues to maintain the conversation are not forthcoming. The interviewer finds it hard to keep the conversation going and to introduce new material successfully.

Speech

The patient produces less speech and their replies are laconic and uninformative. When more severe, speech may become monosyllabic. In extreme cases, the patient is mute. As it follows from poverty of thought, poverty of speech makes maintaining the conversation difficult and unrewarding. Social interactions suffer because of this conversational deficit and the emotional unresponsiveness that goes with it; friends and eventually family tend to give up paying attention to the patient, and relationships founder as a consequence.

Motivation

The patient accomplishes little, spending long periods doing nothing, especially if no external stimulation is received. The ambitions of the patient are lost and former activities are carried out less and less, the ability to work fails, as does the motivation to pursue

leisure interests. In extreme cases, the patients may need constant prompting to do even simple tasks.

Behaviour

The patient sits abnormally still. There is a blank facial expression, which does not change despite the content of conversation. There is decreased use of gesture. Body movements are reduced in number and extent, and are abnormally slow; the gait, in particular, is often lumbering and awkward. The patient takes excessive lengths of time to get anything done.

Self-care

The personal hygiene and appearance of the patient deteriorates. Patients may be malodorous with soiled, unwashed clothes worn for weeks or months. Patients may seem indifferent to dirt, cold and discomfort. In extreme cases, the patients need intensive prompting and assistance with the most basic daily activities, such as getting up, and have to be brought to the table and encouraged to eat.