PSY 210- Abnormal Psychology
Mercer County Community College
Fall 2007

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Textbook: **Durand and Barlow (2006) Essentials of Abnormal Psychology (4th ed.)**

Welcome to Abnormal Psychology!

This class will explore that fascinating world of abnormal behavior…but what is that exactly? By the end of this course you WILL be able to answer that question!
We will focus on the elements of psychopathology and mental disorders, as well as the history and classifications of various disorders.

This class will discuss a variety mental illnesses, such as, depression, schizophrenia, bipolar disorder, PTSD, anxiety disorders, eating disorders, personality disorders and other intriguing ailments that so many people endure on a daily basis; Posing the question, "How abnormal is it really?" This class is great for anyone interested in understanding those "abnormal" behaviors seen very often within our society. This class will also examine the tremendous impact mental illness has on the person living with abnormality.

When I tell people that I work as a therapist, I am usually bombarded with stories like, "I know a guy who sees little green aliens"… "My sister has depression and is on medication… "I think my neighbor is crazy, he stares at the side of his house all day long and talks to his car…what do *YOU* think?"

When examining the behavior of another, we can see many things both normal and abnormal. This is not to think everyone has a diagnosable disorder! As this class progresses, you will learn many tell-tale indicators of mental illness and psychopathology.

It is a common practice for us to enjoy analyzing people and placing them into neat little categories, but please refrain from diagnosing yourself, your friends, your parents and family, your classmates, your professors or anyone else ☺

This class will be interesting and fun, but it will not make you a psychologist!

Academic Integrity
The work a student produces must be their own and should result solely from their own efforts. Plagiarism or cheating on any assignment is regarded as an extremely serious academic offense. Student's who violate this policy will receive an "F" for the course. Please refer to the Mercer County Community College Academic Integrity Handbook, or request one from the professor.

Academic Accommodation
It is the student's responsibility to arrange an accommodation. If you are a student with a disability or special need, please advise the professor so that appropriate accommodations can be made

Attendance and Conduct
As college students you choose to be here. You have elected to register for this course and **YOU** will determine your overall experience in this class. Although I do not expect students to attend every class due to demands of life, attendance is very important and required. If you miss class, it is **YOUR RESPONSIBILITY** to get the information you have missed. Do not expect or request the notes to be supplied by me if you did not attend lecture. Attendance and lecture notes are crucial to success in this class.

Missing 6 or more classes will result in the lowering of your final grade by one letter grade.

Without question, students who attend class perform better than those who do not…make sure you get to class. If you are often absent, it should lead you to ask the question, "**Why did I register for this course**?"

You will not hurt my feelings if this class is not for you, but please don't just disappear- **drop** the class as soon as you realize you don't want to be here. If you choose to not return without officially dropping the course, you will receive an "**F**" on your transcript.

Attendance Bonus
Students who attend every class will receive a **4 point bonus**. If you miss only one class, you will receive a **2 point bonus**. Lateness is not acceptable from college students; therefore two lates will be equal to one absence. If you arrive to class after I have taken

role, it is **YOUR** responsibility to advise me that you arrived late, otherwise you will be marked as absent. All absences without a doctor's note will be considered unexcused. It is at my discretion to add additional points for active participation.

Exams

Your final grade will be based on **four** 50 question multiple-choice exams. All exams will be administered in the testing center. The exams are available at the **West Windsor Testing Center ONLY (LA 215).** Please check the hours of operation to ensure you arrive during testing hours. All students MUST provide a current student ID to take the exams and must know the course number, test number and instructor's name.

Each exam will be available for a 7- day period (review course schedule for dates). It is the **student's responsibility** to take the exam within the allotted amount of time; therefore failure to complete the exam will result in a zero. Make-up exams are not an option in this course. Should you miss an exam, you will have the option of taking EXAM 6, a cumulative exam final, which will replace your lowest exam grade.

Retest policy:

Each student will have the opportunity to retake each exam. Students may retake the exam at any time during the 7 day testing period. The higher grade will be calculated towards the final grade. NO retests will be possible after the exam period has expired.

For example:

Exam #1 (exam period) Sept. 1-8th

- Ask testing center for PSY 101/**Exam 1A**/Jennings

Retest Exam #1 (exam period) Sept. 1-8th

- Ask testing center for PSY 101/**Exam 1B**/Jennings

All students are welcome to come to my office hours and review version A to discuss any questions about the exam before taking the retest. Take advantage of this opportunity!

West Windsor Campus

Fall and Spring			
During the **first two weeks** of each semester		**After the second week** of each semester	
Monday - Thursday	10:30 a.m. - 8 p.m.	Monday – Thursday	9 a.m. - 8:30 p.m.
Friday	9 a.m. - 4 p.m.	Friday	9 a.m. - 4 p.m.
Saturday	9 a.m. - 3:30 p.m.	Saturday	9 a.m. - 3:30 p.m.

For further information go to the testing center on the college's website:
http://www.mccc.edu/student_services_testing.shtml

Position Paper
Each student will be required to submit a position paper. This is a 5-page (minimum) type-written position paper. Details for this assignment are provided in a separate handout at the end of the syllabus. This assignment will be worth **50 points**. Please refer to the handout for details, grading criteria and deadline for this assignment.

(3) Analytical writing assignments
Each student must submit 3 short writing assignments, for a total of **50 points. Each student must submit (1) Film Critique, (1) Case summary and (1) Article Review.** Each writing option will have individual instructions and due date attached below.

Extra Credit Option
Students will have the option of submitting an additional one page, type-written journal summary paper (Instructions included at the end of the syllabus). This paper can be submitted at any time during the semester however, it MUST be submitted before the end of the exam 3 testing period. This paper will be worth **10 extra credit points**.

To encourage good study habits, I will give 4 extra credit points per exam to students who make flash cards of the chapter material. It may not sound like much, but that adds up to **20 extra credits points** which will be added to your overall final points. Take advantage of this opportunity!

Course Grading
Your final grade for this class will be calculated on a point system. Your grade will be based on exam totals for a **total of 300 possible points for the course.**
Use the space below to keep track of your cumulative points from the exams, paper, writing assignments and extra credit.

EXAM 1 _____ pts.	FLASHCARDS EXAM 1 _____ pts.
EXAM 2 _____pts.	FLASHCARDS EXAM 2 _____ pts.
EXAM 3 _____pts.	FLASHCARDS EXAM 3 _____ pts.
EXAM 4 _____pts.	FLASHCARDS EXAM 4 _____ pts.
Total Points _____	Total Points _____

Total Exam Points	/200 points
Position Paper	/50 points
Film Critique	/ 15 points
Journal Review	/ 10points
Case Summary	/ 25 points
Total Flash Card Points	/ 20 points
Extra Credit Paper	/ 10 points
Attendance Bonus	Points
TOTAL EARNED POINTS	/300 points

The final grades can be computed as follows:

Points	Letter Grade	Percentage
279 +	A	93%
270	A-	90%
261	B+	87%
249	B	83%
240	B-	80%
231	C+	77%
210	C	70%
180	D	60%
Below 180	F	

All assignments and exams must be completed by the due date listed below in the course schedule. Therefore, if you want any extra credit in this course YOU MUST PLAN AHEAD. No late assignments will be accepted for any reason (unless accompanied with a "late pass" attached below).

I sincerely hope that you find this subject to be interesting and enjoy this psychology course. It is my goal for each of you to successfully learn in this class, as well as, think critically about issues related to Abnormal Psychology. Please feel free to contact me at any time during the semester in class, during office hours, by phone or email with any questions.

Please cut and staple the "late pass" to the late assignment before turning in your one late assignment

PSY 101 Introduction to Psychology Fall, 2007		"LATE PASS" Name: _____ Assignment and Bingo Board Box: _____ Original Assignment Due Date: _____ Date submitted: _____

Course Schedule:

Week of:	Course material	Exam period	Assignment Due Dates
August 27	Ch.1-Abnormal Psychology in Historical Context		
September 3rd	Ch.3- Clinical Assessment, Diagnosis and Research Methods		
September 10th	Ch.4-Anxiety Disorders	Exam 1 September 10-17th (Ch.1 and 3)	September 13th *Section Zero* (position paper) due
September 24th	Ch. 5- Somatoform and Dissociative Disorders		September 27th Journal Review Due
October 8th	Ch. 6- Mood Disorders	Exam 2 October 8-15th (Ch.4 and 5)	
October 22nd	Ch.8- Eating and Sleep Disorders		October 25th *Bibliography Summary* due
November 5th	Ch. 9- Sexual and Gender Identity Disorders	Exam 3 November 5-12th (Ch.6 and 8)	November 8th Position Paper due
November 12th	Ch.12-Schiozphrenia and related psychotic disorders		
November 26th	Ch.11-Personality Disorders	Exam 4 December 5-12th (Ch. 9, 11 and 12) Exam 5- OPTIONAL make-up exam December 5-12th (Cumulative-all chapters!)	November 26th Case Summary Due
December 11th	Final class!		

Abnormal Psychology (PSY 210)
Position Paper
Professor Jennings
50 points

This assignment requires each student to write a research paper that combines critical thinking with research skills. Once you decide on the topic you are committed to analyzing that specific area. This assignment will require each student to form a position on a topic and provide research to support their position. Regardless of which topic you select, there was something about it that interested you- now develop a position and support it with empirical research.

I am looking for a paper that examines and analyzes an element of abnormal psychology- with purpose. This paper is much like an argument; each paper must have a position or stance and be supported with research. Try to win this argument by finding information that supports what you believe. This paper should not be a platform for your personal beliefs about the topic, supported with your experiences or "case studies" that you present about people you know. Research is systematic and scientifically-based; your experiences are NOT and therefore have no place in a research paper.

I am looking for each of you to think critically and analyze your topic. Develop a position about your topic and support it with research. Learning how to analyze a topic, form a position and provide valid research will help you in future classes or colleges.

The Critical Thinking/Research paper must follow the format listed below for full credit. The thesis statement and bibliography summary MUST be submitted by specific deadlines listed below.

Section Zero: **(5 points)**

- A typed description of the topic and specific purpose of the paper must be submitted
- Topic selection with thesis statement must be submitted
- Identify the purpose of the paper, what you intend to research and prove.
- Deadline: **September 13th**

Section One **(Introduction): (5 points)**

- Identify your position
- The purpose of the paper and what you intend to discuss must be presented.

Section Two **(Discussion): (10 points)**

- Topic overview
- Introduction and discussion of the topic.

Section Three (**Research Section**)**: (15 points)**

- Application of the course material and research information.
- Academic research relevant to your topic and position must be presented.
- Scholarly journals, articles, textbook, or other resources will be presented to support the intended position of the paper.

Section Four **(Summation): (5 points)**

- Conclusion- an organized summation and conclusion of the research and material must be presented
- This section will discuss the analysis of the research and position.
- What did you find during this research? Did your research support the position?

Bibliography: **(5 points)**

- The bibliography summary must be submitted by **October 25th** for instructor review. This should be a brief summary of the article that will be included in the final version of the research paper. BRIEFLY summarize the article and why you chose to include it in your paper.
- A minimum of **FOUR** (4) academic references must appear in your paper
    - Academic references are journals and research-based material
    - The college library has an extensive database available to students- make sure you use this resource.
    - Articles from magazines such as Newsweek, Time, or Cosmopolitan are not appropriate. Psychology Today is acceptable, but not the best source for information.
- The textbook must appear in the paper.

Format guidelines: **(5 points)**

- The body of the paper must be a minimum of 5 full pages typewritten, not exceeding 7 pages in length
- 12 point Times New Roman font.
- 1" margins all around and double-spaced.
- **ALL** resources must be cited within the body of the paper as well as on the reference page
- Citations must follow APA guidelines
- Submit paper stapled with student name and class- please do not place paper in plastic binders or folders.

Very important!

- Papers submitted with citations not matching the reference page or vice versa will receive an automatic deduction of 25 points.
- Papers submitted without citations or reference page will receive an automatic failure (0 points)

Pre-approved topic list:

The following is a list of topics that are acceptable for your paper. You are limited only to these specific topics, unless permission is given.

- Anxiety Disorders (Select one ONLY)
- Bipolar Disorder
- Depression
- Dissociative Fugue
- Dissociative Identity Disorder
- Delusional Disorder
- Eating Disorders- (Select one ONLY)
- Gender Identity Disorder
- Generalized Anxiety Disorder
- Obsessive-Compulsive Disorder
- Paraphilia (Select ONE only)
- Personality Disorders- (Select one ONLY)
- Post Traumatic Stress Disorder
- Schizophrenia (Select ONE form)
- Sleep Disorders (Select one ONLY)
- Social anxiety disorder
- Social Phobia
- Somatoform Disorder (Select one ONLY)
- Specific Phobia- (Select one ONLY)

Due Date: November 8th

Good Luck!

Early submissions are welcome

Analytical Writing Assignment

Recalling and memorizing concepts about abnormal psychology is the easy part, but evaluating and analyzing concepts regarding is far more challenging. Each student must submit **3 writing assignments**, for a total of **50 points**. There are a variety of assignments you can choose from, but each student MUST select one from each category. Be sure to check the guidelines and due dates, as there are variations.

Ψ Hollywood Film Critique- (15 points)

Select one of the following films and review the film as if you were a Psychologist. You will provide a summation of the selected movie and discuss it from a psychological viewpoint. What mental illness was depicted in the film? Based on your knowledge from class and your text, determine if the film accurately depicts the disorder identified or if it is flawed. Be sure to discuss the observable behaviors that support this diagnosis.

- **Nuts** (1987 Drama; Barbara Streisand, Richard Dreyfuss; 1 hr 56 mins).
  - Why do Claudia Draper's parents and psychiatrist argue that Claudia (Barbara Streisand) should be committed? Why does Claudia resist?
  - While watching this film, did you change your mind about whether Claudia was "nuts"? If yes, at what point did you change your mind, and why?

Due September 20th

- **Girl Interrupted** (1999 Drama; Angelina Jolie, Winona Ryder, Whoopie Goldberg)
  - Susana (Winona Ryder) was committed for 18 months. Why was she committed? Does this hospitalization accurately represent psychiatric treatment in the 1960's?
  - Do you think it's useful to classify lifelong personality characteristics as psychological disorders? Do you think Susana (Winona Ryder) appeared to have Borderline Personality Disorder? Why or why not?
  - Select a character and discuss their diagnosis in the movie. Do you agree with that diagnosis, why or why not? What type of treatment would be typical today?

Due September 20th

- **As Good as it Gets** (1997; Comedy, Drama, Romance; Jack Nicholson, Helen Hunt, Greg Kinnear)
    - How did this disorder impact Melvin's (Jack Nicholson) relationships with others? How does Melvin behave when his well-established routine is interrupted? What internal feelings might lead to those behaviors?
    - What form of treatment did Melvin use in the movie? Is this a successful treatment? Explain.

    Due September 27th

- **Primal Fear** (1996 Drama/Suspense; Richard Gere, Edward Norton, Laura Linney; 129 min)
    - Why would this diagnosis acquit him from a murder charge?
    - Did the outcome surprise you? Did this change your opinion about the disorder?

    Due October 11th

- **Insomnia** (2002 Crime/Drama/Thriller; Al Pacino, Robin Williams, Hilary Swank' 1 hr. 58 min)
    - Has your ability to function ever been impaired due to lack of sleep? What signs of sleep deprivation did Will Dormer (Al Pacino) experience? What environmental, situational, and emotional factors contributed to his insomnia?
    - Do you think this film would have ended differently had the detective been able to sleep? Do you think he would have made different choices? Explain.
    - Is there anything the detective could have done to increase the likelihood of being able to sleep while in Alaska?

