Cognition and Psychopathology

Depression

Types of Depression

There are a number of disorders that have as a central feature lowered or depressed mood, including:

- *Major Depressive Disorder*: episodes of depression
- *Bipolar Disorder* ("manic-depression"): episodes of depression and episodes of manic mood
- *Dysthymia*: chronic lowered mood, not as severe as major depression
- *Schizo-affective Disorder*: depressed mood coupled with psychotic features

We’ll only address Major Depression
Diagnostic Criteria (DSM-IV)

5+ of the following symptoms during the same 2-week period nearly every day and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure. (Do not include symptoms due to a general medical condition or mood-incongruent delusions or hallucinations.)

1. depressed mood most of the day
2. markedly diminished interest or pleasure in all or nearly all activities most of the day
3. significant weight loss or gain, or decrease or increase in appetite
4. insomnia or hypersomnia (excessive sleep)
5. psychomotor agitation or retardation (jitteriness of slowing) (objective observation)
6. fatigue or loss of energy
7. feelings of worthlessness or inappropriate guilt (which may be delusional)
8. diminished ability to think or concentrate or indecisiveness
9. recurrent thoughts of death or suicide, or actual suicide attempt
Some of these symptoms are physical, some are mood-related, and some are cognitive.

Epidemiological Aspects

- lifetime prevalence: 10-25% women, 5-12% men
- cross-sectional prevalence: 5-9% women, 2-3% men
- death rate: 15% die by suicide; 4X death rate for over 55
- co-morbidity: often accompanies medical disorders or other psychiatric disorders (e.g., anxiety disorders)
- recurrence: risk of experiencing another episode of depression is 50% of those who have had one episode; 70% for two episodes; 90% for three episodes
- role of stressors: severe psychological stressors (e.g., death of loved one) seem to play more of a role in the onset of initial episodes than in later episodes
Measures of Depression

- Hamilton Rating Scale for Depression (HRSD)
  - A trained rater asks specific questions about the severity of various symptoms, and derives a single number that describes overall severity of depression.

- Beck Depression Inventory (second edition -- BDI-II)
  - Self-report questionnaire that asks about various symptoms (although somewhat differently from HRSD).
  - Used primarily by clinical psychologists, especially those who practice Cognitive Behaviour Therapy (CBT)

The two measures have relatively high correlation (~.60-.70), although the BDI-II is not much used outside of CBT.
Cognitive Accounts of Depression

A central feature of major depression: disturbance in cognition

Important clinical aspects of depressive cognition include:

- negative (often extreme) beliefs or attributions
  - negative view of self
  - negative interpretation of events
  - distortions
  - overgeneralizing, "catastrophizing"
- inability to generate alternatives

Two major cognitive accounts of depression:

- Beck's Schema Theory
- Abramson's Hopelessness Theory
Schema Theory

Negative cognitive products are the central feature of depression. The products include the negative judgements or attributions that depressed individuals make about:

- themselves
- their circumstances
- the future

(the "negative cognitive triad").

These negative thoughts often come about "automatically":

- repetitive
- persistent,
- not easily controllable

Furthermore, they are often not always "conscious" in that the person may not be immediately aware of them.
For Beck, the negative cognitions are the primary cause of depression, and produce the affective, behavioural, and physical symptoms of the disorder.

The negative cognitive products are caused by negative cognitive content that has been organized or structured into schemata.

The concept of schema was originally developed in experimental cognition in the '60s and '70s, to account for the effects of an individual's prior organized knowledge on later cognitive processing.
A schema:

- is a relatively stable knowledge structure
- contains generalizations developed through prior experience with a given area
- guides the processing of incoming information through the filtering and selection of stimuli
- influences the perception, interpretation, and memory of stimuli relevant to area
- produces cognitive effects which are relatively automatic

We possess schemata for a huge variety of domains of knowledge.

Beck argues that we also possess self-schemata that:

- organize information about the self
- interprets incoming information and generates expectations

In depressed individuals, the content of these self-schemata are primarily negative, and produce the dysfunctional core beliefs that are seen to be at the heart of depression.
Schema Theory is a *diathesis-stress* model:

- depressive schema not necessarily always active
- produces a vulnerability or predisposition (diathesis)
- actual cognitive effects do not occur until the appropriate stressor is encountered (e.g., negative event, low mood)

**Schema Theory and CBT**

- Beck’s model was developed for, and forms the basis of, his approach to psychological treatment of depression, Cognitive Behaviour Therapy (CBT).
- In Beck’s account, the negative schemata of depressed individuals produce distorted interpretations of incoming information (e.g., social cues, events, etc.).
Goals of CBT are to:

• get the client to understand the connection between thoughts and feelings
• examine rationally their beliefs
• identify cognitive distortions and dysfunctional thinking
• through experience, change their depressive schemata into one's that are more functional.

Individuals are taught to identify "automatic thoughts", attributions or beliefs that are produced by depressive schemata but of which the individual may not always be aware.

Through a process of "collaborative empiricism" with the therapist, individuals are encouraged to test these dysfunctional beliefs by examining the evidence for and against their attributions.

(Presumes the depressed individual's evaluations are distorted or inaccurate, and that by identifying and explicitly testing these evaluations, the distortions can be recognized and eventually corrected.)

Beck's Schema Theory of depression has been enormously influential, and much of the cognitive research into depression takes this account as a starting point.
Hopelessness Model

The Hopelessness Model of depression is a refinement of an earlier account, the Learned Helplessness Theory originally proposed by Seligman.

