Treatment and Management of Anxiety Disorders with Comorbid Depression
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| Edmonton, Alberta |
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| Department of Psychiatry  
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| Hamilton, Ontario |
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| Chartered Psychologist  
| Grey Nuns Community Hospital  
<p>| Edmonton, Alberta |</p>
<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Faculty</th>
</tr>
</thead>
<tbody>
<tr>
<td>11:00 a.m.</td>
<td>Welcome and Introduction</td>
<td>Dr. Pratap Chokka</td>
</tr>
<tr>
<td>11:05 a.m.</td>
<td>Comorbidity of Mood and Anxiety Disorders: Epidemiological Aspects</td>
<td>Dr. Roger Bland</td>
</tr>
<tr>
<td>11:25 a.m.</td>
<td>Pathophysiology of mood and anxiety disorders: Shared mechanisms</td>
<td>Dr. Glenda MacQueen</td>
</tr>
<tr>
<td>11:50 a.m.</td>
<td>Current Perspectives on the use of CBT for the Treatment of Anxiety with Comorbid Depression</td>
<td>Dr. Stephanie Mitchell</td>
</tr>
<tr>
<td>12:15 p.m.</td>
<td>Faculty Question Period</td>
<td>All faculty members</td>
</tr>
</tbody>
</table>
Comorbidity of Mood and Anxiety Disorders: Epidemiological Aspects

Roger Bland
Dept of Psychiatry
University of Alberta
Purpose

- To review diagnostic concepts related to comorbidity
- To review some of the epidemiological data on comorbidity of mood and anxiety disorders
- To introduce some of the implications and consequences of comorbidity
General Findings

• Prior to, and including DSM-III, comorbidity was obscured by the use of diagnostic hierarchies. DSM-III used ‘due to’ another disorder
  – not clear how ‘due to’ was to be established
  – very specific rules on which disorders excluded which others
  – used exclusion criteria for 60% of the diagnoses

• Epidemiologic studies from the ECA onwards (using hierarchy free rates) demonstrated high comorbidity between psychiatric disorders. Reasons included:
  – hierarchical elimination may yield false low prevalence rates
  – hierarchies obscure relationships between disorders
  – arbitrary hierarchies obscure the possibility of delineating new syndromes
# Hierarchies

Common in medicine (used by Kraepelin, Jaspers in psychiatry)

Achieves diagnostic parsimony

## Hierarchical exclusionary relationships (Karl Jaspers)

<table>
<thead>
<tr>
<th>Organic</th>
<th>Psychotic</th>
<th>Affective</th>
<th>Neurotic and personality disorders</th>
</tr>
</thead>
</table>
ECA (Epidemiologic Catchment Area)

• Relationships existed between disorders either allowed in or ruled out by the DSM III exclusion criteria or hierarchies.

• Relationships were found using both current (i.e. one-month) prevalences and lifetime prevalence (i.e. both contemporaneous and historical).

Boyd JH et al. Arch Gen Psychiatry 1984; 41: 983-989.
Sturt's Hypothesis

- Sturt noted that having any one psychiatric syndrome increased the risk for having other syndromes.

- These syndromes are not randomly distributed with respect to each other.

- This suggests the need to examine disorders that apparently cluster together.

Distribution of symptoms in a general population sample

Conclusions from factor analysis based on the PSE:

Four factors best describe common symptoms:
1) Generalized anxiety
2) Phobic anxiety
3) Depression
4) Low self-esteem

Dimensional rather than a categorical approach was supported

Factors were strongly correlated
Many symptoms not specific to a factor

(PSE administered to 3101 people in 1982 at age 36)
Comorbidity Definitions (1)

- “any distinct additional clinical entity that has existed or that may exist during the clinical course of a patient who has the index disease under study” (Feinstein 1970)

- In psychiatric epidemiology the emphasis is on the relative risk (usually given as the odds ratio). When the patient has an index disorder there may be a relatively greater or lesser risk of another disorder, e.g. a patient in a major depressive episode is 26 times more likely to have agoraphobia than a non-depressed person.
Comorbidity Definitions (2)

- **Clinical studies**: more than one disorder can be diagnosed in the same individual.