    Due November 8th

- **A Beautiful Mind** (2001 Drama/Mystery; Russell Crowe, Jennifer Connelly; 2hrs, 16 min).
    - What type of schizophrenia does John Nash (Russell Crowe) have? Discuss a scene that depicts the "positive symptoms" of schizophrenia. Discuss a scene that depicts the "negative symptoms."
    - Do you think the film implies that a person with schizophrenia can fully recover? Do you think the film implies that a person can recover without medication? Explain and justify your answers.

    Due November 29th

Ψ Journal Article Reviews- (10 Points) Due September 27th

Select any of the following articles and write a one-two page reflection paper. This paper must be typed (double-spaced, 12-point font, 1-inch margins). Summarize and critique the article by discussing the content and your thoughts on the article. Do you agree with the article? Why or why not? What potential problems did you find, if any You must select one of the articles listed below to review. All articles are from the journal *Current Directions in Psychological Science* and are attached in the syllabus.

Articles to choose from:

- Will They Do it Again? Predicting Sex-Offense Recidivism
- Who Develops Posttraumatic Stress Disorder?
- Attention Deficit Hyperactivity Disorder, Psychostimulants and Intolerance of Childhood Playfulness: A Tragedy in the Making?
- Math Anxiety: Personal, Educational, and Cognitive Consequences
- Recalling the Unrecallable: Should Hypnosis be Used to Recover Memories?
- Do Negative Cognitive Styles Confer Vulnerability to Depression?
- Gender Differences in Depression
- Schizophrenia: A Neurodevelopmental Perspective
- Computer and Internet-based Psychotherapy Interventions Offering
- The Role of the Family in the Course and Treatment of Bipolar Disorder

Journal Article Review Grading Criteria

An "A" level paper will receive 9-10 points:
The student writes a review of the article that displays outstanding comprehension of the content found in the article. The student also provides an analysis of the article combined with a personal reflection. The paper is insightful and provides the reader with critical thinking. The paper has less than two spelling errors, is grammatically correct and is clearly written.

A "B" level paper will receive 7-8 points:
The student follows the same procedure as above, and writes a clear and concise summary that captures the essence of the article. The student displays moderate comprehension of the article. The paper is grammatically correct, and has more than 4 spelling errors. The student provides personal insight, but less of a thorough analysis of the content.

A "C" level paper will receive 5-6 points:
The student submits a paper that meets the requirements of the assignment. The article summary was adequate, but lacked critical thought or a thorough analysis of the topic. The student has done a satisfactory job in terms of wiring, but the paper is not as clearly written as an "A" or "B" level paper.

A "D" level paper will receive 4-1 points:
The paper was superficially analyzed and lack reflection or critique. The writing quality of the assignment was below average.

No credit will be given for failing paper

Ψ Case Study- (25 Points) Due November 26th

You will review a case study provided in this packet. You will be responsible for reviewing the case and assigning multi-axial diagnoses. You will also be responsible for providing a rationale for the diagnoses, as well as a discussion of rule outs, differential diagnoses, and prognosis. This assignment should be 2-3 pages in length (typed, double-spaced, one inch margins).

The first page is to be completed in the multi-axial diagnosis format provided below. The remaining pages are to be a discussion of how you determined the diagnoses. It is often helpful to organize the latter pages by axis (see below). This discussion is to include the signs/symptoms of each diagnosis you assign, as well as a complete discussion of differential diagnoses. Differential diagnosis refers to all of the diagnostic categories that you seriously considered during the diagnostic process. Because the symptoms present in the case study suggest the possibility of several disorders, a thorough discussion of disorders that you excluded is warranted. In other words, you should discuss why you assigned the diagnoses that you did and why you ruled out others. You DO NOT need to include diagnostic Numbers.

Multi-axial Diagnosis Format

Axis I: Clinical Disorders
Other Disorders That May Be a Focus of Clinical Attention
Axis II: Personality Disorders
Mental Retardation
Axis III: General Medical Conditions
Axis IV: Psychosocial and Environmental Problems
Axis V: Global Assessment of Functioning

Multi-axial Diagnosis Pointers

Axis I: Includes all of the disorders we will cover in class, with the exception of Personality Disorders and Mental Retardation.

Axis II: Includes only Personality Disorders and Mental Retardation

Axis III: Includes general medical conditions that are relevant to Axis I and Axis II diagnoses.

Axis IV: Includes a listing of any relevant psychosocial and environmental problems or stressors.

Axis V: Includes a numerical rating of current functioning, and occasionally highest functioning over the past year, on a scale of 0 to 100. (See *DSM IV* for anchors to the GAF rating scale.)

You can have multiple diagnoses on any axis. It is also possible that there is no diagnosis on an axis. List every diagnosis for which the diagnostic criteria are met. When no diagnosis exists for a particular axis, "No Diagnosis" is entered on the line.

The first diagnosis listed on Axis I is assumed to be the principal diagnosis unless otherwise specified. If the principal diagnosis is a Personality Disorder or Mental Retardation, it should be listed on Axis II, labeled as the "Principal Diagnosis" in parentheses.

In the instance that diagnostic criteria are minimally met for two similar disorders and one diagnosis appears relatively clear but there remains a question about which diagnosis fits best, you can identify the most likely diagnosis on one line and list the second possibility on the next line followed by the term "Rule Out" in parentheses. This indicates some diagnostic certainty for the former diagnosis with a suggestion to monitor for the possibility of the latter diagnosis.

If there is insufficient information to make any diagnosis on any axis, enter "Diagnosis Deferred" on the line. This is different from "No Diagnosis" in that there is some possibility of a diagnosis on that axis but there is insufficient information to specify which diagnosis or not enough information to consider a "Rule Out" or a "Provisional Diagnosis".

PSY 210- Abnormal Psychology
Professor Jennings
Case Study Assignment
25 points

Due date: November 26th

You will select and review ONE case study provided in this packet. You will be responsible for reviewing the case and assigning multi-axial diagnoses. You will also be responsible for providing a rationale for the diagnoses, as well as a discussion of rule outs, differential diagnoses, and prognosis. This assignment should be 2-3 pages in length (typed, double-spaced, one inch margins). Please review the complete hand out included in the course syllabus for additional instructions.

Case Summary #1

Robin Henderson is a 30-year-old married Caucasian woman with no children who lives in a middle-class urban area with her husband. Robin was referred to a clinical psychologist by her psychiatrist. The psychiatrist has been treating Robin for more than 18 months with primarily anti-depressant medication. During this time, Robin has been hospitalized at least 10 times (one hospitalization lasted 6 months) for treatment of suicidal ideation (and one near lethal attempt) and numerous instances of suicidal gestures, including at least 10 instances of drinking Clorox bleach and self-inflicting multiple cuts and burns.

Robin was accompanied by her husband to the first meeting with the clinical psychologist. Her husband stated that both he and the patient's family considered Robin "too dangerous" to be outside a hospital setting. Consequently, he and her family were seriously discussing the possibility of long-term inpatient care. However, Robin expressed a strong preference for outpatient treatment, although no therapist had agreed to accept Robin as an outpatient client. The clinical psychologist agreed to accept Robin into therapy, as long as she was committed to working toward behavioral change and stay in treatment for at least 1 year. This agreement also included Robin contracting for safety- agreeing she would not attempt suicide.

Clinical History

Robin was raised as an only child. Both her father (who worked as a salesman) and her mother had a history of alcohol abuse and depression. Robin disclosed in therapy that she had experienced severe physical abuse by her mother throughout childhood. When Robin was 5, her father began sexually abusing her. Although the sexual abuse had been non-violent for the first several years, her father's sexual advances became physically

abusive when Robin was about 12 years-old. This abuse continued through Robin's first years of high school.

Beginning at age 14, Robin began having difficulties with alcohol abuse and bulimia nervosa. In fact, Robin met her husband at an A.A (Alcoholics Anonymous) meeting while she was attending college. Robin continued to display binge-drinking behavior at an intermittent frequency and often engaged in restricted food intake with consequent eating binges. Despite these behaviors, Robin was able to function well in work and school settings, until the age of 27.
She had earned her college degree and completed 2 years of medical school. However, during her second year of medical school, a classmate that Robin barely knew committed suicide. Robin reported that when she heard of the suicide, she decided to kill herself as well. Robin displayed very little insight as to why the situation had provoked her inclination to kill herself. Within weeks, Robin dropped out of medical school and became severely depressed and actively suicidal.

A certain chain of events seemed to precede Robin's suicidal behavior. This chain began with an interpersonal encounter, usually with her husband, which caused Robin to feel threatened, criticized or unloved (usually with no clear or objective basis for this perception. These feelings were followed by urges to either self-mutilate or kill herself. Robin's decision to self-mutilate or attempt suicide were often done out of spite- accompanied by the thought, "I'll show you." Robin's self-injurious behaviors appeared to be attention-seeking. Once Robin burned her leg very deeply and filled the area with dirt to convince the doctor that she needed medical attention- she required reconstructive surgery.

Although she had been able to function competently in school and at work, Robin's interpersonal behavior was erratic and unstable; she would quickly and without reason, fluctuate from one extreme to the other. Robin's behavior was very inconsistent- she would behave appropriately at times, well mannered and reasonable and at other times she seemed irrational and enraged, often verbally berating her friends. Afterwards she would become worried that she had permanently alienated them. Robin would frantically do something kind for her friends in an attempt to bring them emotionally closer to her. When friends or family tried to distance themselves from her, Robin would threaten suicide to keep them from leaving her.

During the course of treatment, Robin's husband reported that he could not take her suicidal and erratic behavior any longer. Robin's husband filed for divorce shortly after her treatment began. Robin began binge drinking and taking illegal pain medication. Robin reported suicidal ideation and feeling of worthlessness. Robin displayed signs of improvement during therapy, but this ended in her 14 month of treatment when she committed suicide by consuming an overdose of prescription medication and alcohol.

Case Summary #2

At the time of his admission to the psychiatric hospital, Carl Landau was a 19-year-old single African American male. Carl was a college freshman majoring in philosophy who had withdrawn from school because of his incapacitating symptoms and behaviors. He had an 8-year history of emotional and behavioral problems that had become increasingly severe, including excessive washing and showering; ceremonial rituals for dressing and studying; compulsive placement of any objects he handled; grotesque hissing, coughing, and head tossing while eating; and shuffling and wiping his feet while walking.

These behaviors interfered with every aspect of his daily functioning. Carl had steadily deteriorated over the past 2 years. He had isolated himself from his friends and family, refused meals, and neglected his personal appearance. His hair was very long, as he had refused to have it cut in 5 years. He had never shaved or trimmed hi beard. When Carl walked, he shuffled and took small steps on his toes while continually looking back, checking and rechecking. On occasion, he would run in place. Carl had withdrawn his left arm completely from his shirt sleeve, as if it was injured and his shirt was a sling.

Seven weeks prior to his admission to the hospital, Carl's behaviors had become so time-consuming and debilitating that he refused to engage in any personal hygiene for fear that grooming and cleaning would interfere with his studying. Although Carl had previously showered almost continuously, at this time he did not shower at all. He stopped washing his hair, brushing his teeth and changing his clothes. He left his bedroom infrequently, and he had begun defecating on paper towels and urinating in paper cups while in his bedroom, he would store the waste in the corner of his closet. His eating habits degenerated from eating with the family, to eating in the adjacent room, to eating in his room. In the 2 months prior to his admission, Carl had lost 20 pounds and would only eat late at night, when others were asleep. He felt eating was "barbaric" and his eating rituals consisted of hissing noises, coughs and hacks, and severe head tossing. His food intake had been narrowed to peanut butter, or a combination of ice cream, sugar, cocoa and mayonnaise. Carl did not eat several foods (e.g., cola, beef, and butter) because he felt they contained diseases and germs that were poisonous. In addition, he was preoccupied with the placement of objects. Excessive time was spent ensuring that wastebaskets and curtains were in the proper places. These preoccupations had progressed to tilting of wastebaskets and twisting of curtains, which Carl periodically checked throughout the day. These behaviors were associated with distressing thoughts that he could not get out of his mind, unless he engaged in these actions.

Carl reported that some of his rituals while eating were attempts to reduce the probability of being contaminated or poisoned. For example, the loud hissing sounds and coughing before he out the food in his mouth were part of his attempts to exhale all of the air from his system, thereby allowing the food that he swallowed to enter an air-free and sterile environment (his stomach) Carl realized that this was not rational, but was strongly driven by the idea of reducing any chance of contamination. This belief also

motivated Carl to stop showering and using the bathroom. Carl feared that he may nick himself while shaving, which would allow contaminants (that might kill him) to enter his body.

The placements of objects in a certain way (waste basket, curtains, shirt sleeve) were all methods to protect him and his family from some future catastrophe such as contracting AIDS. The ore Carl tried to dismiss these thoughts or resist engaging in a problem behavior, the more distressing his thoughts became.

Clinical History

Carl was raised in a very caring family consisting of himself, a younger brother, his mother, and his father who was a minister at a local church. Carl was quiet and withdrawn and only had a few friends. Nevertheless, he did very well in school and was functioning reasonably well until the seventh grade, when he became the object of jokes and ridicule by a group of students in his class. Under their constant harassment, Carl began experiencing emotional distress, and many of his problem behaviors emerged. Although he performed very well academically throughout high school, Carl began to deteriorate to the point that he often missed school and went from having few friends to no friends. Increasingly, Carl started withdrawing to his bedroom to engage in problem behaviors described previously. This marked deterioration in Carl's behavior prompted his parents to bring him into treatment.

Case Summary #3

At the time of his admission to a private psychiatric hospital, Sonny Ford was a 24-year-old single Latino male who lived with his adoptive parents. Sonny had been referred for hospital admission by his outpatient psychotherapist. Over the past 2 years, Sonny had struggled with symptoms such as concentration difficulties, anxiety, and obsessional thinking. More significantly, within the year prior to his admission, Sonny began to experience paranoid and delusional thoughts that had become quite persistent. These difficulties began after Sonny smoked marijuana. While experiencing the effects of marijuana, Sonny believed that his mind had gone "numb." From that time on, Sonny believed that the marijuana had permanently "warped" his brain. He became increasingly distressed and frustrated over his inability to get others to agree that marijuana had this effect on him. More recently, Sonny had developed concerns that the police and FBI were "out to get him." In addition, he had begun to feel that certain television shows had special importance to him and important information was embedded in these programs directed specifically at him. Sonny believed that these messages coming to him through the television were sent to remind him that he was at risk for some sort of plot by the authorities. Sonny also heard voices in his head. Although he could not make out what they were saying, Sonny perceived the voices as "angry" and "critical."

Over the past few months, Sonny's symptoms had worsened to the point that they were interfering substantially with his attendance at work as a state office janitor. Because of these factors and the lack of improvement in outpatient counseling, Sonny was referred to this inpatient hospital.

At the intake evaluation for his inpatient admission, Sonny's emotions were restricted. Although appearing tense and anxious, Sonny's face was mostly immobile for the duration of the interview. He engaged in very little eye contact with the interviewer and his body movements were agitated and restless, as evidenced by rocking movements of his legs and body. His speech was hesitant and deliberate, and he often answered the interviewer's questions with brief and empty replies. For example, when the interviewer asked "what difficulties are you having that you would like help for?" Sonny replied, "I think it was the marijuana."

