INTRODUCTION

1. Recognition, Differential Diagnosis & Etiology of Social Anxiety Disorder (SAD)
2. Shame Overview
   a. Recognition
   b. Differential Diagnosis
3. Deconstructing SAD (Two Parts)
   a. Differentiating Shame & Anxiety
   b. Temperament: Neuroticism, Extroversion & SAD
4. Treatment
   a. Pharmacologic
   b. Non-pharmacologic: 1) Cognitive behavioral  2) Relational

INTRODUCTION (3)

TIME    TOPIC
9:00 AM to 10:30 AM Recognition and Diagnosis
10:30 AM to 10:45 AM Break
10:45 AM to 12:00 Noon Shame
12:00 Noon to 1:00 PM Lunch
1:00 PM to 2:30 PM Deconstructing SAD
2:30 PM to 2:45 PM Break
2:45 PM to 4:15 PM Treatment
4:15 PM to 4:30 PM Summary and Evaluations

EVERYTHING YOU ALWAYS WANTED TO KNOW ABOUT RECOGNIZING SOCIAL ANXIETY DISORDER BUT WERE TOO EMBARRASSED TO ASK

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RECOGNITION, DIFFERENTIAL DIAGNOSIS, AND ETIOLOGY

19C: Sunday, March 9, 9:00am - 12 pm
Presented at the ADAA 28th Annual Conference
DEFINITION

1. Synonyms:
   Social Anxiety Disorder (SAD) = Social Phobia (SP).

2. Core Issues:
   Fear of scrutiny and scorn.

3. Simple Description:
   Fear and Avoidance of Social and Performance situations.

COMPLEX DEFINITION (1)

1. Anticipatory anxiety of being scrutinized and scorned which either:
   a. Interferes with functioning, or
   b. results in intense distress.

COMPLEX DEFINITION (2)

2) A situationally bound Panic Attack may occur.

3) Subtypes:
   a. Non-generalized (or “Specific”) = Recurrent and distinct; usually performance related (I.E., A phobia of being observed.).
   b. Generalized = Persistent and global; includes both performance and interpersonal situations (I.E., Intense shyness.).

DSM-IV

Common Somatic Complaints Of Social Anxiety Disorder

Stuttering  ...  Blushing
Palpitations  ...  Sweating
"Butterflies"  ...  Trembling And Shaking

FEAR OF POSITIVE EVALUATION (FPE)

1. Some SAD patients experience increased anxiety after a positive evaluation of their performance. They worry that it will raise the social standard by which they will be evaluated in the future, and they don’t believe that their typical performance will improve.

2. Fear increased status could lead to conflict with more powerful others (c/w both Oedipal and Evolutionary Models).

3. FPE accounts for the “disqualifying the positive” thinking error.

4. Perhaps the fear of evaluation in general, rather than the fear of negative evaluation specifically, is at the core of SAD.


PREVALENCE

2005 NCS-R Data

NCS-R HIGHLIGHTS: Focus on SAD

I. The lifetime prevalence of the most common mental disorders:

1. Categories: Anxiety Disorders 29 %; Mood Disorders 21 %.
2. Individual Disorders: Major Depression 16.6 %; Alcohol Abuse 13.2 %; Specific Phobia 12.5 %; SAD 12.1 %.

II. The comorbidity between Anxiety and Mood Disorders is higher than the comorbidity amongst Anxiety Disorders.

III. Anxiety Disorders are usually primary with respect to onset.

IV. 50 % of SP’s have onset by age 13; 90 % by age 23.

Kessler et al, 2005

COURSE

The course is typically chronic or lifelong.

DSM-IV-TR, 2000; Wittchen et al, 1999

MORBIDITY

1. Educational and Occupational
2. Economic
3. Cardiac

Social Anxiety Disorder: Educational And Occupational Impairment

* LSAS score is controls = 25; ** impairment (%) refers to percentage change in wages and percentage point change in probabilities of college graduation and having a technical, professional, or managerial job.

Katzelnick et al. Presented at 37th Annual Meeting of the American College of Neuropsychopharmacology; December 14-18, 1998; Los Croabas, Puerto Rico.

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1. Phobic anxiety, GAD, panic disorder, and worry are all predictors of MI and/or cardiac death.
2. Anxiety is more highly associated with mortality in CHD than elevated cholesterol.
3. SAD is associated with decreased heart rate variability, (reflects cardiac autonomic imbalance) and hence increased risk of sudden cardiac death.
4. Social anxiousness is highly predictive of CHD development.

Mechanisms: Increased HR, HRV, HTN, Cortisol, Epi, Poor compliance.

Todres JF et al. 2003
Coryell et al. Arch Gen Psychiatry. 1990;47:994-1000.

CAN YOU BE SCARED TO DEATH?

Among those who remembered.

Wanted to die
Wanted to commit suicide
Suicide attempts

Thought a lot about death

*In >80% of cases.
* SAD was present at an earlier age (or in the same year) than the comorbid diagnosis

SUBTYPES

1. Generalized:
   Social interactional and performance anxiety in most situations.

2. Non-generalized (Specific):
   Performance anxiety in one or two situations; or less commonly, social interactional anxiety in one or two situations.