Learned Helplessness

- Seligman (e.g., 1975) conducted a number of animal learning studies that examined how animals reacted to uncontrollable negative events (e.g., shocks). He found that, after experiencing uncontrollable shocks, animals would exhibit many motivational, behavioural, and cognitive deficits.

- E.g., when placed in a situation where the shock could be avoided, animals failed to learn what behaviour would prevent shock, and appeared to "give up"

Seligman argued that the lack of relation ("noncontingency") between behaviour and outcome caused "learned helplessness", by producing an expectation that no action would prevent the negative stressors.

The effects of learned helplessness in animals, he claimed, seemed similar to the deficits observed in human depression.
A revised version of the theory (Abramson et al., 1978; Seligman et al., 1979) held that it was not just the perceived lack of control over negative events that caused the negative effects, but the interpretation of this lack of relation (the attributions made).

Three dimensions of attributions important to learned helplessness:

- *internal/external*: is the outcome produced by some aspect of the individual, or some outside cause?
- *stable/unstable*: will the result happen every time, or is it changeable or random?
- *global/specific*: does the cause produce the outcome in all situations, or only in specific instances?

The attributional style that an individual has may determine their risk for depression, according to this view.

People prone to depression:

- attribute negative outcomes to internal, stable, global causes
  - "I caused it, I can't change it, and it will happen no matter what situation"
- attribute positive outcomes to external, unstable, specific causes
  - "It didn't have anything to do with what I did, it was just luck anyway, and it could only happen in that situation"

(Unlike that original study animals engaged in this sort of complex attributional process.)
Hopelessness Model

In response to criticisms of the Learned Helplessness Theory, along with further empirical work, a revision of this account, termed the "Hopelessness Model", was developed by Alloy and Abramson (e.g., Abramson et al., 1989).

The Hopelessness Model postulates that there is a specific subtype of depression caused by hopelessness. (Note that this is no longer intended to be a comprehensive model for all instances of depression).

The existence of hopelessness will always result in depression; it is a proximal (immediate) and sufficient (no other factor required) cause.

In this revision, the key aspects of inferences about events that determines the development of hopelessness are:

- the perceived cause of the event (stable & global)
- the judged outcome of the event (negative)
- the impact of the event to the status of the individual (negative)

Also important is that the negative event match the content domain of the individual's negative attributional style (e.g., interpersonal or achievement).

In this account, internal/external no longer as important an attributional dimension,
Comparison of Models

Similarities:
• diathesis-stress models
• presume vulnerability for specific stressors only
• only account for simple major depression
• allow for biological factors in other types of depression
• highlight the role of maladaptive inferences
• say little about the specific origin of the diathesis (cf. psychodynamic approaches)

Differences:
• Schema theory claims all depression, Hopelessness accounts for a specific subtype
• Hopelessness does not assume that depressive cognitions are necessarily biases or inaccurate, merely maladaptive
• Hopelessness theory offers account of prophylactic factors against depression (protective attributional style: unstable, specific attributions of negative events)
• Hopelessness tends to focus on the product of cognition, and offer a description of those products without explaining their origin; Schema theory offers account of mechanisms by which dysfunctional cognitions arise
These two accounts are not necessarily incompatible, and may simply be a difference in emphasis.

(Attributional style may be the result of the nature of the schemata that one has, and depressive schemata may produce the type of inferences seen in those with a depressogenic attributional style.)

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**Cognition and Emotion: Mechanisms**

Neither Schema Theory nor Hopelessness offers a specific mechanism that describes the relation between cognition and emotion.

One influential framework which does this is the associative network approach of Bower (initially presented in 1981).

Inspired by the network models of memory proposed in the '60s and '70s.

These approaches modeled the way in which the meaning of concepts was stored in memory.
Semantic memory is a network of interconnected nodes

Each node contains a concept (word meaning)
Nodes are connected to each other, with more highly related nodes connected more strongly than weakly related nodes.

When the cognitive system encounters an instance of a concept, its node is activated.
Activation from a node will travel down its connections to other, related nodes (spreading activation)
A node that receives some activation needs less further activation to become available to the cognitive system (it has been primed)
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Bower argued that, in addition to nodes for concepts, there are nodes for specific emotions (moods) as well.
Activation of a mood node produces the experience of the relevant mood.

If associated concepts activated by events, this will activate emotion node, producing mood.
Note that this account assumes that:

- memory is structured, such that certain concepts are more highly related than others
- activation of a concept will make related concepts more likely to be activated, and thus influence further processing
- negative mood will be more reliably generated by some types of events (those involving specific mood-associated concepts) than other events

Note that these assumptions are similar to some of those found in the more general accounts of depression:

- structure: similar to notion of schema
- processing influences: similar to effects on perception, evaluation, and memory that schemata are supposed to produce
- specificity: similar to notion of specificity of stressors (types of vulnerability)
It may be possible to use a network account to provide the specific mechanisms for a schema approach to depression.

Specifically, depressive schemata may simply be collections of concepts that are highly associated with each other and with sad or depressed mood, such that activation of one concept activates the other, related concepts, and also activates the related mood node.

The associated concepts may well involve a particular domain (such as interpersonal relations, or individual success/failure).

Note, however, that the Bower approach is similar to a number of other semantic network approaches. It might be best to view this account as a framework, a way of thinking about mechanisms and producing specific models, rather than a specific theory in itself.