Clinical History

Sonny was adopted at birth, and no records were available about medical or psychiatric history of his family origin. Sonny was raised in a household of four: in addition to his parents, he had a sister 4 years older who had also been adopted. He could recall very few memories from his early childhood. However, Sonny said that throughout his life he had always been a loner who, to this day, never had any friends. Sonny's parents, who were present at the time of his admission to the hospital, confirmed that Sonny had always been frustrated by social interactions and added that their son had always been hypertensive to real or perceived criticism during his school years. Sonny was very attached to his father and, for may years, experienced considerable distress and

loneliness when he was separated from the family's home or his father for extended periods. Whereas Sonny described his father as "a very accepting person" he claimed that his mother was "excessively critical and not accepting of me as a person." Sonny also claimed that his mother was an alcoholic, a statement that was not supported by either of his parents.

When Sonny was 16, he realized that he was homosexual. Although his father had been accepting Sonny reported that his mother had been very unaccepting of his homosexuality and often referred to him with pejorative labels, such as "fag." While Sonny accepted his sexual orientation, he said that being gay had caused him many troubles one of which was loneliness. Many of Sonny's persistent and obsessive thoughts focused on the possibility of contracting the HIV virus from having unprotected sex on one occasion. Sonny's fears of having HIV had not been quieted by the fact that the person with whom he had sex with was HIV negative or by the fact the all of his recent HIV tests were also negative.

Despite lifelong difficulties with social adjustment, Sonny had been able to meet most of the demands and responsibilities of adolescence. Following his graduation from high school (with a C+ average), sonny decided to attend a local college to take introductory courses. This decision was strongly influenced by his apprehension of moving out of his parent's house to attend school away from his immediate community. However, it was during his freshman year that Sonny had smoked the marijuana that he believed permanently damaged his brain. Following the incident, Sonny dropped out of college due to the worsening of behaviors. Sonny enrolled at a second college for only one semester before dropping out again, because of his inability to cope with sitting in crowded classrooms and completing assignments and tests on time.

Sonny has held his current position as a janitor for the last 18 months, in part because this position allows him to work alone and does not require extensive social interaction.

Abnormal Psychology (PSY 210)
Extra Credit Paper
Professor Jennings

Submission Deadline: On or before November 29th

INSTRUCTIONS FOR PAPER:

This is an extra credit paper worth **10 points** toward your total final grade. Your paper should be a summary of a selected article that you choose from a reputable source related to the topic of behavior. Acceptable sources would include articles of at least 4 pages, but no longer than 12 pages from a scholarly, peer-reviewed journal. The article must be published from 2000 to the present.

Your paper must be typed, double-spaced and **one page only**. The font size should be either 10 or 12 point. You should include your name and section number on the back of the paper only. Please do not submit your paper in a plastic binder or folder.

The title of your article should not be included at the top of the paper or in the body of the paper. The title of the article should only appear in the reference at the bottom of your paper. **The first sentence of your paper should include the author of the article you are summarizing and the date of the publication.** Examples of how you may start include: As Aronson (2002) discovered that…or Aronson (2002) suggests in his article…or Aronson (2002) states that…found that…etc.

The bottom of your paper should include the reference: author (last name, first initial). If there is more than one author, all the authors must be named in the reference, but not the body of the paper.) The body of the paper can say, Aronson et al. (2002)…). An example of the APA (American Psychological Association) method that your paper should follow to cite your reference, looks like this:

Murray, B. (2000). Teaching students how to learn. *Monitor on Psychology*, Vol. 31 (8), pp. 64-68.

The reference should be single spaced and the second line should be indented.

The summary that you write should not be an opinion paper or personal feelings paper. You need to read an article and then summarize the article in your own words. Try not to use direct quotes. If you do, be sure to follow the APA format for direct quotes, do not copy directly from the article. You will be limited to ONE direct quote ONLY should you elect to do so. Your job will be to rewrite what you read…in other words, paraphrase.

Your paper will be graded on the selection of your article, the content, the organization of your paper, and the clarity and coherence of your writing. Spelling, grammar, punctuation, style, all count in the evaluation.

Grading Criteria:

An "A" level paper will receive 7-10 points:
The student uses a scholarly, peer-reviewed article to summarize that is about 4-12 pages in length on a topic clearly related to psychology. The student makes a copy of the article and reads it several times, making comments in the margin and highlighting important sections of the article. The student has full understanding of the article and captures the essence of the article in a clear and concise one page summary. The student manages to address what the article is about, and if it is about an experiment, the student explains the author's hypothesis and if it was supported. If the student is summarizing an experimental research article, the student relies on the Introduction, Methods, Results and Discussion sections to write the summary. It is not necessary for the student to understand the statistics used in the Results section, but the student must report whether the findings support the hypothesis and what the implications are for the research.
The paper has less than two spelling errors and is grammatically correct, captures the essence of the article and is clearly written. The APA format is followed and the reference is properly cited.

A "B" level paper will receive 4-6 points:
The student selects a topic clearly related to psychology but uses a more readable, less scholarly article to summarize such as Psychology Today, Time Magazine or Newsweek. The student follows the same procedure as above, and writes a clear and consise summary that captures the essence of the article. The paper is grammatically correct, and has more than 4 spelling errors. The student follows APA format and the reference is appropriately cited.

A "C" level paper will receive 1-3 points:
The student submits a paper that meets the requirements of the assignment. The student has done a satisfactory job with the assignment, but the paper is not as clearly written as an "A" or "B" level paper and/or may have cited the reference improperly.

No points will be awarded for a paper that falls below a C level of work.

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Attention Deficit Hyperactivity Disorders, Psychostimulants, and Intolerance of Childhood Playfulness: A Tragedy in the Making?

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Attention deficit hyperactivity disorders (ADHDs) are the most common childhood psychiatric problems in our society. From a prevalence of no more than 1% of children, when this type of disorder was first conceptualized by the British physician George Still in 1902, the average incidence in the United States rose to more than 5% at the beginning of this decade (Armstrong, 1995). At such an incremental rate, an estimated 15% of American children (about 8 million) may be so diagnosed by the turn of the century. What are the reasons for this?

There is no indication that the incidence of any true neurological problem has increased during the intervening century, and this ominous diagnostic trend may be emerging from our changing social structures and mores. Increasingly, standardized educational expectations along with a growing intolerance of childhood playfulness may, in fact, be leading to more and more children being labeled with ADHD. Indeed, the ability of psychostimulants to promote attention is surely only one of the reasons why such drugs are increasingly used; a less well-appreciated reason is that psychostimulant drugs also markedly reduce children's impulsive urge to play. This issue becomes all the more troublesome because we do not yet understand the true brain functions of play, and the long-term neurological consequences of psychostimulant drugs remain to be investigated properly. Preclinical work (i.e., animal research) is definitive on this issue: One of the major effects of psychostimulants is to reduce the urge of young organisms to exhibit rough-and-tumble play (Panksepp, Normansell, Cox, Crepeau, & Sacks, 1987; Vanderschuren, Niesink, & Van Ree, 1997). There are many anecdotal reports of similar antiplay tendencies in children taking psychostimulants, but such effects in humans remain to be formally documented. Still, the possibility that the "benefits" of psychostimulants in some children may simply be due to their effects on childhood playfulness adds a poignant new dimension to the ongoing discussion of the propriety of psychostimulant use in children.

The explosion in the diagnosis of ADHD may largely reflect the fact that more and more of our children no longer have adequate spaces and opportunities to express this natural biological need—to play with each other, in vigorous rough-and-tumble ways, each and every day. Because we now know that play is a regulated process similar to food and water intake, a strong case can be made that children need and desire a certain amount of rough-and-tumble play every day, as do other young mammals (Panksepp, Siviy, & Normansell, 1984; Vanderschuren et al., 1997). Without adequate daily outlets for such youthful energies, playful impulses will undoubtedly tend to intrude increasingly into regular classroom activities. From this perspective, much of ADHD may need to be substantially reconceptualized as a symptom of our

Recommended Reading

Armstrong, T. (1995). (See References)
Barkley, R.A. (1997). (See References)
Panksepp, J. (1998b). (See References)

contemporary society and our modes of regulating children's behavior, rather than a symptom of any neurobiological imbalance or disorder.

By failing to recognize the existence of primitive emotional forces of the brain that govern child development, such as those that generate rough-and-tumble play (Panksepp, 1993, 1998a), we may be overlooking some of the fundamental psychobiological needs of our children. Because of the increasingly competitive nature of education, we may be seeing childhood psychopathology where we should be seeing normal human diversity. If that is happening, we must pursue, with renewed vigor (e.g., Armstrong, 1995; Breggin, 1998; Hartmann, 1996), the issue of whether it is appropriate to medicate children with attention-promoting drugs that compromise their desire to play. My aim in this review is to explore this issue, as well as a variety of related critical questions concerning the nature of ADHD.

The following discussion proceeds with the full recognition that psychostimulants are remarkably effective in the symptomatic control of childhood impulsivity and the temporary facilitation and focusing of attention. That robust effects can be achieved is undeniable (Solanto, 1998). Indeed, the effects are so strong and clear that they are recognized despite attacks by psychosocially oriented investigators who wish to intimate that the clinical changes attributable to these drugs are more limited than is commonly believed (see Whalen & Henker, 1997). Indeed, the attention-focusing effects—among the most well-documented findings in child psychiatry—are often deemed so desirable that parents and teachers regard psychostimulants as lifesavers for many troubled children (Spencer et al., 1996). Unfortunately, many key issues surrounding such drug-use practices remain to be adequately aired, and the relevant literature is vast (see Sagvolden & Sergeant, 1998). Here, I discuss half a dozen troublesome issues that we need to address if we are going to make substantive headway in dealing with ADHD as an emerging societal problem. The specific questions I raise are as follows: Is most of what is diagnosed as ADHD simply a natural variant of human personality? What are the major constitutional differences in neural organization in ADHD and more typical children? Are the short-term behavioral management benefits of psychostimulants accompanied by any long-term cognitive or emotional benefits? Can psychostimulants promote permanent neural changes, and, if so, are they desirable or undesirable? Can psychostimulants modify the neural plasticity (learning and other semi-permanent neural changes) that characterizes normal childhood? Are there any reasonable cultural alternatives to the use of psychostimulants that remain to be tried and properly evaluated?

IS ADHD A NORMAL VARIANT OF HUMAN DIVERSITY?

Some children are predestined to be tall and others small. Some are predestined toward obesity, others toward leanness. Some are shy and inhibited, whereas others are open and gregarious. It is now recognized that, to a remarkable degree, human temperament can also be inherited (see the August 1997 Special Issue of this journal). Indeed, analysis of identical twins reared apart indicates that approximately half of human personality variability is due to inheritance, and half is due to environmental causes (Bouchard, 1994). More than half the children who exhibit symptoms of ADHD have a high incidence of "afflicted" siblings and parents who might have been diagnosed similarly (Hewitt et al., 1997). Because of their personality differences, ADHD children certainly clash more easily than other children with various modern societal and educational demands, but is this sufficient reason to call their style of interaction a disorder? Now that we have extremely simple diagnostic criteria (in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders*, at least six negative responses to nine questions about attentional problems and six to the nine questions concerning hyperactivity-impulsivity), an increasing number of children are being diagnosed and receiving drugs that will, almost certainly, make them more attentive and cooperative. Although such interventions can dramatically reduce classroom conflicts, that should not lull us into believing that most of these children have brain disorders.

Except for a minority of cases involving distinct medical problems such as hyperthyroidism and explicit brain injuries, most youngsters diagnosed with ADHD may simply be normal, highly playful children who have difficulty adjusting to certain institutional expectations. Perhaps such temperaments were more adaptive in primitive ancestral environments, when hunting and gathering were the main pursuits of life. A reasonable case can be made for the conclusion that impulsive individuals with rapidly shifting attentional responses may have been excellent hunters (Hartmann, 1996). For instance, "distractibility" may be a useful trait when one needs to efficiently monitor a variable environment. "Impatience" may be a good trait when one needs to rapidly shift into a result-oriented mode of action. We can wonder whether "difficulty following instructions" might not reflect a high dose of

"independent judgment," and whether "acting without regard to consequences" might not reflect a greater "willingness to take risks and face dangers." If this is the case, the societal difficulties we presently have with such personality types may reflect our recent cultural evolution more than the existence of any pathological personality deviance.[2] If this is a defensible thesis, perhaps society should try to nurture this type of human variability or to adjust to it, rather than seeking to pathologize it and eliminate it with attention-focusing psychostimulants. It is certainly more rational to try to solve social problems with social solutions than with drugs, especially when the drugs are so similar to the ones we are trying to purge from our society.

WHAT ARE THE NEURAL DIFFERENCES IN INDIVIDUALS WITH ADHD?

Recent brain-imaging data have revealed that the major difference in the brains of individuals with ADHD is in the frontal areas. At a gross structural level, brains of children diagnosed with ADHD exhibit a 5% reduction in overall size. This difference is accompanied by an absence of the typical right-left asymmetries, with right frontal areas being selectively smaller than those on the left (Castellanos et al., 1996). Reduced frontal lobe functions have also been evident, at times, with metabolic positron emission tomography (PET) imaging in adults (Ernst, Cohen, Liebenauer, Jons, & Zametkin, 1997), and decreased frontal activity in ADHD children is consistently observed with measures of electrical activity in the brain (Chabot & Serfontein, 1996). There are also some neurochemical differences (Solanto, 1998), but none is sufficiently large to permit us to conclude that the differences constitute a medically significant abnormality.

The neural differences in ADHD children have recently been formulated into a coherent theory by Barkley (1997). It has long been recognized that the frontal lobes are essential for long-term planning and the elaboration of complex behavioral strategies. As the frontal lobes mature, children become more able to inhibit their simple emotional tendencies and to conceptualize more complex psychological perspectives. In other words, the frontal lobes normally allow organisms to internalize and plan behaviors prior to emission. Thus, it is no wonder that damage to the frontal lobes can dramatically increase activity and promote rough-and-tumble ludic (playful) impulses (Panksepp, Normansell, Cox, & Siviy, 1995).

Barkley's synthesis is summarized in Figure 1. According to this view, the fundamental problem in ADHD is not a deficit in attention, but an abnormally low level of behavioral inhibition, a global function that allows better reflection, imagination, empathy, and creativity. These abilities promote behavioral flexibility, better foresight, and more mature regulation of behavior. If we accept the existence of a frontal lobe contribution to ADHD, we still must consider whether there are environmental or physical ways to improve such frontal lobe functions permanently. There are many reasonable possibilities that have never been evaluated, not the least being providing more access to play within school schedules (Panksepp, 1998a, 1998b).

DO PSYCHOSTIMULANTS PROMOTE ANY LONG-TERM BENEFITS OR PROBLEMS?

Researchers are beginning to appreciate that the ability of psychostimulants to reduce hyperactivity is not a paradoxical effect but the natural consequence of promoting activity in brain systems that normally facilitate attention and goal-directed behaviors (Pliszka, McCracken, & Maas, 1996). Indeed, these drugs work equally well in most individuals, whether diagnosed with ADHD or not. However, even as we recognize the vigor of such effects, we should remember that these drugs modulate arousal of the same brain systems that mediate various types of drug addiction (Wise & Rompre, 1989). The fundamental effects of the major drugs used to treat ADHD—modulation of the activity of the neurotransmitters dopamine and norepinephrine—are similar to those produced by cocaine, albeit the drug actions are characterized by slower onset, and weaker and more prolonged effects. The temporary benefits that are commonly observed are quite reasonable, given what we presently know about neurochemical controls within the frontal lobes. Pharmacological modulation of dopamine and norepinephrine systems has been shown to sustain attentional processes and on-task behaviors in various studies with animals (Solanto, 1998).