Potential for Suicidal Ideation in Social Anxiety Disorder

Spectrum of Social Discomfort

NON-GENERALIZED
(SPECIFIC or PERFORMANCE)
SUBTYPE

GENERALIZED
(SOCIAL INTERACTIONAL & PERFORMANCE)
SUBTYPE
OFFENSIVE SUBTYPE: TAIJIN KYOFUSHO (TKO)

1) Definition of TKO: Fear of experiencing anxiety in the presence of others (Japan).
2) Comparison with SAD: Fear is of offending others in addition to the fear of embarrassing the self. (“Offensive” Subtype?),
   e.g., Stiff facial expression; Unpleasant body odor; Inappropriate staring; Displaying physical defects.
3) Differing cultural influences on the view of the self may explain the differences in social fear focus:
   Japan (Collectivist): Self seen as primarily interdependent
   USA (Individualistic): Self seen as primarily independent.

DIFFERING EAST/WEST METAPHORS

East: “The nail that protrudes gets hammered down.”

West: “The wheel that squeaks the loudest is the one that gets the grease.”

Los Angeles Times, 1994

DIFFERENTIAL DIAGNOSIS OF ANXIETY

Cued panic attacks
- Social phobia
- OCD
- PTSD
- Specific phobias
- Unexpected
- GAD
- Panic disorder

General medical disorders

Differential Diagnosis

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DIFFERENTIAL DIAGNOSIS

1) Major Depression; Atypical Depression
2) Panic Disorder and Agoraphobia
3) Body Dysmorphic Disorder
4) Paranoia
5) Avoidant, Dependent and Schizoid (Autism?) PD’s

Lubowitz et al, 1999

AUTISM AND SAD

1. Among 96 1st degree relatives in 36 families having an autistic child, SAD was found in 20.2% vs. 2.4% in not ill controls.
2. Parents of autistic probands had higher rates of SAD than parents of Down’s Syndrome probands: 14.6% vs. 3.3%.
3. Positive correlation between 5-HTTLPR polymorphism and autism.

1. Smalley SL et al, 1995
2. Piven J, Palmer P, 1999

ETIOLOGY:

ENVIRONMENTAL

CHILDREARING EFFECTS (1)

1. SAD & Avoidant PD patients retrospectively report:
   a. Hypocritical, shaming parents.
   b. Humiliating experiences at school or home.
2. SAD patients report more overprotection and less warmth from their parents than panic or agoraphobia patients.
3. Parental overprotection conveys messages to child that:
   a. The world is a dangerous place.
   b. The child is unable to cope.

1. Arrindel, 1989; Stravynski, 1989; Arbel, 1991
2. Rapee & Melville, 1997
3. Hudson & Rapee, 2000

CHILDREARING EFFECTS (2)

1. Using normal mother infant dyads, socially anxious and socially confident mother-stranger interactions were staged:
   Infants were significantly more fearful and avoidant of the stranger after experiencing the staged socially anxious mother-stranger interaction (modeling?).
2. The degree of infant social unresponsiveness to a stranger was predicted by the degree that Social Phobic mothers (as compared to normal and GAD mothers) encouraged (I.E., low encouragement) their infants to interact.

1. De Rosny, 2006

CONDITIONED FEAR ETIOLOGY

1. High frequency of retrospectively reported traumatic conditioning in SAD patients.
2. No evidence for increased conditionability to generally aversive (but socially nonspecific) stimuli in SAD patients.


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1. Elevated levels of conditioned fear-potentiated startle found in SAD patients vs healthy controls using social conditioning paradigm that conditioned neutral facial expressions (CS) paired with critical facial and verbal feedback (UCS).

2. Fear-potentiated startle in response to the negative conditioned stimulus increased as SAD patients’ scores on the Liebowitz SAD Scale increased.

COGNITIVE PROCESS

1. Negative bias in the perception of their own but not of others’ performance.

2. Over-allocation of attentional resources to:
   a. External social threat (Stroop test).
   b. Focusing on the self during social interactions (i.e., “watching self”).

BARLOW’S SAD MODEL

1. Expectations of social interaction or performance elicit:

2. An autonomic arousal driven dysfunctional emotional and cognitive process

3. Involving maladaptive shifts of attention (process) and a sense of uncontrollability (belief).

BARLOW’S MODEL OF INHIBITED SEXUAL EXCITEMENT

ETIOLOGY:

BIOLOGIC
Symptoms of Anxiety & Depressive Disorders Overlap

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<tr>
<th>SAD</th>
<th>GAD</th>
<th>MDD</th>
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<tbody>
<tr>
<td>Fear/avoidance</td>
<td>Difficulty 2/3</td>
<td>Guilt/Self-criticism</td>
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<td>of social &amp;</td>
<td>Inability</td>
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<td>performance situations</td>
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<td>Blushing</td>
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<td>Suicidal ideation</td>
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Symptoms of Anxiety & Depressive Disorders Overlap

Low Extroversion (E) & High Neuroticism (N) as Indices of Genetic & Environmental Risk for SAD, Agoraphobia (A), and Animal Phobia (AP)

Lifetime phobias and N and E assessed in 7,800 twins via clinician interview and self-report questionnaire.