Nonetheless, this approach has been very influential in research in mood disorders, especially depression.
Cognition in Depression: Empirical Work

Cognitive theories of depression make a number of predictions about the way in which cognitions will be affected in depression, and how cognition and depression are related.

There are five areas of research that have receive the bulk of attention (Engel & DeRubeis, 1993):

- thinking in depression
- schematic processing in depression
- cognitive biases and distortions in depression
- cognitive vulnerability to depression (i.e., the causal role of cognition in depressive onset)
- automaticity of depressive cognition
General research considerations:

*Mood Congruent Effects*

- Material similar in affective tone to current mood is recollected better
- to partial out *mood-congruent effects* from other cognitive effects, shouldn't use mood-related stimuli
  - e.g., in memory studies, "unhappy", "blue", and "depressed" may be affected by mood-congruent recall.
  - Should only use non-affect terms, like "failure", "alone", "stupid" if one wants to look at cognitive aspects.

*Idiosyncratic Nature of Depressive Cognitions*

- not every depressed person will have the same depressive cognitions, e.g., sociotropy vs. autonomy
  - using the same stimuli for every depressed person may not be a very powerful test of hypothesis.
  - individualized stimuli may provide better test (although makes comparing across individuals more difficult).
Heterogeneity of Population

• clinical populations are generally very heterogenous (vary greatly)

• an effect of interest may be influenced by many different variables

  - e.g., current level of depression, past number of episodes, treatment history (e.g., psychotherapy vs. pharmacotherapy), etc.

• A number of co-variates must be controlled for (through careful subject selection/matching, statistically, etc.) when doing such research.

Do depressed individuals show more negative thinking?

Negative cognition is central to both the Beck's schema model and the Hopelessness Model of depression. To what extent do depressed individuals show greater negative thoughts than non-depressed controls?
There are a number of questionnaires that have been specifically designed to assess the type of thoughts that depressed people have. Two common measures are:

- **Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980):** lists 30 common negative thoughts and asks the individual to rate how frequently they have each thought, and the degree to which they believe them.

- **Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978):** presents 40 statements of dysfunctional beliefs, and asks individual to rate how strongly they agree or disagree with each statement.

On these questionnaires, and on many other similar measures, depressed individuals show greater negative cognitions than do non-depressed controls (generally supporting Beck's negative cognitive triad: negative view of self, world, and future).
Are negative cognitions enduring, or are they simply symptom?

- *In episode* individuals report more negative thinking
- *remitted* (recovered) individuals in a non-depressed mood state show significantly less negative thinking (e.g., Dobson & Shaw, 1986), similar to never-depressed

  - suggests negative cognitions do not result from some enduring part of the disorder, but are more related to symptom severity.

  - These and similar results have been used to argue against the causal efficacy of cognition in depression.

*These results will be qualified in a very important way later.*
• Also, negative thinking is found in other psychiatric disorders with secondary depressive symptoms (e.g., Hollon, Kendall, & Lumry, 1986)

  - suggests negative thinking is associated with depressive symptoms, and not only depressive disorder
  
  - negative cognitions alone may be a necessary, but not sufficient, criterion for major depression

How automatic is depressive cognition?

Beck (e.g., 1967) argues that negative thoughts arise automatically in depression.

To what extent has this been demonstrated?
For a process to be automatic, it should:

- occur without requiring attentional resources
- not be subject to conscious intervention

Generally, this issue has not been addressed explicitly in many studies using clinically depressed samples. However, there are some studies that are suggestive.

**Bargh and Tota (1988):**

- college students who were dysphoric
  - (i.e., reported high levels of negative mood, but had not been diagnosed as depressed),
- non-dysphoric controls.

The participants rated whether depressed and nondepressed content adjectives were self-descriptive.

Variable of interest was *rating reaction time.*
• Key manipulation: half of the individuals in both groups also had to keep in mind 6 digits (memory load condition).

- memory load requires attentional resources
  ‣ if rating task also requires attention, the memory load condition should interfere and slow reaction time
  ‣ if self-constructs are automatically activated, and do not need a great deal of attentional resources, load condition will have little impact on rating latencies.

Results:

Dysphoric participants:
• memory load produced smaller increase in rating time for depressed-content words than for non-depressed adjectives

Non-dysphoric participants:
• smaller increase under memory load for nondepressed adjectives.
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<th>Pos</th>
<th>Neg</th>
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<tbody>
<tr>
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<td>+</td>
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<tr>
<td>Non-dysphoric</td>
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<td>No Load</td>
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For dysphoric participants, self-rating required less attention (was more automatic) when the content was negative than when it was positive.

Conclusion: for dysphoric individuals, processing of negative information is relatively more automatic than processing of positive (the reverse is true for nondysphorics).

(Keep in mind that this is an "analogue" study - the individuals examined were dysphoric, but were not clinically depressed.)
Although Bargh and Tota found automaticity effects for simple judgement of self-description, automaticity can also be demonstrated in more complex judgements.

Andersen and Limpert (2001) looked at future-event schema processing in depressed and non-depressed individuals.

Future-event schemas are used by individuals to predict likelihood of future events, positive and negative.

Are judgements of future events automatic in depressed individuals?
• Depressed and non-depressed made judgements of how likely various future negative and positive events were.