Although short-term behavioral improvements certainly occur with psychostimulants, long-term benefits have rarely been evident. When medication is terminated, ADHD symptoms typically return, suggesting the children are not learning to manage their lives any better. Evidence for long-term improvements of cognitive functions and other abilities remains practically nonexistent (for a summary of such work, see Barkley, 1997).

Short-term benefits would be acceptable (as they are for most medicines) if any long-term problems did not outweigh the benefits. Psychostimulants do have some negative bodily consequences: A

small decrease in physical growth has been repeatedly observed, along with relatively rare incidence of seizures, tics, mania, and delusional tendencies. Also, many children do not like the feelings induced by psychostimulants. Moreover, there is a potential for certain difficulties when these children become adults. They may have continued behavior problems if they do not take medication, and they may have an increased tendency toward drug abuse. Although some investigators report no elevated drug abuse, such problems have been evident in other studies (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993). Unfortunately, most follow-up studies cannot resolve whether such consequences are due simply to constitutional differences or to the accumulating consequences of the medications. The key issue—whether the medications promote undesirable and permanent personality changes, such as increased craving and addictive tendencies—remains unresolved for humans, but the emerging animal data are troubling.

CAN PSYCHOSTIMULANTS PROMOTE PERMANENT NEURAL CHANGES?

Preclinical work indicates that both short- and long-term exposure to psychostimulants promote lasting changes in neural tissues. This process is termed *sensitization.* Animals repeatedly exposed to psychostimulants become more responsive to those drugs (Pierce & Kalivas, 1997), and humans are not immune to such effects (Strakowski, Sax, Setters, & Keck, 1996). Sensitized animals exhibit elevated addictive tendencies as well as chronic personality changes characterized by intensified cravings—elevated "I want" types of approaches to the world. In other words, sensitized animals become more active, and they exhibit greater arousability of brain incentive-seeking systems and a greater desire (behavioral persistence) for material rewards (Robinson & Berridge, 1993).

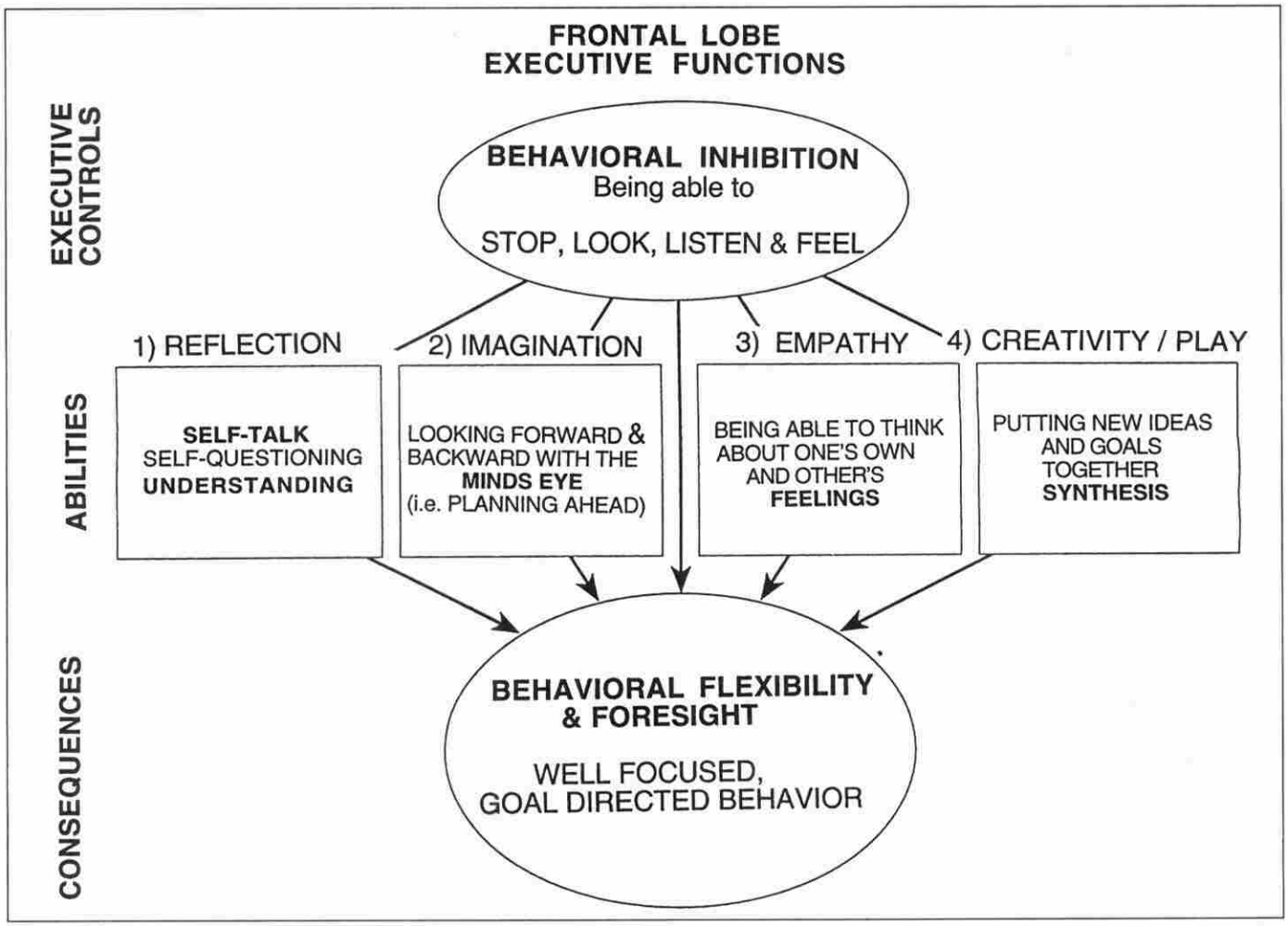

Fig. 1. A synoptic interpretation of the frontal lobe functions that may be deficient in attention deficit hyperactivity disorder (adapted from Barkley, 1997).

We may seek some solace in the fact that the preclinical data also indicate that sensitization is generally less intense in young organisms than in adults (Duke, O'Neal, & McDougall, 1997). However, researchers do not know whether these findings generalize to human children. Because the concept of sensitization is so pregnant with important societal implications, it will be important to determine whether children who have been chronically treated with psychostimulants exhibit larger behavioral responses to the drugs than those about to be treated for the first time.

CAN PSYCHOSTIMULANTS REDUCE NEURAL PLASTICITY?

Early childhood is a time for abundant learning, partly because the intrinsic plasticity of the nervous system is greatest during early development. Scientists are just beginning to learn about the many "fertilizers" that exist in the brain to promote full neural maturation. They include a large variety of neurotrophins (one of the growing classes of growth factors) that have precise roles in neural maturation. Some of these growth factors are activated by specific types of sensory stimulation that may be especially abundant during rough-and-tumble play (Panksepp, 1998b). Hence, a key question that deserves to be addressed is whether psychostimulants modify any of these processes in beneficial or undesirable ways.

At present, the types of scenarios that could be hypothesized range from the rosy to the scandalous. On the rosy side, one might suppose that pharmacological activation of frontal lobe tissues may facilitate desirable neural activities and thereby promote neural maturation. However, there is presently no evidence for this hypothesis, and there are reasons to believe that psychostimulants may reduce plasticity: A great deal of brain plasticity is initiated by the most important excitatory neurotransmitter of the brain, glutamate (Kaczmarek, Kossut, & Skangiel-Kramska, 1997), which helps mediate all our thoughts, intentions, and emotions (Panksepp, 1998a). In general, dopaminergic arousal, as can be achieved with psychostimulants, tends to reduce glutamate-mediated neural transmission (Olney & Farber, 1995), which may decrease neural plasticity. However, this scenario might not be the correct one because it remains possible that psychostimulants reduce ADHD symptoms more by reducing dopaminergic arousal than by increasing it (Solanto, 1998). Clearly, more research on such important issues is desperately needed. If the data eventually demonstrate that psychostimulants reduce plasticity, our society will need to promptly reassess the wisdom of such drug use in children.

BUT WHAT ELSE MIGHT WE DO TO ADDRESS SUCH CHILDHOOD PROBLEMS?

Even if this scientific story does not unfold as negatively as seems possible, we should always be entertaining environmental alternatives to deal with cultural problems. Considering that ADHD has existed as a formal psychiatric entity—whether called hyperkinesis or minimal brain damage—for all of this century, one would think that everything that could be done environmentally has already been tried. Regrettably, that is far from true. We have neglected some of the simplest and most straightforward strategies, such as modifying aspects of our educational systems to better accommodate the human diversity that ADHD may represent.

The existing intervention literature does not hold out much promise that either cognitive or parental interventions will have strong and lasting effects on ADHD symptoms (see Barkley, 1997), although combining various interventions can reap some benefits (Barkley et al., 1996). However, perhaps the simplest approach has never been tried: To my knowledge, rough-and-tumble interventions remain to be formally evaluated.

As noted earlier, the urge to indulge in rough-and-tumble play is a birthright of the mammalian brain (Panksepp et al., 1984), and such activities may promote the normal maturation of children's brains, perhaps through the arousal of neurotrophins in neural circuits (Panksepp, 1998b). As a society, we need to fully consider whether ADHD is largely a tendency of young nervous systems to play too rambunctiously. If it is, we should determine if simply permitting more space for such natural childhood activities as well as other creative bodily arts might diminish ADHD-type symptoms within our school systems. Indeed, it has long been recognized that ADHD symptoms are rarely as disturbing to adults in unstructured home environments as they are to teachers in structured classrooms. Unfortunately, at present, there is hardly a state left in the Union that still mandates physical education from kindergarten through 12th grade. This deemphasis of physical education will be deemed shortsighted if it turns out that providing young children abundant play opportunities reduces their impulsivity in adolescence.

Even though no one has yet unraveled the precise functions of the ludic brain process, there are good

reasons to believe that they promote both neural and psychological development. Social play may be an *experience-expectant,* or preparatory, process that helps program higher brain areas that will be required later in life. Indeed, "youth" may have evolved to give complex organisms time to play and thereby to exercise the natural skills they will need as adults. We already know that as the frontal lobes mature, frequency of play goes down, and animals with damaged frontal lobes tend to be more playful (Panksepp, Normansell, Cox, & Siviy, 1995). Might access to rough-and-tumble play promote frontal lobe maturation?

To evaluate some of these ideas, my colleagues and I have recently produced an animal model of ADHD by unilaterally reducing the size of the frontal lobes in young rats. Such animals are very hyperactive and playful. However, when these animals were given abundant opportunities to play throughout their youth, they exhibited a greater than normal decline in the amount of play as they matured. Thus, play circuits may be regulated beneficially, in the long term, through playful experiences (Panksepp, Burgdorf, Turner, & Walter, 1997).

CONCLUSIONS AND FUTURE PROSPECTS

It is likely that the "therapeutic" effects of psychostimulants in our children reflect, to some degree, not only their ability to sustain focused attention, but also their ability to decrease the desire for vigorous social engagement. Indeed, the normal desire to play may readily be interpreted as an impulse-control disorder when it is unwanted. These observations raise a dilemma: Is it appropriate for society to use pharmacological agents to reduce the natural and positive brain functions of developing children (i.e., playfulness)? What is our responsibility to children when we pursue such courses of action without having first studied, in animals, the consequences of these manipulations for long-term psychological and neurological development? One of the functions of early childhood playfulness, especially of the rough-and-tumble variety, may be to promote the development of higher brain systems, such as the executive functions of the frontal lobes—functions that mature slowly in all children, but especially so in those labeled with ADHD. Are we now diminishing the natural patterns of brain maturation by pharmacologically inhibiting the playful impulses of our children? Only when we have answered these questions will we be able to adequately weigh the long-term benefits and costs of such interventions.

After a half-century of research, not a single piece of credible evidence yet indicates that ADHD children exhibit sufficiently extreme degrees of biological or psychological deviance that they should be deemed an abnormal population in the strict medical sense. Thus, the use of psychostimulants with these children must be deemed a grand experiment in cosmetic psychopharmacology, and the results have certainly been impressive. The evidence is definitive: Children's behaviors can be modified with these drugs to make classroom management much easier. Many children have been helped to adjust to educational demands with psychostimulants, and their lives have improved in certain ways because they are less likely to be ostracized as their behaviors conform better to prevailing norms. I would not want to suggest that parents have erred in allowing their children to be medicated, but at the same time, there are some very disturbing scientific issues that desperately need to be answered. Some of the answers may not be pleasant for those of us who have supported such medication practices, but we should all be willing to seriously consider the emerging evidence, especially when the abuse of such drugs is increasing among our young.

At present, it seems highly likely that the powerful antiplay effect of psychostimulants is one reason those medications are so widely used to treat children. When children have these drugs in their brains, they are simply less rambunctious in their classrooms. Their attentional focus is narrowed, and they no longer exhibit as much of the expansive and impulsive joy of life that is a hallmark of childhood. Regrettably, as the availability of physical education and recess time has declined, all our children have little chance to dissipate their ludic energies during the school day. Thus, a substantial proportion of the present generation may no longer be getting sufficient amounts of the presently unknown neural and psychological benefits of play for their brains to mature optimally.

One fear that teachers and other professionals are bound to voice is that allowing ADHD children more rough-and-tumble activities will only make them wilder. This is a reasonable logical concern, but the existing animal data indicate just the opposite: Rough-and-tumble play is a well-regulated brain process, and just like our other appetites, it appears to be homeostatically controlled (physiologically regulated) in both short- and long-term time frames (Panksepp et al., 1984). Indeed, animals need no more than a couple of hours of energetic play each day, and there is no good reason to believe that human children will not follow this pattern. It is only when

they are not allowed to play that their drive for such activity markedly increases and comes to disrupt classroom activities.

There is reason to believe that unless children get some play before the start of classes each day, their biological urges to indulge in ludic activities will remain insistent. These impulses can emerge in ways that professionals trained to see the world in terms of childhood disorders will readily diagnose as ADHD, and children will be tracked toward medications without any other reasonable intervention being taken. Why not institutionalize a period, early in the morning, when all schoolchildren are encouraged to dissipate their playful energies under the positive guidance of adults? Not only might such children be able to better attend to their lessons later in the day, but perhaps they would also have an abundance of neural growth factors circulating in their brains, promoting the ability of their brains to learn, to grow, and to mature more effectively.

Now that we are beginning to recognize how much of human personality is genetically controlled and how these genetic potentials are brought to life by psychobehavioral processes such as rough-and-tumble play, we must scientifically seek to determine how much we can truly modify human tendencies in positive directions through systematic play interventions (Panksepp, 1993). We must better evaluate our preschool and Head Start programs and modify them in ways suggested by the best evidence. Even if the results eventually indicate that our efforts can only have modest effects, it will still be a worthy goal to pursue. In any event, as a society that respects the human "pursuit of happiness," we should be willing to create more spaces in our world where children can express their natural ludic urges. We all appreciate how dangerous the postmodern world can be and how important it is to protect our children, but there are many straightforward ways to help restore a more balanced joyous existence to our children. The school system, especially in the lower grades, is a reasonable place to encourage such projects. We may be taking a major step toward promoting childhood mental and physical health when we, as a society, begin to recognize and affirm the right of children to really frolic for a few hours each day. If we do that well, we may eventually have less ADHD, and a much healthier society.

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Notes

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2. For additional discussion of this idea, visit the following site on the World Wide Web: http://www.conix.com/~hypercog/add.htm.