- **Pathogenic comorbidity**: when a disease leads to complications or other diseases which are considered to be etiologically related. (Also prognostic comorbidity) (Concepts of “primary” and “secondary” disorders)

- **“Dual diagnosis”**: is a term covering comorbidity usually of a psychiatric disorder and a substance use disorder, but sometimes used to include mental deficiency
Anxiety and Depression: Lifetime Prevalence

Any anxiety disorder (15% - 25%)
- Specific phobias 7-8%
- Social phobia 1.5-3%
- GAD and agoraphobia 4-6%
- OCD and Panic 1-3.5%

Any depressive disorder (10%-18%)
- MDE 9-17%
- Dysthymia 2-6%

Data from studies in USA, Canada, Switzerland, Netherlands, Norway, Germany, Italy, Great Britain, Australia.

Anxiety and Depression: Onsets

• Anxiety disorders except GAD have onsets early in life – mostly before age 20, often in childhood

• GAD is different and increases with age usually starting after adolescence (more like affective disorders), they frequently had another anxiety disorder in childhood

• MDE onsets start in adolescence, peak by age 30, but can occur throughout life

(Data from several studies)

Anxiety disorders: Pattern of course

- Chronic-mild and persistent
  - Waxes and wanes in distress and disability
  - Frequently switches type of anxiety e.g. social phobia → panic → agoraphobia → GAD
- 1 year remission rate about 40%
- Duration (in remitted cases)
  - Panic 7 years (mean)
  - Phobias 15 years (mean)
  - OCD 6 years (mean)
  - GAD in 40% duration >5 years

(Data from several studies)

Depressive disorders: Pattern of course

- 3 month duration of episodes in community cases
- \( \frac{1}{3} \) remit with no recurrences
- \( \frac{1}{3} \) recurrent episodes with remission in between
- \( \frac{1}{3} \) chronic course, fluctuating or unremitting
- 75% have recurrences at some time
- Dysthymia plus depression has worst outcome especially in adolescents

(Data from several studies)

### Associations between mood and anxiety: Odds Ratios based on 12 month prevalences

<table>
<thead>
<tr>
<th></th>
<th>MDE</th>
<th>GAD</th>
<th>Agoraphobia</th>
<th>Panic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysthymia</td>
<td>99.0</td>
<td>17.6</td>
<td>29.4</td>
<td>10.0</td>
</tr>
<tr>
<td>GAD</td>
<td>33.7</td>
<td></td>
<td>25.7</td>
<td></td>
</tr>
<tr>
<td>Social Phobia</td>
<td>10.2</td>
<td>13.5</td>
<td>21.6</td>
<td>11.6</td>
</tr>
<tr>
<td>Specific Phobia</td>
<td>6.2</td>
<td>6.7</td>
<td>24.2</td>
<td>7.8</td>
</tr>
<tr>
<td>PTSD</td>
<td>20.7</td>
<td>15.1</td>
<td>12.5</td>
<td>17.6</td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>25.8</td>
<td>25.7</td>
<td></td>
<td>25.8</td>
</tr>
<tr>
<td>Panic</td>
<td>29.4</td>
<td>20.3</td>
<td>25.8</td>
<td></td>
</tr>
<tr>
<td>Alcohol Abuse</td>
<td>2.6</td>
<td>2.5</td>
<td>2.9</td>
<td>3.3</td>
</tr>
<tr>
<td>Alcohol Dependence</td>
<td>6.7</td>
<td>11.2</td>
<td>10.7</td>
<td>6.8</td>
</tr>
</tbody>
</table>

Note low comorbidity odds ratios with alcohol disorders

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Proportion of anxiety and mood disorders that are comorbid (1)

<table>
<thead>
<tr>
<th>Anxiety Disorder</th>
<th>% Comorbid in 12 month and LT</th>
</tr>
</thead>
<tbody>
<tr>
<td>GAD</td>
<td>82.6</td>
</tr>
<tr>
<td>Panic</td>
<td>88.1</td>
</tr>
<tr>
<td>Social Phobia</td>
<td>68.1</td>
</tr>
<tr>
<td>Specific Phobia</td>
<td>68.3</td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>82.2</td>
</tr>
<tr>
<td>PTSD</td>
<td>69.5</td>
</tr>
<tr>
<td>Any anxiety</td>
<td>58.6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mood disorder</th>
<th>% Comorbid in 12 month and LT</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDE</td>
<td>69.9</td>
</tr>
<tr>
<td>Dysthymia</td>
<td>88.0</td>
</tr>
<tr>
<td>Bipolar I</td>
<td>98.1</td>
</tr>
<tr>
<td>Any mood disorder</td>
<td>69.9</td>
</tr>
</tbody>
</table>