• Half of each group had a load condition (like Bargh & Tota)

• Measured reaction time for judgement

Results:

• Depressed showed less effect of load on judgement RT, suggesting automaticity in processing

• Also predicted fewer positive events

Depressed individuals show automaticity in depression-relevant complex judgements
Automaticity of processing is sometimes also seen in attentional bias for depressive stimuli, but this result is not consistent -- the possible significance of this inconsistency will be discussed later.

Does depressive cognition result from schematic processing?

A central claim of Beck's account of depression is that depressive cognition results from schemata, organized knowledge structures that guide processing.

What is the evidence?
Unfortunately, most studies have conceptual or methodological flaws that prevent a definitive examination of this issue (Segal, 1988).

- Responses on questionnaire measures of depressive cognition (e.g., DAS) have sometimes been taken to show that depressed individuals possess negative self-schemata.

But, Coyne and Gotlib (1986) note that this argument is circular:

- "Depressed persons make negative verbalizations because of...hyperactive negative schemata, and we know this because they make negative verbalizations."
Other studies have used the Self-Referent Encoding Task (SRET) to look at this question (and somewhat address the above criticism):

- Participants given list of adjectives
  - positive, neutral, negative
- make a decision about the self-descriptiveness of each item.
- Later, participants have to recall or recognize items in an incidental memory test.

In this type of paradigm, schemata are assumed to affect:

- which adjectives are judged to be self-descriptive
- how well the adjectives are remembered
- how long such judgements will take (response latencies)

(Bargh & Tota study is example of SRET paradigm.)
Generally speaking, SRET studies find that depressed participants:

- rate more negative adjectives as self-descriptive (controls more positive)
- take less time to make self-referent judgement for negative adjectives (controls less time for positive)
- recall more negative than positive adjectives (controls more positive than negative)

This pattern of results could arise from schemata (organized, interrelated stable knowledge structure).

However, all the above results show is that negative information is generally more accessible in depressed individuals, and not that it is organized in any fashion.

- Perhaps sad mood simply activates unorganized negative constructs. Under this account, negative adjectives would still be more often rated as self-descriptive, rated faster, and recalled better.
<table>
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What is needed is to demonstrate that the negative information about the self is structured in some fashion.
In general, information structure can be revealed through semantic priming paradigms (see, e.g., Meyer & Schvaneveldt, 1971).

- Presentation of a stimulus (prime) changes performance on a later-presented related stimulus (target), relative to target performance without the prime.
  - Activation of one concept serves to activate a related concept.

E.g., lexical decision task

- Participant indicates if presented letter string is a word or not
- Stimuli presented in series
dog

globnast
frobly

tree
doctor

nurse
slibbing

cloud
Chair

- Key comparison conditions:
  - target word preceded by *related* prime
    - doctor -> nurse
  - target word preceded by *unrelated* prime
    - cloud -> chair

If items organized semantically, related prime should “activate” target, make it easier to process
Segal, Truchon, Gemar, Horowitz, and Guirguis (1995) used a priming methodology to look for schematic processing in depression.

- Used depressed subjects and nondepressed controls in a primed Stroop task with idiosyncratically-generated (personalized) stimuli

**Task:**

- Subject presented with a prime phrase in white on black background, then with a target word in a color
- Subject had to name color as quickly as possible (Stroop task)
I AM UNLOVED

LONELY
green
Does nature of prime, target, and (most critically) their relationship affect color naming?

• If prime activates target, should expect color naming to be slowed.

Design

• positive and negative material

• primes and targets

• both primes and targets could be self-descriptive or non-self-descriptive (neutral)

• primes and targets could be related or unrelated
For target items:

• Subjects rated how self-descriptive positive and negative adjectives were.

• Those in each valence rated most self-descriptive were chosen as self-descriptive targets

• Those with most neutral rating (not lowest) were chosen as non-self-descriptive targets

For primes:

• Subjects filled out the Inventory of Interpersonal Problems

  - questionnaire asking about areas of difficulty in the individual's life around a number of basic themes (e.g., achievement, interpersonal relations)

  - each theme can be positive or negative
• For each valence of each theme, short prime phrases were developed
  
  – e.g., "ABLE TO BE LOVED", "I AM LIKED"/ "UNABLE TO BE LOVED", "I AM NOT LIKED"
  
  – Phrases thought to be more similar to content of automatic thoughts than single words
  
  – The most positive themes were used to produce positive self-descriptive primes, the most negative themes were used for negative self-descriptive primes, and the neutral themes produced non-self-descriptive primes

To get related and unrelated prime-target pairs:

• Subjects paired up the self-descriptive primes in each valence with half of the self-descriptive targets, according to how well the primes and targets related in their own life.

• The pairs made in each valence served as the self-descriptive prime, self-descriptive target (SS) prime-target pairs. E.g.:
  
  – UNABLE TO BE LOVED - lonely
  
  – I AM ACCOMPLISHED - successful
• The non-self-descriptive (neutral) primes were randomly paired with the remaining half of the self-descriptive targets, to generate the non-self-descriptive prime, self-descriptive target (US) condition.

In summary:
• stimuli are positive and negative
• The target is always self-descriptive
• For prime-target pairs
  - half the individual relates the prime and target to each other (SS)
  - half of prime-target pairs are unrelated (US)
Primary question: Does nature of the prime-target relationship affect color-naming time for the target?