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The Role of the Family in the Course and Treatment of Bipolar Disorder

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ABSTRACT—*Bipolar disorder is a highly recurrent and debilitating illness. Research has implicated the role of psychosocial stressors, including high expressed-emotion (EE) attitudes among family members, in the relapse–remission course of the disorder. This article explores the developmental pathways by which EE attitudes originate and predict relapses of bipolar disorder. Levels of EE are correlated with the illness attributions of caregivers and bidirectional patterns of interaction between caregivers and patients during the postepisode period. Although the primary treatments for bipolar disorder are pharmacological, adjunctive psychosocial interventions have additive effects in relapse prevention. Randomized controlled trials demonstrate that the combination of family-focused therapy (FFT) and pharmacotherapy delays relapses and reduces symptom severity among patients followed over the course of 1 to 2 years. The effectiveness of FFT in delaying recurrences among adolescents with bipolar disorder and in delaying the initial onset of the illness among at-risk children is currently being investigated.*

KEYWORDS—*family-focused treatment; expressed emotion; psychosocial treatment; pharmacotherapy; childhood-onset bipolar disorder*

By the year 2020, bipolar disorder will be the sixth leading cause of disability worldwide among all medical illnesses (Murray & Lopez, 1996). Persons with the disorder vary between the extremes of mania (a highly energized, elated, or irritable state) and depression (a deflated, withdrawn, morose, and often suicidal state). The *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., American Psychiatric Association, 1994) defines two primary types of bipolar disorder: Bipolar I (at least one episode of mania or mixed disorder) and Bipolar II (episodes of hypomania alternating with severe periods of depression). Over 2% of the US population has Bipolar I or II disorder, and another 2.4% has the disorder in its various subclinical (or subsyndromal) forms (Merikangas et al., 2007).

The illness is highly recurrent, with 60% of patients experiencing recurrences of mania or depression within 2 years and up to 75% experiencing recurrences within 5 years. Patients experience significant symptoms during approximately half of the weeks of their lives and have multiple impairments in school, at work, and in relationships (Judd et al., 2002). Up to 15% die by suicide, and as many as 50% attempt suicide one or more times (Miklowitz & Johnson, 2006).

Mood stabilizers and atypical antipsychotics have a substantial success record in hastening recovery from episodes and decreasing risk of recurrence. However, these medications do not fully prevent recurrences of the disorder, nor do they eliminate the significant residual symptoms—notably depression—that many patients experience between episodes.

The family environment is an important context for understanding the development and maintenance of severe psychopathology (Repetti, Taylor, & Seeman, 2002) and mood disorders in particular (Hooley & Parker, 2006; Miklowitz, 2004). This article concerns the role of family relationships as risk or protective processes in the course of bipolar illness and the role of family-focused therapy (FFT) as an adjunct to drug therapy during the postepisode period.

THE ROLE OF THE FAMILY IN THE COURSE OF BIPOLAR ILLNESS

Current thinking about the relapse–remission course of bipolar disorder emphasizes a biopsychosocial model that incorporates the interactive roles of genetic vulnerability, biological predispositions, family or life events stress, and psychological vulnerability. The

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illness is clearly heritable, and there is substantial evidence for dysfunction of the neurotransmitter systems (notably dopamine and serotonin) and of the limbic–cortical system. Specifically, elevated activity in the amygdala and diminished activity of the frontal-cortical regions may interfere with the capacity to regulate emotion (for review, see Miklowitz & Johnson, 2006).

Family stress has been operationalized as whether or not the patient resides with relatives characterized by high expressed-emotion (EE) attitudes (Brown, Birley, & Wing, 1972). High EE refers to high levels of criticism, hostility, and/or emotional overinvolvement from a caregiving relative (typically a parent or spouse) during or immediately following a patient's acute episode of illness. It is typically assessed through the Camberwell Family Interview, although briefer alternative assessments with acceptable reliability and validity are available (Hooley & Parker, 2006). Patients with schizophrenia, bipolar disorder, or recurrent major depressive disorder who return home to high-EE families following an acute episode are two to three times more likely to relapse in the subsequent 9 months than are patients who return to low-EE families (Barrowclough & Hooley, 2003; Miklowitz, 2004).

A Developmental Psychopathology Approach to Expressed Emotion

Research on the mechanisms underlying the association between EE and relapse have addressed two questions: (a) How do caregiving relatives become high-EE? (b) What variables mediate the association between EE and patients' relapses? A developmental psychopathology framework for understanding the causal and reactive roles of parental EE in mood disorders (Miklowitz, 2004) begins with a child who has temperamental disturbances (e.g., irritability, low frustration tolerance, mood instability, high anxiety) or compromised cognitive functioning. These early disturbances partially reflect the child's genetic vulnerability to bipolar, schizophrenic, or other psychiatric disorders. The child is paired with a parent who, by virtue of his or her own neurobiology and social history, reacts to the child's behavior with frustration and hostility (expressed as frequent criticisms of the child) or with guilt and anxiety (expressed in overly protective behaviors). Repeated exposure to criticisms, anxiety, or overinvolvement during the period when a child is developing a sense of identity may contribute to self-doubt, self-criticism, and core beliefs about relationships as aversive and conflict-ridden. These schemata for the self may interfere with the child's acquisition of emotional self-regulatory skills, as reflected in depression, anxiety, aggression, and the inability to tolerate negative states of affect. In turn, his or her negative counterreactions fuel high-EE attitudes and behaviors in parents, which recursively contribute to the child's emotional and cognitive vulnerabilities. The model in Figure 1 clarifies how similar recursive processes may maintain high-EE attitudes and contribute to relapse in families in which patients have already had episodes of bipolar disorder.

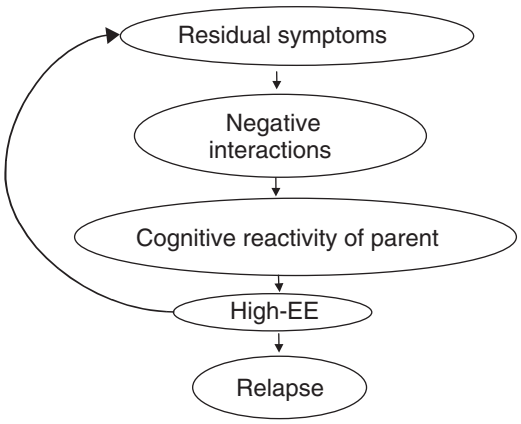

Fig. 1. Bidirectional relationship between parental expressed emotion (EE) and patients' mood symptoms. The pathway begins with unresolved residual symptoms in the patient (e.g., depression, irritability, mild delusional thinking, withdrawal, hypomania) that contribute to the intensity of the patient's reactions to caregivers. Escalating negative interactions reduce the threshold for caregivers to react with fear, frustration, and hopelessness; remember and exaggerate negative experiences from prior illness episodes; and make attributions of controllability and negative predictions about the future (e.g., ''She's doing this to hurt me''; ''I'll always have to take care of him''). This ''cognitive reactivity'' of the caregiver may fuel his or her expression of high-EE attitudes toward the patient, resulting in temporary exacerbations of the patient's residual symptoms and a worsening pattern of dyadic interaction. In combination with biological and genetic vulnerability factors, repeated exposure to negative family interactions may contribute to the patient's overall liability to early relapse.

Research on Pathways to EE

Although the pathway in Figure 1 is hypothetical, it is supported by cross-sectional research on the correlates of EE among relatives of psychiatric patients. Notably, high-EE relatives are more likely to attribute the negative behaviors of patients to personal and controllable factors (i.e., personality, lack of effort) than are low-EE relatives, who are more likely to attribute the behaviors to uncontrollable factors (i.e., illness or external stressors; Barrowclough & Hooley, 2003). Several studies find that high-EE relatives and patients are more likely than low-EE relatives and patients to be engaged in negative cycles of verbal and nonverbal interaction during the postepisode period (for review, see Miklowitz, 2004).

A study of family interactions among patients with schizophrenia is particularly relevant to this hypothesized pathway (Rosenfarb, Goldstein, Mintz, & Nuechterlein, 1995). During the period following a hospitalization for psychosis, patients with schizophrenia from high-EE families showed significantly more odd and disruptive behaviors with parents during laboratory-based family interactions than did patients from low-EE families. High-EE parents were more likely than low-EE parents to respond with a criticism to the first unusual thought expressed by the patient with schizophrenia, which increased the probability that the patient would express a second unusual thought. A study with similar methodology in a small sample of bipolar patients found that there was a strong correlation ($r = .53$) between relatives' harsh criticisms and patients' "odd and grandiose thinking" during family interactions,

but only among patients who relapsed in the subsequent 9 months (Rosenfarb et al., 2001). The correlation was low ($r = .12$) among patients who did not subsequently relapse.

The pathways from high-EE attitudes in caregivers to relapses among patients may be mediated by patients' biological and psychological vulnerabilities. Hooley, Gruber, Scott, Hiller, and Yurgelun-Todd (2005) examined neural activation (as measured by functional magnetic resonance imaging) among college students with and without a history of depression while they listened to tapes of their mothers expressing critical, supportive, or neutral statements. In students with a prior history of depression, the dorsolateral prefrontal cortex failed to activate in response to maternal criticism, although activation in response to criticism was observed among students with no depression history. The dorsolateral prefrontal cortex plays a major role in working memory, problem-solving, affective expression, interpersonal effectiveness, and the conscious control of behavior.

These and other cognitive vulnerabilities of patients may affect their processing of critical comments from relatives and may contribute to their overall levels of distress. A longitudinal study found that bipolar patients who reported being more emotionally distressed by criticisms from relatives had higher depression scores and fewer days well during a 1-year prospective period than did patients who reported less distress from criticisms (Miklowitz, Wisniewski, Miyahara, Otto, & Sachs, 2005). Patients who become especially distressed by signs of interpersonal rejection from family members may internalize the content of criticisms, which may contribute to their subsequent mood dysregulation.

Thus, high-EE attitudes emerge through a complex interplay between historical events, personal variables, attributional styles, and current relationship factors. Family interventions should therefore consider (a) the developmental processes by which high-EE attitudes originated, (b) the ways in which patients process and react to negative affect from parents or other caregivers, (c) the cognitive reactivity of relatives, (d) the ability of patients to manage stressful family interactions, and (e) the behaviors of patients that provoke negativity among caregivers.

FAMILY INTERVENTION FOR BIPOLAR DISORDER

Family interventions for bipolar disorder are *psychoeducational* in orientation, meaning that families (spouses, parents) and patients are taught to recognize the signs and symptoms of bipolar disorder, develop strategies for intervening early with new episodes, and assure consistency with medication regimens (see Box 1). The psychoeducational approach recognizes that lack of information about the disorder, along with uncertainties about the future, fuel patients' denial of the diagnosis and contribute to caregivers' high-EE attitudes. Thus, in addition to providing prescriptive information, clinicians address the patients' and family members' affective reactions to the illness, its prognosis, and its expected treatments and assist them in developing coping strategies relevant to their individual situation.

BOX 1
Key Features of Family-Focused Treatment

- Commences shortly after an acute episode of mania, depression, or mixed disorder
- Involves the patient and one or more relatives (spouse, parents, siblings)
- Conducted in 21 sessions over 9 months (weekly for 3 months, biweekly for 3 months, monthly for 3 months)
- Consists of three consecutive modules:
  1. *Psychoeducation*: didactic information and interactive discussion about the symptoms of bipolar disorder, early warning signs, relapse prevention plans, roles of risk and protective factors, and the importance of medication adherence (7 sessions)
  2. *Communication enhancement training*: behavioral rehearsal of effective speaking, listening, and negotiating skills, with homework practice (7–10 sessions)
  3. *Problem-solving skills training*: identify and define specific family problems, brainstorm solutions, evaluate the advantages and disadvantages of each solution, choose one or a combination of solutions, develop implementation plans; homework between sessions (4–5 sessions)

  Booster sessions as needed

The first randomized trial of FFT (Miklowitz, George, Richards, Simoneau, & Suddath, 2003) involved 101 bipolar patients (mean age = 36 years; mean prior episodes = 6.4) who had had an acute episode of mania or depression in the 3 months prior to the trial. Of the 101 patients, 82 began the trial in the hospital. Patients were randomly assigned as outpatients to FFT and drug therapy or to a comparison crisis-management treatment consisting of two sessions of family psychoeducation, crisis-intervention sessions as needed over a period of 9 months, and drug therapy. Over a 2-year follow-up, patients in FFT were more likely to survive the full follow-up without relapsing (52%) than were patients in crisis management (17%) and also had less severe depressive and manic symptoms (Miklowitz et al., 2003; see Fig. 2).

A second trial (Rea et al., 2003) compared FFT and pharmacotherapy with an individual psychoeducational therapy of identical duration and intensity (21 sessions over 9 months) and equivalent drug therapy. Over a period of 2 to 3 years, patients in FFT had longer periods of stability prior to recurrence than did patients in individual therapy. In the 1- to 2-year period after treatment ended, only 12% of the FFT patients were rehospitalized, compared to 60% of the individual therapy patients.

The multisite Systematic Treatment Enhancement Program for Bipolar Disorder contrasted the effectiveness of FFT and other psychotherapies to a brief psychosocial intervention across 15 U.S. treatment centers (Miklowitz et al., 2007). Acutely depressed bipolar patients ($N = 293$) were randomly assigned to one of three intensive (30 sessions) psychotherapies with drug therapy—FFT, interpersonal and social-rhythm therapy (therapy focused on interpersonal problem solving and the regulation

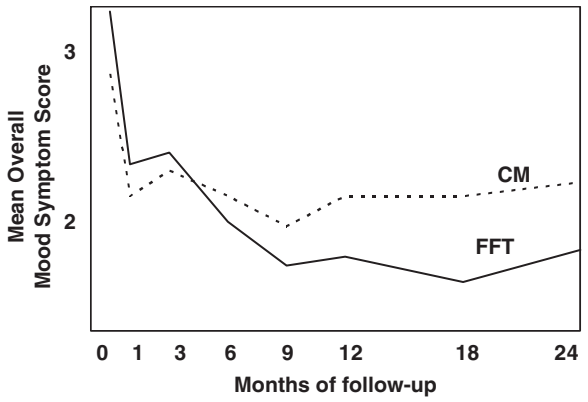

Fig. 2. Results of a 2-year randomized trial ($N = 101$) showing that family-focused treatment (FFT; $n = 31$) and medications improve bipolar patients' mood symptoms more than crisis management (CM; $n = 70$) and medications. From "A Randomized Study of Family-Focused Psychoeducation and Pharmacotherapy in the Outpatient Management of Bipolar Disorder," by D.J. Miklowitz, E.L. George, J.A. Richards, T.L. Simoneau, and R.L. Suddath, 2003, *Archives of General Psychiatry, 60*, p. 909.

of daily routines and sleep–wake cycles), or cognitive-behavioral therapy—or to a minimal (3 sessions) "collaborative care" (CC) psychoeducational treatment and drug therapy. Over the course of 1 year, all three intensive psychotherapies were associated with more rapid recoveries (mean = 169 days) from bipolar depression in comparison with the recoveries of those assigned to CC (mean = 279 days). The rates of recovery were 64% for patients in intensive treatment (77% for FFT, 65% for interpersonal therapy, 60% for cognitive-behavioral therapy) and 52% for those in CC. Patients in the intensive treatments were also more likely to stay well during any given study month than were patients in CC. Differences among the three 30-session treatments were nonsignificant. The Systematic Treatment Enhancement Program concluded that intensive therapies developed in academic settings (including FFT) can be successfully exported to community settings in which clinicians have had minimal previous exposure to manual-based interventions.