Kessler RC. NCS data
Proportion of anxiety and mood disorders that are comorbid (2)

Only 1/5 of anxiety cases were never comorbid. Highest comorbidity in:
- Panic
- GAD
- Agoraphobia

• Only 1/6 of mood disorder cases were never comorbid. Highest comorbidity in:
  - Bipolar I
  - Dysthymia (also personality disorder comorbidity)
How likely is first onset depression if you have a primary anxiety disorder?

Data from the early developmental study of psychopathology (EDSP) Munich.

Who’s on first?

- Anxiety disorders start earlier in life than depressive disorders (except GAD).
- In 80% of cases where there is comorbid anxiety, the anxiety disorder is the primary disorder.
- Depression is less frequently the primary disorder, usually emerging after the onset of anxiety.
- GAD and panic may be exceptions to this.

Kessler RC. NCS data (cross-sectional data based on recall)
Development of anxiety comorbidity with depression over 5 years in adolescents

Initial assessment age 14-17

- Any anxiety: 19% (1.3% pure, 17.7% comorbid)
- GAD: 13% (1.3% pure, 11.7% comorbid)
- Panic: 0.8% (0.2% pure, 0.6% comorbid)

5 years later

- Any anxiety: 19.5% (1.5% pure, 18% comorbid)
- GAD: 15.7% (3.2% pure, 12.5% comorbid)
- Panic: 0.8% (1.6% comorbid)

Numbers in bars are prevalence %

Sociodemographic associations based on 12 month prevalence (Odds Ratios)

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Pure mood disorder</th>
<th>Pure anxiety disorder</th>
<th>Comorbid mood and anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>1.8</td>
<td>2.3</td>
<td>2.9</td>
</tr>
<tr>
<td>Unmarried</td>
<td>1.5</td>
<td>NS</td>
<td>1.8</td>
</tr>
<tr>
<td>Unemployed</td>
<td>1.5</td>
<td>NS</td>
<td>1.7</td>
</tr>
<tr>
<td>Education</td>
<td>NS</td>
<td>High 1.3</td>
<td>Low 2.0</td>
</tr>
</tbody>
</table>

Predictors of Help Seeking in those with Disorders

- Younger age (<45)
- Female
- Comorbidity
- Specific diagnosis

Help seeking in the last year by those with a psychiatric disorder: Diagnosis effect

28.3

46.7

36.0

16.0

Help seeking in the last year by those with a psychiatric disorder: comorbidity effect

Comorbidity increases impairment (1)
Proportion reporting 50% or more reduction in activities in last month

Comorbidity increases impairment (2)

More than 6 days impaired in last month by diagnosis and comorbidity

GAD, 34.3
MDE, 20.8
Comorbid GAD+MDE, 47.6

Odds ratios for suicide attempts by number of diagnoses

Conclusions (1)

• All psychiatric disorders (except organic) occur in about 30% of the population.

• Therefore comorbidity is common – not just between anxiety and depression.

• In the NCS 56% of adults aged 15-54 years with a lifetime history of at least one DSM III R disorder had also experienced another mental disorder.

• Comorbidity rates will be higher when hierarchies are not used and the longer the period under study.
Conclusions (2)

• For anxiety and depression comorbidity,
  – Anxiety usually precedes depression
  – Anxiety disorders have earlier onset
    • Except GAD which has later onset (like mood disorders)
  – Eventually over 80% show comorbidity of anxiety and depression
    • Are they 2 separate disorders (anxiety/MDE)?
    • Or 3 disorders (anxiety/mixed/MDE)?
    • Or one disorder with different presentations?
Conclusions (3)

- **Comorbidity:**
  - Increases impairment and functional disability
  - Longer duration of illness
  - Increases use of health care resources
  - Increases risk of suicide attempts

- **Risk factors for comorbidity include:**
  - Female
  - Unmarried
  - Unemployed
  - Low education
  - Family history increases risk to offspring (both anxiety and MDE)
  - Severity of anxiety disorder
Pathophysiology of mood and anxiety disorders: Shared mechanisms