1. The familial co-occurrence of SAD and A is entirely explained by genetic, not shared environmental factors.
2. Genetic liability for SAD & A is completely (100%) accounted for by the genetic factors that determine both E (moderate) and N (high).
3. Most of the genetic risk for AP’s is not accounted for by E (0%) and N (16%); it probably involves preparedness.


Low Extroversion (E) & High Neuroticism (N) as Indices of Genetic & Environmental Risk for SAD, Agoraphobia (A), and Animal Phobia (AP)

BEHAVIORAL INHIBITION (BI)

BI to the unfamiliar = withdrawn, passive, avoidant behavior in novel situations or with strangers.

(BI, along with Trait Anxiety and Harm Avoidance, combine aspects of both Extroversion and Neuroticism into one dimension.).

3) Turner and Beidel, 1996

BEHAVIORAL INHIBITION (BI) AND SAD

1) BI children develop higher rates of SAD than non-BI children.
2) Parents of BI children have more SAD than parents of non-BI children.
3) BI is most closely associated with disorders characterized by social evaluative anxiety. BI is familial and likely genetic.

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EVOLUTIONARY MODEL (1)

1. Evolved attachment and defense systems.
2. Defense system copes with 2 main threats:
   a. Predators.
   b. Conspecifics (Members of the same species.).
3. Biological preparedness:
   Epidemiologic over-representation of phobias of snakes, closed spaces, the dark, strangers, places of restricted escape.
**EVOLUTIONARY MODEL (2)**

1. Primates are biologically prepared (i.e., hardwired) to fear dominant angry faces (Dominant animals use angry facial expressions during ritualized, combative displays.).

2. Humans infants show superior conditioning (i.e., resistance to extinction) to angry faces vs. happy and neutral faces.

3. Social anxiety promotes peaceful organization of primate social systems by the use of submissive (e.g., gaze aversion, appeasing smiles) or threatening (e.g., angry, contemptuous facial expressions) gestures.

4. SAD patients probably have too low a threshold for eliciting submissive behavior, i.e., a “social ranking system disorder.”


**ATTACHMENT THEORY**

A. An integration of ideas from Evolutionary Biology (e.g., imprinting) with Object Relations Theory.

B. Secure attachment style:
   Have trusting, intimate relationships.

C. Insecure attachment styles:
   1. Anxious:
      a. Value relationships over autonomy.
      b. Prone to abandonment worry, jealousy & hyper vigilance.
   2. Avoidant:
      a. Deny the importance of relationships; others untrustworthy.
      b. Prone to value rationality over emotions.

Bowlby, 1982

**ATTACHMENT AND SOCIAL RANK**

1. 140 healthy adolescents assessed with Attachment, Social Rank, Submissive Behavior, Anxiety and Depression scales:

   Attachment:  | Secure | Insecure |
   Social Comparison  | +      | -       |
   Depression/Anxiety | -      | +       |
   Submissive Behavior | -      | +       |

2. Insecure attachment sensitizes individuals:
   A. To be more social rank and threat sensitive.
   B. To defend with avoidant and submissive strategies.

Irons & Gilbert, 2005
### VOLE ATTACHMENT NEUROBIOLOGY

1. Monogamous Prairie (not Montane) Vole partner preference formation depends on:
   a. Oxytocin (OT) in females.
   b. Vasopressin (AVP) in males.

2. Human brain oxytocin:
   a. Modulates social behaviors: maternal care, aggression, pair bonding, sexual behavior, social memory & support, and trust.
   b. Downregulates: stress responses, including anxiety.

### HUMAN ATTACHMENT NEUROBIOLOGY

1. OT and AVP are associated with both stress regulation and social bonding.

2. The basic neuroendocrine response to distress begins with a rapid release of OT, AVP and CRF from the Paraventricular N. of the Hypothalamus (AVP & CRF -> ACTH release.).

3. OT receptors are part of the neural reward circuit that includes the N. Accumbens (This circuit likely confers a sense of security and protection that makes interactions rewarding in the developing infant; prairie voles have high levels of OT receptors in the N. Accumbens.).

### OXYTOCIN (OT) STUDIES

1. Intranasal OT specifically promotes trust & prosocial behavior in humans: In Game Theory studies OT increased interpersonal risk taking.

2. fMRI Study: Intranasal OT vs placebo dampened Amygdala responsivity to social (threatening faces, i.e., angry or fearful) > nonsocial (threatening scenes) stimuli.

3. 4½ year old children reared by parents evinced an increase in OT levels after interaction with their mothers, whereas institutionally reared children (3 years post adoption) did not.

### SUMMARY

1. SAD patients excessively fear Scrutiny and Scorn.

2. SAD, the 4th most common mental disorder, has an early onset, is chronic, and has significant morbidity associated with it.

3. SAD is highly co-morbid, the rate of suicide attempts increases 16 X in co-morbid SAD.

4. Etiologic theories include Cognitive, Evolutionary, Attachment, and Genetic.

5. The majority of genetic variance in SAD is accounted for by a common genetic factor shared with other internalizing disorders.