TREATMENT MECHANISMS FOR FFT

The Miklowitz et al. (2003) trial identified two variables that mediated the effects of FFT on mania and depression, respectively: improvements in medication adherence and augmentation of positive family communication. Patients in FFT were more likely to adhere to lithium or anticonvulsant drug regimens than were patients in crisis management, and adherence was associated with less severe mania symptoms over the course of 2 years. Patient–relative interactions were more positively toned after FFT than they were after crisis management, and improvements in patient–relative interaction were correlated with improvements in depressive symptoms among patients over the course of 1 year. Negative communication did not change in either treatment condition. Thus, FFT may enhance the protective qualities of family relationships rather than directly reducing the frequency of criticisms or aversive patterns of family interaction.

CONCLUSIONS

Episodes of bipolar disorder are strongly associated with family discord, criticism, and conflict. There is increasing evidence that family psychoeducational treatments are effective in relapse prevention and symptom control when combined with standard drug treatment.

Much remains to be learned about the subpopulations of patients most likely to benefit from family interventions. It is not clear, for example, whether only patients with high-EE families should be given FFT, and whether patients who do not report significant family conflicts or who are disengaged from their families would be better suited to individual or group approaches. The mediating mechanisms by which family interventions achieve their effects—which may include enhancing medication adherence, family communication and problem-solving, or the family's ability to recognize and intervene with early warning signs of recurrence—deserve further examination in randomized trials that measure mediators at systematically controlled intervals.

Longitudinal high-risk studies should clarify which family risk or protective processes operate among children who are genetically at risk for bipolar disorder. Specifically, investigators should identify early childhood temperamental or symptom attributes that bear a resemblance to manic or depressive symptoms (e.g., extreme moodiness or impulsiveness), clarify the circumstances under which these attributes evoke criticism or overprotectiveness among parents, and determine which of these children actually develop bipolar disorder in adulthood. It will be important in such studies to measure the psychiatric background of parents, as well as protective factors (e.g., a supportive secondary parent) that may reduce the likelihood that the child develops the disorder under conditions of high genetic risk.

Two uncontrolled treatment trials found that FFT alone or the combination of FFT and cognitive-behavioral therapy helped stabilize the course of bipolar disorder in adolescent and school-aged children, respectively (Miklowitz, Biuckians, & Richards, 2006; Pavuluri et al., 2004). Early preventative interventions involving the family are currently being developed (Miklowitz et al., 2006). Notably, teaching communication, problem-solving, and emotional self-regulation skills to at-risk children and their parents may help ameliorate stress within the family and contribute to delaying the onset of full manic episodes. Early-intervention studies involving high-risk populations should be a central focus for the next generation of research on bipolar disorder.

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might require the joint efforts of philosophers, neuroscientists, and cognitive psychologists.

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Computer- and Internet-Based Psychotherapy Interventions

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Abstract

Computers and Internet-based programs have great potential to make psychological assessment and treatment more cost-effective. Computer-assisted therapy appears to be as effective as face-to-face treatment for treating anxiety disorders and depression. Internet support groups also may be effective and have advantages over face-to-face therapy. However, research on this approach remains meager.

Keywords

computer applications; Internet applications; psychotherapy and technology

In recent years, the increasing number of users of computer and Internet technology has greatly expanded the potential of computer- and Internet-based therapy programs. Computer- and Internet-assisted assessment methods and therapy programs have the potential to increase the cost-effectiveness of standardized psychotherapeutic treatments by reducing contact time with the therapist, increasing clients' participation in therapeutic activities outside the standard clinical hour, and streamlining input and processing of clients' data related to their participation in therapeutic activities. Unfortunately, the scientific study of these programs has seriously lagged behind their purported potential, and these interventions pose important ethical and professional questions.

COMPUTER-BASED PROGRAMS

Information

A number of studies have demonstrated that computers can provide information effectively and economically. An analysis of a large number of studies of computer-assisted instruction (CAI) found that CAI is consistently effective in improving knowledge (Fletcher-Flinn & Gravatt, 1995). Surprisingly, few studies evaluating the use of CAI for providing information related to mental health or psychotherapy have been conducted.

Assessment

Traditional paper-based self-report instruments are easily adapted to the computer format and offer a number of advantages that include ensuring data completeness and standardization. Research has found that computer-administered assessment instruments work as well as other kinds of self-report instruments and as well as therapist-administered ones. Clients may feel less embarrassed about reporting sensitive or potentially stigmatizing information (e.g., about sexual behavior or illegal drug use) during a computer-assisted assessment than during a face-to-face assessment, allowing for more accurate estimates of mental health behaviors. Studies show that more symptoms, including suicidal thoughts, are reported during computer-assisted interviews than face-to-face interviews. Overall, the evidence suggests that computers can make assessments more efficient, more accurate, and less expensive. Yet computer-based assessment interviews do not allow for clinical intuition and nuance, assessment of behavior, and nonverbal emotional expression, nor do they foster a therapeutic alliance between client and therapist as information is collected.

Recently, handheld computers or personal digital assistants (PDAs) have been used to collect real-time, naturalistic data on a variety of variables. For example, clients can record

their thoughts, behaviors, mood, and other variables at the same time and when directed to do so by an alarm or through instructions from the program. The assessment of events as they occur avoids retrospective recall biases. PDAs can be programmed to beep to cue a response and also to check data to determine, for instance, if responses are in the right range. The data are easily downloaded into computer databases for further analysis. PDAs with interactive transmission capabilities further expand the potential for real-time data collection. Although PDAs have been demonstrated to be useful for research, they have not been incorporated into clinical practice.

Computer-Assisted Psychotherapy

Much research on computer-based programs has focused on anxiety disorders (Newman, Consoli, & Taylor, 1997). Researchers have developed computer programs that direct participants through exercises in relaxation and restfulness; changes in breathing frequency, regularity, and pattern; gradual and progressive exposure to aspects of the situation, sensation, or objects they are afraid of; and changes in thinking patterns. Although the majority of studies report symptom reduction, most are uncontrolled trials or case studies and have additional methodological weaknesses (e.g., small sample sizes, no follow-up to assess whether treatment gains are maintained, focus on individuals who do not have clinical diagnoses).

Computer programs have been developed to reduce symptoms of simple phobias, panic disorder, obsessive-compulsive disorder (OCD), generalized anxiety disorder, and social phobia. In a multicenter, international treatment trial (Kenardy et al., 2002), study participants who received a primary diagnosis of panic disorder were randomly assigned to one of four groups: (a) a group that received 12 sessions of therapist-delivered cognitive behavior therapy (CBT), (b) a group that received 6 sessions of therapist-delivered CBT augmented by use of a handheld computer, (c) a group that received 6 sessions of therapist-delivered CBT augmented with a manual, or (d) a control group that was assigned to a wait list. Assessments at the end of treatment and 6 months later showed that the 12-session CBT and the 6-session CBT with the computer were equally effective. The results suggested that use of a handheld computer can reduce therapist contact time without compromising outcomes and may speed the rate of improvement.

An interactive computer program was developed to help clients with OCD, which is considered one type of anxiety disorder. The computer provided three weekly 45-min sessions of therapy involving vicarious exposure to their obsessive thoughts and response prevention (a technique by which clients with OCD are taught and encouraged not to engage in their customary rituals when they have an urge to do so). Compared with a control group, the clients who received the intervention had significantly greater improvement in symptoms. In a follow-up study with clients diagnosed with OCD, computer-guided telephone behavior therapy was effective; however, clinician-guided behavior therapy was even more effective. Thus, computer-guided behavior therapy can be a helpful first step in treating patients with OCD, particularly when clinician-guided behavior therapy is unavailable. Computers have also been used to help treat individuals with other anxiety disorders, including social phobia and generalized anxiety disorder, a condition characterized by excessive worry and constant anxiety without specific fears or avoidances.

CBT also has been adapted for the computer-delivered treatment of depressive disorders. Selmi, Klein, Greist, Sorrell, and Erdman (1990) conducted the only randomized, controlled treatment trial comparing computer- and therapist-administered CBT for depression. Participants who met the study's criteria for major, minor, or intermittent depressive disorder were randomly assigned to computer-administered CBT, therapist-administered CBT, or a wait-list control. Compared with the control group, both treatment groups reported significant improvements on depression indices. The treatment groups did not differ from each other, and treatment gains were maintained at a 2-month follow-up.

Little information exists on the use of computer-assisted therapy for treating patients with complicated anxiety disorders or other mental health problems. Thus, further study is needed.

THE INTERNET

Internet-based programs have several advantages over stand-alone computer-delivered programs. The Internet makes health care information and programs accessible to individuals who may have economic, transportation, or other restrictions that limit access to face-to-face services. The Internet is constantly available and accessible from a variety of locations. Because text and other information on the Internet can be presented in a variety of formats, languages, and styles, and at various educational levels, it is possible to tailor messages to the learning preferences and strengths of the user. The Internet can facilitate the collection, coordination, dissemination, and interpretation of data. These features allow for interactiv-

ity among the various individuals (e.g., physicians, clients, family members, caregivers) who may participate in a comprehensive treatment plan. As guidelines, information, and other aspects of programs change, it is possible to rapidly update information on Web pages. The medium also allows for personalization of information. Users may select features and information most relevant to them, and, conversely, programs can automatically determine a user's needs and strengths and display content accordingly.

Information

Patients widely search the Internet for mental health information. For example, the National Institute of Mental Health (NIMH) public information Web site receives more than 7 million "hits" each month. However, the mental health information on commercial Web sites is often inaccurate, misleading, or related to commercial interests. Sites sponsored by nonprofit organizations provide better and more balanced information, but search engines often list for-profit sites before they generate nonprofit sites. Furthermore, education Web sites rarely follow solid pedagogical principles.

Screening and Assessment

Many mental health Web sites have implemented screening programs that assess individuals for signs or symptoms of various psychiatric disorders. These programs generally recommend that participants who score above a predetermined cutoff contact a mental health provider for further assessment. The NIMH and many other professional organizations provide high-quality, easily accessible information combined with screening instruments. Houston and colleagues (2001) evaluated the use of a Web site that offered a computerized version of the Center for Epidemiological Studies' depression scale (CES-D; Ogles, France, Lunnen, Bell, & Goldfarb, 1998). The scale was completed 24,479 times during the 8-month study period. Fifty-eight percent of participants screened positive for depression, and fewer than half of those had previously been treated for depression. The Internet can incorporate interactive screening, which already has been extensively developed for desktop computers. Screening can then be linked to strategies that are designed to increase the likelihood that a participant will accept a referral and initiate further assessment or treatment.

On-Line Support Groups

Because Internet-delivered group interventions can be accessed constantly from any location that has Internet access, they offer distinct advantages over their face-to-face counterparts. Face-to-face support groups often are difficult to schedule, meet at limited times and locations, and must accommodate inconsistent attendance patterns because of variations in participants' health status and schedules. On-line groups have the potential to help rural residents and individuals who are chronically ill or physically or psychiatrically disabled increase their access to psychological interventions.

A wide array of social support groups is available to consumers in synchronous (i.e., participants online at the same time) or asynchronous formats. The Pew Internet and American Life Project (www.pewinternet.org) estimated that 28% of Internet users have attended an on-line support group for a medical condition or personal problem on at least one occasion. After a morning television show featured Edward M. Kennedy, Jr., promoting free on-line support groups sponsored by the Wellness Community (www.wellness-community.org), the organization received more than 440,000 inquiries during the following week! The majority of published studies on Internet-based support groups suggest that the groups are beneficial; however, scientific understanding of how and when is limited. Studies that examine the patterns of discourse that occur in these groups indicate that members' communication is similar to that found in face-to-face support groups (e.g., high levels of mutual support, acceptance, positive feelings).

Only a few controlled studies have examined the effects of Internet-based support programs. One such study investigated the effects of a program named Bosom Buddies on reducing psychosocial distress in women with breast cancer (Winzelberg et al., in press). Compared with a wait-list control group, the intervention group reported significantly reduced depression, cancer-related trauma, and perceived stress.

On-Line Consultation

On-line consultation with "experts" is readily available on the Internet. There are organizations for on-line therapists (e.g., the International Society for Mental Health Online, www.ismpo.org) and sites that verify the credentials of on-line providers. However, little is known about the efficacy, reach, utility, or other aspects of on-line consultation.

Advocacy

The Internet has become an important medium for advocacy and political issues. Many organizations

use the Internet to facilitate communication among members and to encourage members to support public policy (e.g., the National Alliance for the Mentally Ill, www.nami.org).

Internet-Based Psychotherapy

The Internet facilitates the creation of treatment programs that combine a variety of interactive components. The basic components that can be combined include psychoeducation; social support; chat groups; monitoring of symptoms, progress, and use of the program; feedback; and interactions with providers. Although many psychotherapy programs developed for desktop computers and manuals are readily translatable to the Internet format, surprisingly few have been adapted in this way, and almost none have been evaluated. Studies show that Internet-based treatments are effective for reducing symptoms of panic disorder. Compared with patients in a wait-list control group, those who participated in an Internet-based posttraumatic stress group reported significantly greater improvements on trauma-related symptoms. During the initial 6-month period of operation, an Australian CBT program for depression, MoodGYM, had more than 800,000 hits (Christensen, Griffiths, & Korten, 2002). In an uncontrolled study of a small subsample of participants who registered on this site, program use was associated with significant decreases in anxiety and depression. Internet-based programs also have been shown to reduce symptoms of eating disorders and associated behaviors. Users consistently report high satisfaction with these programs.

Treatment programs for depression, mood swings, and other mental health disorders are being designed to blend computer-assisted psychotherapy and psychoeducation with case management (in which a therapist helps to manage a client's problems by following treatment and therapy guidelines) and telephone-based care. These programs might also include limited face-to-face interventions, medication, and support groups. The effectiveness of these programs remains to be demonstrated.

Eventually, the most important use of the Internet might be to deliver integrated, home-based, case-managed, psychoeducational programs that are combined with some face-to-face contact and support groups. Unfortunately, although a number of such programs are "under development," none have been evaluated in controlled trials.

ETHICAL AND PROFESSIONAL ISSUES

Web-based interventions present a number of ethical and professional issues (Hsiung, 2001). Privacy is perhaps the most significant concern. The Internet creates an environment where information about patients can be easily accessed and disseminated. Patients may purposely or inadvertently disclose private information about themselves and, in on-line support groups, about their peers. Although programs can be password-protected, and electronic records must follow federal privacy guidelines, participants must be clearly informed that confidentiality of records cannot be guaranteed.

Internet interventions create the potential that services will be provided to patients who have not been seen by a professional or who live in other states or countries where the professionals providing the services are not licensed to provide therapy. Professional organizations are struggling to develop guidelines to address these concerns (e.g., Hsiung, 2001; Kane & Sands, 1998).

Because of its accessibility and relative anonymity, patients may use the Internet during crises and report suicidal and homicidal thoughts. Although providers who use Internet support groups develop statements to clearly inform patients that the medium is not to be used for psychiatric emergencies, patients may ignore these instructions. Thus, providers need to identify ancillary procedures to reduce and manage potential crises.

Given the continuing advances in technology and the demonstrated effectiveness and advantages of computer- and Internet-based interventions, one might expect that providers would readily integrate these programs into their standard care practice. Yet few do, in part because programs that are easy to install and use are not available, there is no professional or market demand for the use of computer-assisted therapy, and practitioners may have ethical and professional concerns about applying this technology in their clinical practice. Thus, in the near future this technology may primarily be used for situations in which the cost-effectiveness advantages are particularly great.

CONCLUSION

Computers have the potential to make psychological assessments more efficient, more accurate, and less expensive. Computer-assisted therapy appears to be as effective as face-to-face therapy for treating anxiety disorders and depression and can be delivered at lower cost. However, applications of this technology are in the early stages.