• If schematic processing is not taking place, then relation between prime and target shouldn't matter

- Color naming for negative targets might be slower overall than positive for depressed patients, BUT

- prime condition shouldn't affect response time

• If schematic processing is occurring, then presentation of a related prime should slow down color naming of the target

Results:

Relative differences in conditions:

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<th>Non-depressed</th>
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<td>Related Prime</td>
<td>Unrelated Prime</td>
<td>Related Prime</td>
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<tr>
<td>Positive</td>
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<td>Negative</td>
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<td>Negative</td>
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Depressed subjects:

- Slower color naming in related prime condition for negative material.
- No difference between conditions for positive material.

Controls:

- No differences between prime-target conditions for either valence.
Conclusions:

• Negative information in depressed patients is organized, in keeping with schema notion.

  - Positive information does not appear to be schematically organized in depressed patients.

  - No evidence of schematic organization for either type of information in non-depressed controls.

  ▶ (might have expected organization of positive information)

What happens to schematic organization of negative material when a depressive episode ends?

Segal and Gemar (1997) looked at the same depressed subjects after they recovered through CBT.

• Once remitted, formerly depressed individuals showed no differences between conditions for either valence (like never-depressed controls).

• This suggests that successful therapy either alters depressive schemata, or makes such schemata less available (perhaps by developing alternative, more positive schemata).
(The possible persistence of dysfunctional schemata will be discussed later.)

Do depressed individuals show biases and distortions in information processing?

To what extent does depression produce biases in cognitive processing?

Biases can potentially occur at a variety of "levels" in the cognitive system, from lower level processes such as perception and memory, all the way up to complex judgements of probability.
*Perceptual Bias*

- Some studies have shown an attentional bias to the perception of depressive stimuli.

- Most commonly used task is the Stroop.

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e.g., Gotlib & McCann (1984):

- mildly depressed individuals and non-depressed controls

- colour-named negative, neutral, and positive words

Results:

- Depressed group slower to name negative words compared to neutral or positive.

- No difference in colour-naming RTs in non-depressed group.
Similar results have been found in a small handful of other unprimed Stroop studies in depression.

However, some studies looking at the issue of attentional bias in depression have reported null findings.

- E.g., Hill and Knowles (1991) found no difference between depressed and control subjects on colour naming of negative, neutral, and positive words.

Tasks other than the Stroop have also failed to show attentional bias effects:

- *lexical decision*: numerous studies of depressed mood, including Matthews & Southall, 1991, who used depressed patients

- *perceptual threshold*: identifying briefly presented words (Gerrig & Bower, 1982)

-
• facilitation of spatial attention tasks: tasks of this nature involve some sort of detection or identification of a neutral nonverbal stimulus that appears in the same location as a prior target word.

- Facilitation for the task should result if prior target engaged attention for that location.

- Gotlib, McLauchlan & Katz (1988):
  
  ‣ presented two words on screen, either could be positive, negative, or neutral

  ‣ two color patches appeared in the same location as words

  ‣ Task was to judge which patch appeared first
loved
failure

blue
• both colours were actually presented simultaneously

• compared to depressed subjects, non-depressed controls identified color patches that replaced positive words as appearing faster
  - selectively attended to positive words

• Later replication by Mogg et al. (1991) showed that the effect was not due to level of depression, but instead level of anxiety

• Visual dot-probe tasks
  - pairs of words appearing on screen
    ‣ participant to identify one member, based on location
  - occasionally, dot appears in either location.
    ‣ participant must indicate when dot appears
MacLeod, Mathews, and Tata (1986):

- Three groups
  - dysphoric (high BDI)
  - anxious (high State-Trait Anxiety Inventory)
  - controls
• Presented threat and neutral words

• Results

  - dysphoric: no difference in RT based on word

  - anxious: faster for threat words
    ‣ attended to threat

  - controls: slower for threat words
    ‣ threat avoidant

Mathews, Ridgeway, and Williamson (1996) found depressed subjects attended to social threat, whereas anxious attended to physical threat.
Maybe these effects are just generally difficult to find.

Why the long litany of null findings?

Because in anxiety disorders, such perceptual biases appear everywhere!

Williams, Watt, MacLeod and Mathews (1997) reviewed Stroop studies of attentional bias in depression and various anxiety disorders.

They reported 45 studies demonstrating attentional bias in the case of anxiety, but only 4 for depression.

A similar review by Mogg and Bradley (2005) likewise found robust bias effects for GAD, but equivocal results for studies involving depression.
This may in part indicate the relative level of research interest in using this paradigm in these two disorders,

BUT

Also likely a function of the relatively difficult in generating positive findings for depression (null results are generally not published).

Also, Mogg et al.'s replication of Gotlib et al. color-judgement task showed that it was level of state anxiety, and not depression, that predicted performance.

Likewise, the MacLeod et al. visual dot-probe study found very robust biases (faster dot detection) in anxiety.
So, perceptual biases in depression seem to be much more difficult to demonstrate than in anxiety.

It is possible, however, that lack of findings are due in part to inappropriate stimuli

- Joorman and Gotlib (2007) developed a dot-probe task where happy and sad faces were used instead of words.
  - Faces may be particularly salient given social difficulties in depression

- Currently depressed showed bias for sad faces (faster RT for probe detection when dot replaced sad face). Controls showed bias for happy faces.
Memory

In discussing memory performance, it is important to keep in mind the distinctions between:

- **explicit** and **implicit** memory
  - *explicit*: conscious recollection of prior events or information
  - *implicit*: remembering without awareness
• perceptual and conceptual processes

  - perceptual: involving the form or appearance of a stimulus
    - "data-driven processing"

  - conceptual: involving the meaning or semantics of the stimulus
    - "conceptually-driven processing"

• activation and elaboration

  - activation: the accessing of item-specific information, integration of item's perceptual features

  - elaboration: the encoding and retrieval of relations between items
We have already discussed biases in explicit memory performance when looking at evidence for schematic processes.