Glenda MacQueen, MD, PhD, FRCPC
Mood Disorders Program
Hamilton, ON
## Anxiety in patients with chronic mood disorders

### Demographic Variable

<table>
<thead>
<tr>
<th>Variable</th>
<th>No Anxiety</th>
<th>Co-Morbid Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N (% of sample)</strong></td>
<td>61 (44.2)</td>
<td>77 (55.8)</td>
</tr>
<tr>
<td><strong>Males/Females (% Males)</strong></td>
<td>24/37 (39.3)</td>
<td>20/57 (26.0)</td>
</tr>
<tr>
<td><strong>BAD Type I/II (% BAD-I)</strong></td>
<td>42/19 (68.9)</td>
<td>55/22 (71.4)</td>
</tr>
<tr>
<td><strong>Y/N Rapid Cycling (% yes)</strong></td>
<td>13/48 (21.3)</td>
<td>28/49 (36.4)</td>
</tr>
<tr>
<td><strong>Age of Onset of Depression</strong></td>
<td>23.0 (10.0)</td>
<td>19.4 (10.3)</td>
</tr>
<tr>
<td><strong>Age at assessment</strong></td>
<td>46.0 (12.7)</td>
<td>42.0 (9.1)</td>
</tr>
</tbody>
</table>

### Outcome Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>No Anxiety</th>
<th>Co-Morbid Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Euthymic/Non-Euthymic</strong></td>
<td>27/34 (44.3)</td>
<td>17/60 (22.1)</td>
</tr>
<tr>
<td><strong>Percent of Year Spent Ill</strong></td>
<td>40.2 (39.8)</td>
<td>57.3 (37.4)</td>
</tr>
<tr>
<td><strong>Illness Severity</strong></td>
<td>14.6 (17.3)</td>
<td>21.5 (18.4)</td>
</tr>
<tr>
<td><strong>Depression Severity</strong></td>
<td>12.0 (16.5)</td>
<td>20.2 (18.5)</td>
</tr>
<tr>
<td><strong>Mania/Hypomania Severity</strong></td>
<td>2.6 (6.2)</td>
<td>1.3 (3.5)</td>
</tr>
<tr>
<td><strong>Average GAF</strong></td>
<td>70.1 (6.6)</td>
<td>67.9 (6.8)</td>
</tr>
</tbody>
</table>

Boylan et al., 2004: J Clin Psychiatry
The Limbic System

cingulate

hippocampus

hypothalamus

amygdala

http://www.giccs.georgetown.edu/~ivy/10
The regions: hippocampus and amygdala

Ventral hippocampus: anxiety-related behaviours
Amygdala: fear.

McHugh et al., 2004; DeGroote & Treit, 2004
The model: factors that influence neuronal integrity

- Neuroplasticity: neuronal sprouting
- Cell loss: apoptosis (bcl2), others (grp78/94)

Factors influencing neuronal integrity:
- Genetic predisposition
- Environment
  - NE
  - 5HT
  - DA
  - GLU/GABA

Brain regions:
- PFC / Orbital
- Amygdala
- HC

Pathways:
- NMDA
- Gas
- AC
- cAMP
- PKA
- CaMK
- Ca^2+
- CREB
- c-Fos, c-Jun

Gene expression:
- Neuroplasticity: protective/adaptive
- Cell loss: progressive
Patient Studies

- Postmortem brain
- Cognitive Testing
- Neuroimaging
Temporal cortex CREB levels in patients with depression (Dowlatshahi et al., Lancet 1998)

43 kDa

<table>
<thead>
<tr>
<th>CTL</th>
<th>MDD</th>
<th>BD</th>
<th>SCZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.5</td>
<td>1</td>
<td>1.5</td>
</tr>
</tbody>
</table>

Relative Immunoreactivity

Antidepressant treatment

CTL: n=15, MDD: n=5, BD: n=7, SCZ: n=9

BDNF in post mortem samples of patients

- Hilus
  - AD (+), n = 19
  - AD (-), n = 31

- DG
  - BDNF immunoreactivity

- SG1
  - BDNF immunoreactivity

- SG2
  - BDNF immunoreactivity

- MDD
  - BDNF immunoreactivity

- BD
  - BDNF immunoreactivity

- SCZ
  - BDNF immunoreactivity
Patient Studies

- Postmortem brain
- Cognitive assessment
- Brain imaging
Habit and Recollection Memory