A high priority is to clearly demonstrate the efficacy of this ap-

proach, particularly compared with standard face-to-face, "manualized" treatments that have been shown to be effective for common mental health disorders. Studies that compare two potentially efficacious treatments require large samples for us to safely conclude that the therapies are comparable if no statistically significant differences are found. Kenardy et al. (2002) demonstrated that multisite, international studies sampling large populations could be conducted relatively inexpensively, in part because the intervention they examined was standardized. If a treatment's efficacy is demonstrated, the next step would be to determine if the therapy, provided by a range of mental health professionals, is useful in large, diverse populations. Examination of combinations of therapies (e.g., CBT plus medication) and treatment modalities (Taylor, Cameron, Newman, & Junge, 2002) should follow. As the empirical study of this technology advances, research might examine the utility and cost-effectiveness of adapting these approaches to treating everyone in a community who wants therapy.

Continued use of the Internet to provide psychosocial support and group therapy is another promising avenue. As in the case of individual therapy, research is needed to compare the advantages and disadvantages between Internet and face-to-face groups, determine which patients benefit from which modality, compare the effectiveness of professionally moderated groups and self- or peer-directed groups, and compare the effectiveness of synchronous and asynchronous groups.

As research progresses, new and exciting applications can be explored. Because on-line text is stored, word content can be examined. This information may teach us more about the therapeutic process or may automatically alert providers to patients who are depressed, dangerous, or deteriorating.

Although research in many aspects of computer-assisted therapy is needed, and the professional and ethical concerns are substantial, computers and the Internet are likely to play a progressively important role in providing mental health assessment and interventions to clients. Thus, mental health professionals will need to decide how they will incorporate such programs into their practices.

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Gender Differences in Depression

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Abstract

From early adolescence through adulthood, women are twice as likely as men to experience depression. Many different explanations for this gender difference in depression have been offered, but none seems to fully explain it. Recent research has focused on gender differences in stress responses, and in exposure to certain stressors. I review this research and describe how gender differences in stress experiences and stress reactivity may interact to create women's greater vulnerability to depression.

Keywords

gender; depression; stress

Across many nations, cultures, and ethnicities, women are about twice as likely as men to develop depression (Nolen-Hoeksema, 1990; Weissman et al., 1996). This is true whether depression is indexed as a diagnosed mental disorder or as subclinical symptoms. Diagnosable depressive disorders are extraordinarily common in women, who have a lifetime prevalence for major depressive disorder of 21.3%, compared with 12.7% in men (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993).

Most explanations for the gender difference in depression have focused on individual variables, and studies have attempted to show that one variable is better than another in explaining the difference. In three decades of research, however, no one variable has single-handedly accounted for the gender difference in depression. In recent years, investigators have moved toward more integrated models, taking a transactional, developmental approach. Transactional models are appropriate because it is clear that depression impairs social and occupational functioning, and thus can have a major impact on an individual's environment. Developmental models are appropriate because age groups differ markedly in the gender difference in depression. Girls are no more likely than boys to evidence depression in childhood, but by about age 13, girls' rates of depression begin to increase sharply, whereas boys' rates of depression remain low, and may even decrease. By late adolescence, girls are twice as likely as boys to be depressed, and this gender ratio remains more or less the same throughout adulthood. The absolute rates of depression in women and men vary substantially across the life span, however.

In this review, I focus on two themes in recent research. First, because women have less power and status than men in most societies, they experience certain traumas, particularly sexual abuse, more often than men. They also experience more chronic strains, such as poverty, harassment, lack of respect, and constrained choices. Second, even when women and men experience the same stressors, women may be more likely than men to develop depression because of gender differences in biological responses to stressors, self-concepts, or coping styles.

Frequent stressful experiences and reactivity to stress are likely to have reciprocal effects on each other. Stressful experiences can sensitize both biological and psychological systems to future stress, making it more likely that individuals will react with depression. In turn, reactivity to stress is associated with impaired problem solving, and, as a result, with the accumulation or generation of new stressors, which may contribute to more depression.

STRESSFUL LIFE EVENTS

Women's lack of social power makes them more vulnerable than men to specific major traumas, particularly sexual abuse. Traumas may contribute directly to depression, by making women feel they are helpless to control their lives, and may also contribute indirectly, by increasing women's reactivity to stress. Women's social roles also carry a number of chronic strains that might contribute directly or indirectly to depression. Major changes in the frequency of traumatic events and in social roles coincide with the emergence of gender differences in depression in adolescence, and may help to explain this emergence.

Victimization

Women are the victims of sexual assault—defined as being pressured or forced into unwanted sexual contact—at least twice as often as men, and people with a history of sexual assault have increased rates of depression (see Weiss, Longhurst, & Mazure, 1999). Sexual assault during childhood has been more consistently linked with the gender difference in depression than sexual assault that first occurs during adulthood. Estimates of the prevalence of childhood sexual assault range widely. Cutler and I reviewed the most methodologically

sound studies including both male and female participants and found rates of childhood sexual assault between 7 and 19% for females and between 3 and 7% for males (Cutler & Nolen-Hoeksema, 1991). We estimated that, in turn, as much as 35% of the gender difference in adult depression could be accounted for by the higher incidence of assault of girls relative to boys. A few studies have examined whether depression might be an antecedent rather than a consequence of sexual assault. Depression does appear to increase risk for sexual assault in women and men, but sexual assault significantly increases risk for first or new onsets of depression.

Childhood sexual assault may increase risk for depression throughout the life span because abuse experiences negatively alter biological and psychological responses to stress (Weiss et al., 1999). Children and adolescents who have been abused, particularly those who have been repeatedly abused over an extended period of time, tend to have poorly regulated biological response to stress. Abuse experiences can also negatively alter children's and adolescents' perspectives on themselves and others, contributing to their vulnerability to depression (Zahn-Waxler, 2000).

Chronic Strains

Women face a number of chronic burdens in everyday life as a result of their social status and roles relative to men, and these strains could contribute to their higher rates of depression (see Nolen-Hoeksema, 1990). Women make less money than men, and are much more likely than men to live in poverty. Women are more likely than men to be sexually harassed on the job. Women often have full-time paid jobs and also do nearly all the child care and domestic work of the home. In addition, women are increasingly "sandwiched" between caring for young children and caring for sick and elderly family members. This role overload is said to contribute to a sense of "burn out" and general distress, including depressive symptoms, in women.

In the context of heterosexual relationships, some women face inequities in the distribution of power over important decisions that must be made, such as the decision to move to a new city, or the decision to buy an expensive item such as a car (Nolen-Hoeksema, Larson, & Grayson, 1999). Even when they voice their opinions, women may feel these opinions are not taken seriously, or that their viewpoints on important issues are not respected and affirmed by their partners. My colleagues and I measured chronic strain by grouping inequities in workload and heterosexual relationships into a single variable, and found that this variable predicted increases in depression over time, and partially accounted for the gender difference in depression (Nolen-Hoeksema et al., 1999). Depression also contributed to increased chronic strain over time, probably because it was associated with reductions in perceptions of control and effective problem solving.

Gender Intensification in Adolescence

Social pressure to conform to gender roles is thought to increase dramatically as children move through puberty. For girls, this may mean a reduction in their opportunities and choices, either real or perceived. According to adolescents' own reports, parents restrict girls' more than boys' behaviors and have lower expectations for girls' than for boys' competencies and achievements. Girls also feel that if they pursue male-stereotyped activities and preferences, such as interests in math and science or in competitive sports, they are rejected by their peers. For many girls, especially white girls, popularity and social acceptance become narrowly oriented around appearance.

This narrowing of acceptable behavior for girls in early adolescence may contribute to the increase in depression in girls at this time, although this popular theory has been the focus of remarkably little empirical research (Nolen-Hoeksema & Girgus, 1994). There is substantial evidence that excessive concern about appearance is negatively associated with well-being in girls, but these findings may apply primarily to white girls. In addition, very little research has examined whether appearance concerns and gender roles are risk factors for depression or only correlates.

REACTIVITY TO STRESS

Even when women and men are confronted with similar stressors, women may be more vulnerable than men to developing depression and related anxiety disorders such as posttraumatic stress disorder (Breslau, Davis, Andreski, Peterson, & Schultz, 1997). Women's greater reactivity compared with men's has been attributed to gender differences in biological responses, self-concepts, and coping styles.

Biological Responses to Stress

For many years, the biological explanations for women's greater vulnerability to depression focused on the direct effects of the ovarian hormones (especially estrogen and progesterone) on women's moods. This literature is too large and com-

plicated to review here (but see Nolen-Hoeksema, 1990, 1995). Simply put, despite widespread popular belief that women are more prone to depression than men because of direct negative effects of estrogen or progesterone on mood, there is little consistent scientific evidence to support this belief. Although some women do become depressed during periods of hormonal change, including puberty, the premenstrual period of the menstrual cycle, menopause, and the postpartum period, it is unclear that these depressions are due to the direct effects of hormonal changes on mood, or that depressions during these periods of women's lives account for the gender differences in rates of depression.

More recent biological research has focused not on direct effects of ovarian hormones on moods, but on the moderating effects of hormones, particularly adrenal hormones, on responses to stress. The hypothalamic-pituitary-adrenal (HPA) axis plays a major role in regulating stress responses, in part by regulating levels of a number of hormones, including cortisol, which is released by the adrenal glands in response to chemicals secreted by the brain's hypothalamus and then the pituitary. In turn, cortisol levels can affect other biochemicals known to influence moods. People with major depressive disorder often show elevated cortisol responses to stress, indicating dysregulation of the HPA response.

An intriguing hypothesis is that women are more likely than men to have a dysregulated HPA response to stress, which makes them more likely to develop depression in response to stress (Weiss et al., 1999). Women may be more likely to have a dysregulated HPA response because they are more likely to have suffered traumatic events, which are known to contribute to HPA dysregulation. In addition, ovarian hormones modulate regulation of the HPA axis (Young & Korszun, 1999). Some women may have depressions during periods of rapid change in levels of ovarian hormones (the postpartum period, premenstrual period, menopause, and puberty) because hormonal changes trigger dysregulation of the stress response, making these women more vulnerable to depression, particularly when they are confronted with stress. The causal relationship between HPA axis regulation and the gender difference in depression has not been established but is likely to be a major focus of future research.

Self-Concept

Although the idea that girls have more negative self-concepts than boys is a mainstay of the pop-psychology literature, empirical studies testing this hypothesis have produced mixed results (Nolen-Hoeksema & Girgus, 1994). Several studies have found no gender differences in self-esteem, self-concept, or dysfunctional attitudes. Those studies that do find gender differences, however, tend to show that girls have poorer self-concepts than boys. Again, negative self-concepts could contribute directly to depression, and could interact with stressors to contribute to depression. Negative self-concept has been shown to predict increases in depression in some studies of children (Nolen-Hoeksema & Girgus, 1994).

One consistent difference in males' and females' self-concepts concerns interpersonal orientation, the tendency to be concerned with the status of one's relationships and the opinions others hold of oneself. Even in childhood, girls appear more interpersonally oriented than boys, and this gender difference increases in adolescence (Zahn-Waxler, 2000). When interpersonal orientation leads girls and women to subordinate their own needs and desires completely to those of others, they become excessively dependent on the good graces of others (Cyranowski, Frank, Young, & Shear, 2000). They may then be at high risk for depression when conflicts arise in relationships, or relationships end. Several recent studies have shown that girls and women are more likely than boys and men to develop depression in response to interpersonal stressors. Because depression can also interfere with interpersonal functioning, an important topic for future research is whether the gender difference in depression is a consequence or cause of gender differences in interpersonal strain.

Coping Styles

By adolescence, girls appear to be more likely than boys to respond to stress and distress with rumination—focusing inward on feelings of distress and personal concerns rather than taking action to relieve their distress. This gender difference in rumination then is maintained throughout adulthood. Several longitudinal and experimental studies have shown that people who ruminate in response to stress are at increased risk to develop depressive symptoms and depressive disorders over time (Nolen-Hoeksema et al., 1999). In turn, the gender difference in rumination at least partially accounts for the gender difference in depression. Rumination may not only contribute directly to depression, but may also contribute indirectly by impairing problem solving, and thus preventing women from taking action to overcome the stressors they face.

AN INTEGRATIVE MODEL

Women suffer certain stressors more often than men and may be

more vulnerable to develop depression in response to stress because of a number of factors. Both stress experiences and stress reactivity contribute directly to women's greater rates of depression compared with men. Stress experiences and stress reactivity also feed on each other, however. The more stress women suffer, the more hyperresponsive they may be to stress, both biologically and psychologically. This hyperresponsiveness may undermine women's ability to control their environments and overcome their stress, leading to even more stress in the future. In addition, depression contributes directly to more stressful experiences, by interfering with occupational and social functioning, and to vulnerability to stress, by inciting rumination, robbing the individual of any sense of mastery she did have, and possibly sensitizing the biological systems involved in the stress response.

Important advances will be made in explaining the gender difference in depression as we understand better the reciprocal effects of biological, social, and psychological systems on each other. Key developmental transitions, particularly the early adolescent years, are natural laboratories for observing the establishment of these processes, because so much changes during these transitions, and these transitions are times of increased risk.

Additional questions for future research include how culture and ethnicity affect the gender difference in depression. The gender difference is found across most cultures and ethnicities, but its size varies considerably, as do the absolute percentages of depressed women and men. The processes contributing to the gender difference in depression may also vary across cultures and ethnicities.

Understanding the gender difference in depression is important for at least two reasons. First, women's high rates of depression exact tremendous costs in quality of life and productivity, for women themselves and their families. Second, understanding the gender difference in depression will help us to understand the causes of depression in general. In this way, gender provides a valuable lens through which to examine basic human processes in psychopathology.

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Recalling the Unrecallable: Should Hypnosis Be Used to Recover Memories in Psychotherapy?

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Our observations have shown . . . that the memories which have become the determinants of hysterical phenomena persist for a long time with astonishing freshness and with the whole of their affective colouring . . . these experiences are completely absent from the patients' memory when they are in a normal psychical state, or are only present in highly summary form. Not until they have been questioned under hypnosis do these memories emerge with the undiminished vividness of a recent event. (Breuer & Freud, 1893–1895/1955, p. 9)

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Of course, not too long after this famous quote, Freud spurned hypnosis[2] in favor of other techniques such as free association, dream analysis, and interpretation. But the idea that hypnosis is a royal road to unconscious or suppressed memories lingers to the present day. Survey research (cf. Lynn, Myers, & Malinoski, in press) indicates that between 20% and 34% of modern psychotherapists use hypnosis to help patients "recall the unrecallable" and to establish the historical "truth" or basis of current problems. Hypnosis would be valuable in such instances if it were a reliable technique for recovering accurate memories. However, in this review, we contend this is not the case.

It is worth noting at the outset that a review of the use of hypnosis in forensic situations (see Karlin & Orne, 1996; Scheflin, in press) is beyond the scope of this article, and that when hypnotic procedures are combined with behavioral and psychophysiological procedures, there is a proven benefit for interventions that are not focused on retrieving memories (Kirsch, Montgomery, & Sapirstein, 1995). The concerns and caveats we present here apply specifically to the use of hypnosis as a technique for unearthing historically accurate memories[3] in psychotherapy.