In general, depressed individuals demonstrate a mood-congruent memory (MCM) bias.

Material similar in affective tone to the mood the person is experiencing is recalled better.

This result is one of the most reliable lab-based cognitive effects that depression produces (replicated numerous times in various studies).
What about implicit tests of memory in depression?

Why ask this question?

In part, because implicit memory seems to act very differently from explicit memory:

• profoundly affected by perceptual context

• relatively unaffected by age, amnesia, encoding manipulations, retention interval

(Some have argued that implicit and explicit memory involve different neurological systems.)

Is this type of memory affected by depression? Does it demonstrate affective bias?

Very few studies to date have looked at potential bias in implicit memory in depression.
Watkins, Mathews, Williamson, and Fuller (1992)

- depressed and non-depressed subjects
- incidental study task (not told to memorize) on a list of negative, neutral, and positive words
- then performed:
  - a word stem completion task (e.g., GUIL___, "complete the stem with the first word that comes to mind")
  - an explicit recall task

Results:

- *recall task* (explicit):
  - usual mood-congruent memory effect (better recall of negative material for depressed subjects).

- *word stem* (implicit):
  - no difference between groups on the different word types for rate of completion with studied items
Similar results were found by Denny and Hunt (1992) using word fragments (e.g., G_I_T_) instead of word stems.

Roediger and McDermott (1992) suggested that the lack of bias in the implicit tasks of these studies is due to the nature of the type of implicit tasks used.

- Word-stem and word-fragment completion are primarily *perceptually-driven* tasks
  - involve the integration of perceptual information and the activation of a matching representation in memory
  - (this is generally true of most, but not all, tasks used to examine implicit memory)
• By contrast, most explicit memory tasks involve the processing of the meaning of the stimulus, and are *conceptually-driven*.

If the primary problem in depression involves the *semantics* of the material, then most implicit tasks, which are not sensitive to this dimension, will show no bias effects, whereas conceptually-based explicit tasks will.

• Previous studies confounded implicit/explicit with perceptually-/conceptually-driven

What is needed, therefore, is a study utilizing an *implicit* measure that is *conceptually-driven*. 
Watkins, Vache, Verney, Muller, and Mathews (1996)

• depressed and non-depressed participants

• visualization task for positive and negative words

• later generate associates to other words, some of which were related to the "studied" items.

They found that depressed participants generated more of the negative target items relative to the controls.

Similar results demonstrating implicit memory bias have been found in other studies (e.g., Bradley, Mogg, & Millar, 1996), although in many cases the paradigms used involved primarily perceptual processing (e.g., standard priming tasks).
In their review of the area, Barry, Naus, and Rehm (2004) extended Roediger and McDermott’s argument, claiming that key feature of studies showing implicit memory effects is not just whether implicit task is conceptual, but whether there is a match between the type of processing at encoding and at retrieval.

This principle is known as Transfer Appropriate Processing (Roediger & Blaxton, 1989).

This approach explains prior studies that found effects for perceptual tasks, as these studies also used primarily perceptual encoding.

In their general approach to affect disorders, the overall pattern of these results has led Williams, Watts, MacLeod and Mathews (1997) to propose that a crucial distinction in understanding the cognitive aspects of affect disorders may involve pre-attentive processing vs. elaborative processing.
• **Pre-attentive** (priming) processes involve the automatic activation of representations, and initial allocation of processing resources based on affective valence (e.g., threat level) of an incoming stimulus.

• **Elaborative** processes involve the strategic elaboration of the incoming item with related items in memory, and further (less automatic?) allocation of processing resources can take place.

An advantage of this approach is that it allows dissociations between the kinds of biases that affect disorders might show.

Thus, anxiety may primarily affect pre-attentive processes, thus producing perceptual biases, while not affective elaborative processes, thus generating no memory bias. Vice-versa for depression.

Such results are difficult to accommodate in a schema model or network account.
Judgement Biases

Alloy and Abramson (1979) proposed the phenomenon of "depressive realism"

- the judgements of depressed persons are more accurate (less optimistic) than non-depressed individuals.

Typical study:

- dysphoric students and non-depressed controls
- perform a task in which they were to estimate the degree of contingency between their pressing of a button and the onset of a light.
In various conditions, judgements of how much control subjects had over the appearance of the light was less accurate for the non-depressed subjects:

- In cases where the light came on frequently, or was associated with winning money, non-depressed overestimated their control

- In cases where the light came on infrequently, and infrequent appearance involved loss of money, non-depressed underestimated their control

Alloy and Abramson concluded that depressed (actually, in the above study, dysphoric) individuals showed "depressive realism", and were "sadder but wiser" than their non-depressed counterparts.

Similar results have been found in many studies, suggesting that Beck's notion that depression necessarily involves cognitive distortions is false.
However, various researchers have challenged the notion of "depressive realism" (e.g., Ackerman & DeRubeis, 1991). Some of these challenges are on conceptual and methodological grounds.

• Most studies of depressive realism have used dysphoric students, and not clinically depressed individuals (i.e., they were "analogue" studies).
  - dysphoric individuals may be more realistic (less positive) in their judgements, but that deeper levels of mood disturbance produce negative distortions.