• HABCON
  – assesses habit and recollection memory in addition to guessing scores

Two phases:
  habit creation
  effortful memorization
  distractor task
  recollection testing

Process dissociation task

King - re___
Bridge - ____er
Barn - __ar__
Table - __l_t__
Memory in the first episode of depression

![Bar chart showing memory performance in different tasks (Guess, Habit, Recollect) for Control, ME, Control, and FE groups.](chart.png)
## PTSD and recollection memory

### Impaired
- 73 rape victims, 92% with PTSD: Nixon et al., 2004
- Holocaust survivors: Yehuda et al., 2004
- Patients with PTSD: Vermetten et al., 2003
- Traumatized refugees from the former Yugoslavia: Kivling-Boden et al., 2003
- Women with early childhood sexual abuse-related PTSD: Bremenr et al., 2003

### Not Impaired
- Prepubescently abused women: Pederson et al., 2004
- Combat veterans: Neylen et al., 2004
- Early middle-aged community dwelling veterans: Crowell et al., 2002
- IPV; with/out PTSD: Stein et al., 2002
Adolescents at high risk for mood disorders

- **Low-Risk Control**
- **High-Risk: Unaffected**
- **High Risk: Affected**

The graph shows the percentage errors and millisecond response times for different time intervals (ISI) between target and mask. The data points indicate varying levels of performance across the risk groups.
Practical tips

- Traditional neuropsych may be normal: it may be turned against your patients
- BZP may negatively influence performance
- ADs may improve performance – especially with long term treatment
- Euthymic patients may be accurate in their assessment of function
- These are stress sensitive illnesses
Patient Studies

- Postmortem brain
- Neuropsych Testing
- Brain imaging
Relation of HC volumes to length of illness

MacQueen et al., 2004
Meta analysis of studies examining HC volume in depression

Campbell et al, 2004, AJP
Impact of anxiety disorders on HC volume

- Association between PTSD in trauma exposed twin and HC volume of unaffected twin
- Genetic vulnerability to small HC predisposing to PTSD following trauma
Vithilingan found that women with depression and early abuse had hippocampal volumes that were almost 20% smaller than women without a history of abuse.
Consequences of early adverse experience

Male C57BL/6 mice that undergo maternal separation early in life demonstrate higher levels of anxiety upon reaching adulthood compared to normally reared offspring.

- noradrenaline in limbic regions
- 5HIAA levels in frontal cortex and hippocampus

Romeo et al., 2004; Daniels et al., 2004
What about antidepressants?
Serotonin may increase dopamine and acetylcholine levels in the brain.

Serotonin Receptor Concentration
Meltzer et al. from Lieberman
Early intervention with antidepressants

- MatS+DMI
- MatS+Sal
- NonS+DMI
- NonS+Sal

MacQueen et al., 2003
Hippocampus

Genetics, early experiences

Normal
Normal Survival and Growth

Stress
↑ Glucocorticoids
↓ BDNF
Atrophy or Death of Neurons

ADs
↑ Serotonin and NE
Increased Survival and Growth

Other Neuronal Insults

<table>
<thead>
<tr>
<th>Hypoxia-ischemia</th>
<th>Hypoglycemia</th>
<th>Neurotoxins</th>
<th>Viruses</th>
</tr>
</thead>
</table>

Viruses, Genetics, early experiences, Hypoxia-ischemia, Hypoglycemia, Neurotoxins.
What’s the point?

- Anxiety and mood disorders appear to share a number of pathophysiological processes.
- Failure to recognize and treat these illnesses may lead to worsening of underlying structural changes.
Topics of presentation

- Cognitive therapy treatment effectiveness summary
- Depression versus medication comparison
- CBT theory of depression and anxiety
- Cognitive and behavioral intervention strategies
- CBT active ingredients for depression and anxiety disorders
- Comorbidity
Butler, A.C., & Beck, J. S. (2000) reviewed 14 meta-analyses that investigated the efficacy of CT. These analyses covered 9,138 subjects and involved 465 comparisons regarding 14 disorders or populations.
## Effect size categorization

<table>
<thead>
<tr>
<th>Classification</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>no effect</td>
<td>= 0 to 0.2</td>
</tr>
<tr>
<td>low effect</td>
<td>= 0.2 to 0.5</td>
</tr>
<tr>
<td>medium effect</td>
<td>= 0.5 to 0.8</td>
</tr>
<tr>
<td>high effect</td>
<td>= greater than 0.8</td>
</tr>
</tbody>
</table>
**SUMMARY OF META-ANALYTIC FINDINGS**

Comparisons of cognitive therapy to no-treatment, wait list, and placebo controls.