• The tasks used in these studies bear little resemblance to the domains that clinicians believe depressed individuals show distortions (i.e., the studies are not ecologically valid).
In a review of the literature, Ackermann and DeRubeis concluded that:

- When estimating control, or rating themselves and other people, dysphoric and depressed individuals appear to lack the positive, optimistic bias of non-depressed individuals.

- When asked to judge their impact on other persons, or to recall information about self-relevant evaluative information, such individuals show negative distortions (e.g., underestimate how well they are liked, or under-recall how well they have performed).

Do cognitive factors play a role in vulnerability to depression?

Are cognitive factors responsible for vulnerability to depression, or are cognitive effects simply a symptom of depression?

- "vulnerability model" vs. "symptom model"

Important question for the issue of causality of cognitive factors.
One way to address question: Examine whether processes similar to those in depressed individuals are found in those known to be vulnerable to depression.

*Formerly depressed*

One group especially vulnerable to depression are those who have previously been depressed.

Question: Do formerly depressed individuals have aspects of cognitive processing similar to those found in currently depressed people?
A number of studies from the 1970s to early 1990s looked at cognition in recovered depressed patients to try to identify remaining cognitive effects.

- Some of these studies have used questionnaires thought to tap aspects of depressive cognition, e.g., DAS, ATQ, etc.
- Others have used laboratory-based cognitive tasks, e.g., SRET or Stroop.

E.g. Gotlib and Cane (1987)

- Tested depressed individuals prior to and after treatment using a Stroop colour naming task with negative and positive words.

- Compared to controls, depressed individuals showed greater interference for negative material prior to treatment.

- Post-treatment, no differences between groups was found.
Generally speaking, on both questionnaires and laboratory tasks

- early cross-sectional studies failed to demonstrate any cognitive differences between recovered depressed individuals and never-depressed controls

- longitudinal studies showed that dysfunctional thinking in depressed individuals "remits" as depressive episode ends.

Cognitive effects appeared to be correlated with depressive episode, but not an underlying cause (supporting "symptom model").

In a review article, Persons and Miranda (1992) argued that these prior studies have a fatal flaw, namely:

- These studies tested formerly depressed individuals when in a normal, nondysphoric mood.

But

- Cognitive models of vulnerability are diathesis-stress models. They presume a stressor is necessary to activate the relevant cognitive processes.
Persons and Miranda claimed that in order to demonstrate that depressive cognitive processes persist after recovery, it is necessary that the individual be in the appropriate mood state.

They termed this the "Mood-State Hypothesis".

- essentially a re-emphasizing of the diathesis-stress nature of cognitive models of depression.

Note that this is not simply a tautological restatement of the Symptom Model, "depressive cognition will only be seen when people are depressed". The hypothesized stressor can be a very mild shift in mood.

- In vulnerable individuals, such mild shifts may induce negative cognitions, which reinforce the dysphoric mood, which reinforces the negative cognitions, and so on in a depressive spiral.
How to produce the appropriate stressor?

One way may be mood induction:

- use a procedure to induce the appropriate mood state in the subject

Methods used include:

- reading self-statements (Velten)
- watching film-clips
- listening to appropriate music
- recalling mood-appropriate personal events (autobiographical recall)
These methods have been used extensively in the study of normal mood variation.

(Mood produced is generally short-lived and very mild.)

Since the late 1980s, a number of studies have incorporated mood induction procedures in their examination of cognitive vulnerability.

This approach is analogous to "challenge paradigms" in medicine, where a introduced substance can cause a reaction in an individual that would otherwise not be detected.

- E.g., allergy tests

Therefore, these cognitive paradigms are sometimes called "mood challenge" studies.
What happens when formerly depressed individuals are tested under conditions of mild negative mood?

Miranda and Persons (1988):

- tested nondepressed women, some of whom had a history of depression
- filled out the DAS before and after either a positive or negative mood induction
- correlated mood change with change on the DAS
Results:

• *previously depressed*: significant correlation between mood and dysfunctional attitudes
  - the sadder their induced mood, the higher the DAS

• *never-depressed*: change in mood did not produce a correlated change in DAS.

The findings suggest that depressive cognitions are not simply the result of change in mood --, only individuals vulnerable to depression show a change in cognition with a change in mood.

Teasdale and Dent (1987) used a SRET procedure with formerly depressed individuals and never-depressed controls to examine incidental recall of adjectives under normal and induced negative mood.

• Under normal mood, no difference between groups for negative adjective recall.

• Under induced negative mood, formerly depressed patients recalled more self-descriptive negative adjectives than did controls.
Hedlund & Rude, 1995, found similar results using a self-focus manipulation

- causes the individual to reflect more on themselves and their self-presentation

- thought to be similar to the ruminative state that depressed individuals are often in.

Generally, when results are compared

- studies that test under normal mood only show no difference between formerly depressed and controls,

- mood challenge studies reveal depressive cognitive processing in recovered individuals compared to controls.

Mood challenge studies show that, in general, formerly depressed individuals under negative mood can exhibit depressive cognitive processing.
Findings with formerly depressed are informative, but do the effects arise as some specific impact of prior episodes?

Is effect due to increased risk per se, or due to some “scar” from original episodes?

How to determine this?

Examine individuals who are at-risk, but not previously depressed.

Never-depressed at-risk individuals

Some groups are known to be at elevated risk for depression even though they have never been depressed.