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Average effect size</th>
<th>% of CT patients superior to controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult unipolar depression</td>
<td>.82</td>
<td>79%</td>
</tr>
<tr>
<td>Adolescent unipolar depression</td>
<td>1.11</td>
<td>87%</td>
</tr>
<tr>
<td>Generalized anxiety disorder</td>
<td>1.04</td>
<td>85%</td>
</tr>
<tr>
<td>Panic disorder with or without agoraphobia</td>
<td>.91</td>
<td>82%</td>
</tr>
<tr>
<td>Social phobia</td>
<td>.93</td>
<td>82%</td>
</tr>
<tr>
<td>Childhood depression and anxiety disorders</td>
<td>.90</td>
<td>82%</td>
</tr>
<tr>
<td>Marital distress</td>
<td>.71</td>
<td>76%</td>
</tr>
<tr>
<td>Anger</td>
<td>.70</td>
<td>76%</td>
</tr>
<tr>
<td>Childhood somatic disorders</td>
<td>.47</td>
<td>68%</td>
</tr>
<tr>
<td>Chronic pain (not headache)</td>
<td>.46</td>
<td>68%</td>
</tr>
</tbody>
</table>

Depression treatment response following medication or cognitive therapy
(R.J. DeRubeis et al, 1999)
Comparisons of cognitive therapy with alternative treatments

Cognitive therapy versus antidepressant medication:

Cognitive therapy was somewhat superior to antidepressant medications in the treatment of adult unipolar depression (average effect size = 0.38).

Importantly, one year after treatment discontinuation, depressed patients who had been treated with cognitive therapy had half the relapse rate of depressed patients who had been treated with antidepressant medication (30% versus 60%).

CBT theory of depression

- Depressed patients hold unrealistically negative views about themselves, others, and the world (cognitive triad)

- Maladaptive, systematic distortions in information processing
  - bias in cognitive processing of information
  - unbidden negative ruminations
  - attentional bias
“I thought I was falling into a vast, deep, dark pit of despair but it was only a pothole.”
CBT theory of anxiety disorders

- Anxious individuals tend to endorse automatic thoughts related to harm and danger (i.e., bias in cognitive processing of information)
  - over-prediction of fear
  - attentional bias
  - anxiety sensitivity
Model for conceptualizing anxiety disorders

Anxiety = \frac{Overestimation of Danger}{Underestimation of Coping/Resources}
Cognitive behavioral interventions

**Cognitive interventions**

- In cognitive therapy patients are encouraged to treat their beliefs as hypotheses to be tested.
- They are trained to conduct behavioral experiments to test the accuracy of their beliefs.

(Beck, 1991)

**Behavioral interventions**

- Depression – *Behavioral Activation* - reengagement in constructive and rewarding activities
- Anxiety disorders – *Exposure* - Deliberately come into contact with the cues that evoke anxiety, and remain in contact with those cues until the anxiety is reduced (habituation)
Professor Gallagher and his controversial technique of simultaneously confronting the fear of heights, snakes, and the dark.
CT active ingredients

- structure in the therapy session

- use of concrete cognitive and behavioral techniques
  (Feeley et al., 1999)

- homework compliance
  (Burns and Spangler, 2000)
## Thought record
(Greenberger, Padesky, 1995)

<table>
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<tr>
<th></th>
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<tr>
<td>In the bosses office being instructed on the use of new computer software</td>
<td>Overwhelmed 95%</td>
<td>I’ll never understand this.</td>
<td>I was never very good at school.</td>
<td>At work, I learned the computer filing system, which is complicated.</td>
<td>Even though this seems complicated now, I’ve learned other complicated things in the past. With practice it might get easier. 90%</td>
<td>Overwhelmed (40%)</td>
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<td></td>
<td>Depressed (85%)</td>
<td>This is too complicated for me to learn.</td>
<td>I listen to my boss explain the new software and I get confused.</td>
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<td></td>
<td>Depressed (60%)</td>
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<td></td>
<td></td>
<td>I’m doomed to always be depressed</td>
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What changes are effective?

- The ability to be less extreme and more balanced in information processing styles (Note: Patients who became unrealistically positive were at greater risk for relapse)

- This change in the attributional style mediates CT’s relapse prevention effect (Hollon, Evans, & DeRubeis, 1990)

- Patients treated to remission with CT were about half as likely to experience a depressive relapse following treatment termination as patients treated to remission with medications (Hollon & Shelton, 2001)
CT active ingredients for anxiety disorders

- Agoraphobic disability has a significant bearing on panic treatment effectiveness (Williams and Falbo, 1996)
  - 94% of low-agoraphobic Ss panic free after treatment
  - 52% of high-agoraphobic Ss panic free after treatment

- CT dealing with catastrophic misinterpretations of bodily sensations is highly effective in reducing panic attacks but does not automatically lead to an abandonment of agoraphobic avoidance behavior (Van den Hout, van Oppen et al., 1994)

- Exposure in vivo (and interoceptive) is superior to CT for panic and agoraphobia (Williams and Falbo, 1996)

- Cognitive therapy was more effective than applied relaxation in reducing the number of panic attacks (Ost and Westling, 1995)
Social anxiety disorder

• Hope, Heimberg, and Bruch, 1995
  – 70% of patients treated with exposure were rated as clinically improved compared with
  – 36% of patients treated with cognitive behavior group therapy rated as clinically improved

• Exposure in vivo recommended for both generalized and specific (e.g., fear of public speaking, writing, blushing) social anxiety disorder
Obsessive compulsive disorder

- Both exposure (in vivo) to distressing stimuli and response prevention of the compulsive ritual are essential components (Abramowitz, 1997; Emmelkamp, 1994)

- Gradual exposure in vivo (with response prevention) is as effective as flooding in vivo (Emmelkamp, 1994)
Comorbidity

• Does treatment for a principal anxiety disorder have an impact on comorbid conditions?
The effects of cognitive-behavior therapy for panic disorder on comorbid conditions
(Tsao, Lewin, Craske, 1998)

**Fig. 4. Anxiety Disorders Interview Schedule-Revised severity ratings for comorbid conditions at pre- and posttreatment.**
Comorbid anxiety and mood disorders with social anxiety disorder

- Examined the differential effects of comorbid mood and anxiety disorders on
  - symptom severity
  - outcome of CBT for social anxiety disorder and
  - maintenance of treatment gains a year later

- Ss with comorbid mood disorder had greater symptom severity than Ss with comorbid anxiety disorders

- Findings:
  - After 12 weeks of CBT, all groups evidenced near parallel improvement
  - All comorbid groups reported reductions in depression, suggesting that depression improves with CBT for social anxiety disorder
  - 12 months after treatment, the groups maintained parallel improvement.

(Erwin, Heimberg, Juster, Mindlin, 2002)
Comorbidity (con’t)

• Improvement rates in
  – panic disorder (McLean et al., 1998; Tsao et al., 2002) and
  – PTSD (Blanchard et al., 2000; Tarrier et al., 2000) for patients with comorbid depression are similar to those of their nondepressed counterparts
  – Following treatment, nondepressed OCD patients evidenced less severe OCD symptoms than patients with comorbid MDD; however, patients with co-morbid depression still made considerable treatment gains (Abramowitz and Foa, 2000)
### Mean Y-BOCS scores for OCD patients with and without MDD at pre- and post-treatment and at follow-up (Abramowitz and Foa, 2000)

<table>
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<tr>
<th>Assessment</th>
<th>OCD with MDD</th>
<th>OCD without MDD</th>
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<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
</tr>
<tr>
<td>Pre-treatment</td>
<td>28.51 (8.86)</td>
<td>26.26 (4.30)</td>
</tr>
<tr>
<td>Post-treatment</td>
<td>13.60 (9.23)</td>
<td>8.40 (5.59)</td>
</tr>
<tr>
<td>Follow-up</td>
<td>13.70 (6.60)</td>
<td>8.11 (5.90)</td>
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Abramowitz and Foa, 2000
Merci
Faculty Question Period