One such group: children of mothers with history of depression.

Many studies demonstrate that children with parents who have depression history are more likely to become depressed as adults.

Do these at-risk but never-depressed individuals show cognitive reactivity under mood challenge?
Joorman, Talbot, and Gotlib (2007):

- Tested never-depressed girls 9-14 years
- Half had mothers with history of depression, half not
- Used mood induction paradigm
- Dot-probe task with happy, sad, neutral faces
  - happy-neutral and sad-neutral pairs
  - dot replaced one of the faces, RT measured

Results:

- High-risk girls detected dots that replaced sad faces faster relative to neutral faces. No difference between happy and neutral

- Control girls showed opposite pattern (happy faster than neutral, no difference between sad and neutral)
To summarize:

Compared to controls, individuals at elevated risk for depression show increased negative cognitive processing following a mood challenge.

BUT

Is this cognitive reactivity to a mood challenge *causally* related to vulnerability?

The implicit assumption of mood challenge studies is that any the mood-related cognitive changes found are somehow causally involved in vulnerable individuals increased risk for depression.

However, no studies had actually demonstrated a connection between such mood-related cognitive reactivity and *risk for recurrence*. 
Does cognitive reactivity predict how likely a person is to become depressed in future?

Segal, Gemar and Williams (1999):

- tested formerly depressed patients (treated either with pharmacotherapy [PT] or CBT)
- filled out DAS and a mood rating before and after a mood induction (music & autobiographical recall)
- recontacted the subjects thirty months later, to determine if they had had a relapse during the intervening period
Results: The degree of cognitive reactivity to a given shift in mood significantly predicted later risk for depressive recurrence.

- those who showed a greater increase in dysfunctional attitudes with the same increase in dysphoria were more likely to have an episode of depression later on

This result demonstrates that mood-related cognitive changes are linked to later return of symptoms.

• CBT group showed less cognitive reactivity (change in dysfunctional attitudes for a given change in mood) relative to PT group.

  - This latter result is expected if CBT really does target cognitions.

  - Also may suggest that CBT actually changes depressive schemata, rather than simply creating competing, more positive schemata.

    ‣ If the latter were true, the negative schemata should reassert themselves in the face of appropriate stressors
Study replicated by Segal, Kennedy, Gemar, Hood, Pederson and Buis (2006) using improved methodology

- currently depressed patients recruited
- randomly assigned to CBT or PT
- monitored after recovery for 18 months

Results:

- Patients recovered through PT showed greater cognitive reactivity (increase in DAS after mood induction)
- Regardless of treatment, degree of cognitive reactivity predicted relapse risk over 18 month followup
- Patients whose DAS increased 8 points or more following mood induction relapsed significantly faster than those who showed less cognitive reactivity.
These studies strongly suggest that cognitive reactivity plays a causal role in future depression.

**Prophylaxis - Preventing Future Depressive Episodes**

If cognitive reactivity is involved in development of future depressive episodes, can cognitive interventions be developed to prevent these effects?
One promising approach: *Mindfulness-based cognitive therapy* (MBCT)

- Developed by Teasdale, Segal, and Williams (1996)
- Based on *mindfulness-based stress reduction* techniques created for management of chronic pain

Mindfulness meditation (developed originally in Buddhist practice) involves the development of "detached observation", moment-to-moment effort to perceive a phenomenon and allow it to register with full awareness, as is, without evaluation.

John Kabat-Zinn at the University of Massachusetts Medical Center has pioneered the use of this form of meditation in the treatment of chronic pain.
Kabat-Zinn uses mindfulness to train chronic pain sufferers not to try and avoid the experience of pain, but to experience it as an event separate from themselves.

- ("You are not your pain.")

Through meditation techniques such as breath focus, patients learn to focus on the sensation of pain without making evaluations about it, and to experience pain without having it overwhelm them.

In a large number of carefully-controlled studies, mindfulness techniques have been shown to be extremely effective in helping chronic pain sufferers reduce their perception of pain and the activity interference that pain causes.
Mindfulness-Based Cognitive Therapy is an adaptation of mindfulness techniques to the concerns of formerly depressed individuals.

The theoretical underpinning is that relapse may arise when an individual is prone to becoming personally invested in their negative thoughts and mood shifts (i.e., when negative mood recruits negative thoughts and vice versa). This may cause such thoughts or moods to spiral into a new major depressive episode.

MBCT teaches individuals both to be aware of their thoughts and feelings, and to see them with detachment, as separate from themselves ("you are not your thoughts.").

Individuals are taught techniques (such as attention to breath) to help them produce such detachment under periods of stress.
Goal is to teach skills that allow individuals to disengage from automatic dysfunctional cognitions, particularly depression-related ruminative thought patterns.

The techniques help prevent attention from being engaged solely by negative cognitive, but to allow the individual to “step back” from negative thoughts while not attempting to suppress them.

Is it effective?

Teasdale, Segal, Williams, Ridgeway, Soulsby and Lau (2000) conducted an international study (Toronto; Cambridge, England; Bangor, Wales) to test MBCT

• 145 recovered patients assigned to MBCT or control

• MBCT involved 8 weekly sessions, then four monthly followups

• All patients followed for 60 weeks
Results

- For patients at highest risk (3 or more prior episodes), risk of relapse was *reduced by half*.

The effectiveness of this intervention, if supported in further studies, would make MBCT the most effective approach of any treatment (psychological or pharmacological) in preventing future depressive episodes.