ACCURATE AND INACCURATE MEMORIES IN HYPNOSIS

On the basis of his review of 34 studies, Erdelyi (1994) concluded that hypnosis does not increase recognition of previously presented meaningful stimuli (e.g., poetry, meaningful pictures) or recognition or recall of nonmeaningful stimuli (e.g., nonsense syllables, word lists). Although Erdelyi noted that hypnosis increases recall of meaningful stimuli, it also increases false recollections. Indeed, when hypnotic and nonhypnotic conditions are compared and the sheer volume of responses is controlled, hypnotic recall is no more accurate than nonhypnotic recall (e.g., Erdelyi, 1994).

Support for Erdelyi's conclusions can be found in a meta-analysis reported by Steblay and Bothwell (1994). Their analysis summarized 24 studies, among which were studies that appeared after those included in Erdelyi's review. Steblay and Bothwell found no reliable differences in performance on structured tests of accurate recall[4] when subjects were hypnotized versus when they were not hypnotized. It is true that three studies in Steblay and Bothwell's analysis did report a superiority of recall in hypnotized subjects when

recall was measured using unstructured free recall tests. However, in four more recent studies conducted in our laboratory (e.g., Abrams & Lynn, 1996), hypnotized subjects either fared no better or performed worse than nonhypnotized subjects on tests of accurate recall whether these tests were unstructured or structured. Furthermore, motivational instructions that urged subjects to "try your best on the recall test" yielded equivalent or superior recall compared with hypnosis.

Thus, the evidence does not seem to support the conclusion that hypnosis improves accurate recall. Whether hypnosis reduces inaccurate recall (e.g., distortions of presented stimuli, intrusions of nonpresented stimuli or events that never occurred) is a separate question. Recall errors are not uncommon. However, Steblay and Bothwell's (1994) analysis of six studies revealed that hypnotized participants, compared with nonhypnotized control subjects, produced more false memories in response to misleading questions or false information. Moreover, Steblay and Bothwell's analysis of five studies of recall revealed that hypnotized participants, compared with control subjects, generated more errors that were not prompted by misleading questions or the stimuli themselves. In short, hypnosis is not a reliable technique for augmenting accurate recall and generally results in a trade-off of errors for accurate remembrances.

Unwarranted Recall Confidence

Nonhypnotized persons are often overconfident about the accuracy of their memories (Spanos, 1996). However, hypnotized individuals are often (but not always) more confident about what they recall than nonhypnotized individuals, regardless of whether the information is accurate or not (Steblay & Bothwell, 1994). The magnitude of the overconfidence effect associated with hypnosis ranges from small to substantial, when present. An association between hypnotizability and confidence has also been documented, with highly hypnotizable participants particularly prone to what are called confident errors (i.e., being confident of inaccurate memories; Sheehan, 1988).

Hypnosis and Emotional Stimuli

It has been claimed that hypnosis may have particular utility as a memory recovery technique with traumatized persons because trauma blocks memory due to the state-dependent nature of memory. That is, retrieving a traumatic memory may depend on the congruence of the current context and mood with the context and mood at the time the event occurred, and hypnosis has the ability to reinstate those original conditions (Hammond et al., 1995, p. 15). This conclusion is not warranted or is questionable for the following reasons. First, although research has not compared hypnotic versus nonhypnotic recall in the presence of traumatic stimuli, studies with emotional and arousing yet not personally threatening stimuli (e.g., films of shop accidents and fatal stabbings, a mock "live" assassination, and a murder videotaped serendipitously) yield an unambiguous conclusion: Hypnosis does not improve recall of emotionally arousing events, and arousal level does not affect hypnotic recall (Lynn et al., in press). Second, controversy exists (Ofshe & Singer, 1994; Scheflin & Brown, 1996) regarding whether and to what degree emotional trauma can block memory for single, repeated, or prolonged events. And third, as Shobe and Kihlstrom note in this issue, hypnosis often involves relaxation suggestions that would not be expected to reinstate the traumatic context.

We agree that it is appropriate to question the generalizability of laboratory research to real traumatic situations, and to exhort researchers to devise creative designs that better approximate real-life situations. Nevertheless, the available evidence fails to support the contention that hypnosis has special promise for helping traumatized individuals regain lost memories.

Hypnotic Age Regression

In a review of more than 60 years of research on hypnotic age regression (a technique in which a subject is asked to respond to specific hypnotic suggestions to think, feel, or act like a child at a particular age), Nash (1987) found that the behaviors and experiences of age-regressed adults were often different from those of actual children. No matter how compelling such age-regression experiences appear to observers, they reflect participants' fantasies and beliefs and assumptions about childhood; they rarely, if ever, represent literal reinstatements of childhood experiences, behaviors, and feelings.

In one illustrative study (Nash, Drake, Wiley, Khalsa, & Lynn, 1986), subjects age-regressed to age 3 years reported the identity of their transitional objects (e.g., blankets, teddy bears). Parents of 14 hypnotized subjects and 10 role-playing control subjects were asked to verify this information. The results showed that hypnotized subjects were less accurate than control subjects in identifying the specific transitional objects they had used. Hypnotic subjects' hypnotic recollections, for example, matched their parents' reports only 21% of the time, whereas role-

players' reports were corroborated by their parents 70% of the time. This research, like other studies reported in the age-regression literature (cf. Nash, 1987), indicates that age-regression experiences can be compelling yet inaccurate.

DETERMINANTS OF PSEUDOMEMORIES

In the studies we review in this section, the usual procedure was to provide participants with deliberately misleading suggestions during hypnosis or nonhypnotic control procedures and measure the extent to which the participants accepted the false information as true following hypnosis or the control procedure. When such information is accepted, it is referred to as a pseudomemory. Sometimes people with high hypnotizability scores report more pseudomemories than people with medium hypnotizability scores, but in general, both groups report more pseudomemories than people with low hypnotizability scores. The fact that people with medium and even low hypnotizability scores report pseudomemories indicates that the effect is not limited to a small and highly select segment of the population (Lynn & Nash, 1994; Orne, Whitehouse, Dinges, & Orne, 1996). Interestingly, highly hypnotizable subjects report more pseudomemories than other people in nonhypnotic as well as hypnotic conditions, implicating a general suggestibility factor in the genesis of pseudomemories (see Lynn et al., in press).

The rates at which pseudomemories are reported are influenced by the perceived verifiability and memorability of the to-be-remembered events. Rates for distinctive events (e.g., a telephone ringing in a classroom) that do not often occur in the real world are generally low (12% to 25%) in hypnotic contexts. However, when the events are impossible to verify (e.g., whether a person was awakened on a particular night by a noise), or are not particularly memorable (e.g., a door slamming in a hall the previous week), pseudomemory rates are much higher (45% to 80%; Lynn et al., in press).

Situational variables are influential determinants of pseudomemory reports. Pseudomemory rates decrease (but are by no means eliminated) when previously hypnotized subjects are offered a monetary reward for distinguishing between a false suggestion and an actual occurrence, when rapport with the experimenter is degraded, and when subjects are cross-examined (cf. Lynn et al., in press).

Several studies (see Lynn et al., in press) have compared pseudomemory rates of hypnotized subjects with pseudomemory rates of nonhypnotized imagining subjects or of role-playing subjects instructed to respond in terms of their understanding of how hypnotized subjects would respond in the experimental situation. Because these studies used very leading suggestions, it is not surprising that hypnotized and nonhypnotized persons responded comparably. This research indicates that false memories are by no means limited to hypnotic conditions and underscores the role of perceptions of the situation and situational cues in the formation of pseudomemories. However, this research does not mean that hypnotized persons are not genuinely confused or misled with respect to the false information they remember.

In another line of research, McConkey, Labelle, Bibb, and Bryant (1990) found that if testing took place immediately after hypnosis, approximately 50% of hypnotizable subjects reported a pseudomemory. However, when subjects were contacted by telephone at home 4 to 24 hr later by an experimenter who was not part of the earlier session, the rate decreased dramatically to 2.5%. Barnier and McConkey (1992) found that the pseudomemory rate declined from 60% (for a false suggestion that a thief depicted in a series of slides was wearing a scarf) to 10% when the experimental context shifted to imply that the experiment had ended.

By questioning subjects at their homes by telephone after the formal experiment was completed, and by implying that the experiment was terminated, these studies might have engendered subtle pressure on subjects to reverse their earlier pseudomemory reports. Hence, this program of research does not satisfactorily resolve the issue of whether pseudomemory reports reflect genuine memory alterations or merely alterations in reports in conformance with variations in the situational context.

Although McConkey's research indicates that pseudomemory reports are malleable, other research indicates this is not always the case. For instance, Spanos and McLean (1986) showed that participants reversed their initial hypnotic pseudomemory reports when they were informed they could distinguish "real" and "false" memories if they accessed a "hidden observer" that could discriminate them. However, in three studies, we (see Lynn et al., in press) were unable to reverse pseudomemory reports by informing participants that they would be able to distinguish false and accurate memories. Hence, pseudomemory reports are not invariably sensitive to contextual manipulations and can be obdurate to modification.

The American Society of Clinical Hypnosis (ASCH) recently advanced guidelines (Hammond et al., 1995) intended to define principles of practice in the use of hyp-

nosis for exploring, uncovering, and working through memories. The guidelines refer to the potentially contaminating effects (e.g., increase in volume of information reported and confidence that what is recalled is true) of suggestions and expectations that memory will increase during hypnosis (e.g., "You can and will recall everything") and say that such effects may be controlled considerably "when neutral expectations are created prior to hypnosis and during hypnotic induction and age regression" (p. 28).

The guidelines inspired a recent study of prehypnotic expectancies (Green, Lynn, & Malinoski, in press) comparing the pseudomemory rates of highly hypnotizable participants "warned" prior to hypnosis that hypnosis can lead to false memories with pseudomemory rates of highly hypnotizable participants who received no special instructions prior to hypnosis. During hypnosis, all subjects were given the suggestion that they had been awakened by a noise during a night of the previous week.[5] Prior to hypnosis, all of the participants indicated they had slept through the night.

Participants who were warned were less likely to accept the suggestion during hypnosis: 38% of the warned participants did so, versus 75% of the unwarned participants. Hence, warnings reduced participants' suggestibility during hypnosis. However, an analysis of those persons who accepted the suggestion during hypnosis showed that the warning had no effect on their posthypnotic pseudomemories: Among this group, 75% of those persons who had been warned and 58% of those who had not been warned stated immediately after hypnosis that the noise had occurred in reality (i.e., reported a pseudomemory). After extensive questioning, during a final confidential assessment, 58% of the warned participants who had accepted the noise suggestion during hypnosis reported the pseudomemory, compared with 50% of the unwarned participants. Furthermore, warned participants were just as confident in their false memories as were unwarned participants.

In summary, when participants are warned about the deleterious effects of hypnosis on memory, suggestibility is reduced, but the risk of pseudomemories is by no means eliminated. Future research should evaluate the possibility that this risk will be further reduced when the full ASCH guidelines (e.g., avoiding leading questions and attempting to establish neutral expectations about the effects of hypnosis on memory prior to, during, and after hypnosis) are followed.

CONCLUSIONS

If clinicians were concerned only about accurate information, then hypnosis might be a useful memory recovery technique insofar as it can lower the threshold for reporting both accurate and inaccurate memories. To be sure, hypnosis does not always produce memory errors. Kluft (in press), for example, has reported that he was able to corroborate a number of hypnotically evoked memories of sexual abuse reported by patients in his clinical practice diagnosed with dissociative identity disorder (formerly known as multiple personality disorder). In most instances, however, it is not only impractical or inappropriate, but impossible to corroborate memories of patients in psychotherapy. Because clinicians and clients are not, as a rule, able to differentiate accurate and inaccurate memories, the yield of accurate memories must be weighed against the risk of memory errors associated with the hypnotic context. As Steblay and Bothwell (1994) concluded, "Hypnosis is not necessarily a source of accurate information; at worst it may be a source of inaccurate information provided with confident testimony" (p. 649).

Such concerns raise the question of whether hypnosis should be demonized and banished from the arena of psychotherapies. We contend that to do so would be a serious mistake that would deprive clinicians of a valuable technique that can be used successfully in many contexts outside that of memory recovery, including the treatment of persons who remember traumatic experiences without the use of any special techniques.

The literature on memory recovery and hypnosis is complex, and future research may change our assessment. For instance, if the available evidence indicated that safeguards can eliminate memory errors while preserving a recall advantage for hypnosis, we would acknowledge that hypnosis may have a useful role in improving recall. However, this has not, as yet, been demonstrated.

Nor has it been shown that the memory recovery component of psychotherapies contributes to their efficacy to begin with. Indeed, many nonhypnotic procedures geared toward memory recovery are inherently suggestive in nature (i.e., there is "something" to be recalled that will improve present functioning), and may well carry a pseudomemory risk equal to or greater than that of hypnosis. The attempt to recover suppressed memories is complex and risky business whether hypnosis is used or not. Certainly each clinician must ultimately weigh the costs versus the benefits of any psychotherapeutic technique. In our view, however, the data indicate that the answer to the question of whether hypnosis should be used to recover historically accurate memories in psychotherapy is "no."

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Notes

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2. The term hypnosis refers to a social situation in which a person designated as a hypnotist attempts to influence the experiences and behaviors of a subject or patient. The suggestions administered in the hypnotic situation typically call for changes in sensation, perception, affect, cognition, and control over behavior or psychophysiological processes (e.g., heart rate). Hypnotizability, or hypnotic responsiveness, refers to observed or reported responsivity to suggestions following a hypnotic induction. Participants range on a continuum of hypnotizability according to how many suggestions they accept, or pass. People who pass 3 or fewer suggestions out of 12 have a score that is conventionally considered low (about 15%–20% of the population), those who pass 4 to 8 suggestions have scores considered medium (about 60%–70% of the population), and those who pass 9 to 12 suggestions are regarded as highly hypnotizable (about 15%–20% of the population).

3. Of course, it could be, and has been, argued that memories retrieved during hypnosis, or any psychotherapeutic technique for that matter, need not be "historically accurate" to have therapeutic value. We acknowledge that all memories produced in psychotherapy and other contexts are not necessarily accurate, that memory is not an unbiased and permanent record of events as they unfolded in the past, and that it is possible that behavioral change in psychotherapy may come about regardless of the historical truth of memories. However, we agree with Spence (1994) and with Kihlstrom (in press) that, as a rule, "narrative truth is no substitute for historical truth" (Kihlstrom, p. 38), and that when clients place stock in false narratives, they may be diverted from confronting and resolving important issues in therapy.

4. In structured recall tests, subjects are required to respond to specific questions about the to-be-recalled material. Such tests can be contrasted with unstructured recall tests, in which subjects are not cued or questioned about the specific content of the information to be recalled (e.g., "Write down everything you can remember").

5. It is important to emphasize that because this study did not evaluate the full ASCH guidelines, generalization to the complete set of procedures mandated by ASCH may be hazardous.

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Who Develops Posttraumatic Stress Disorder?

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ABSTRACT—*Nearly half of U.S. adults experience at least one traumatic event in their lifetimes, yet only 10% of women and 5% of men develop posttraumatic stress disorder (PTSD). Why this is so is among the most central questions in current PTSD research. This article reviews the current status of knowledge about who develops PTSD, discussing the strengths and weaknesses of the evidence. We describe the major models used to understand responses to traumatic events, as well as future research directions. We also propose that an exclusive focus on individual differences and individual intervention overlooks opportunities to reduce the prevalence of PTSD by modifying factors at the neighborhood, community, or national level.*

KEYWORDS—*PTSD predictor; dissociation; traumatic event; prevention*

The response to traumatic stress varies widely, ranging from transient disruption of functioning to the chronic clinical condition known as posttraumatic stress disorder (PTSD). Interest in and knowledge about PTSD increased dramatically after its diagnosis was formalized in 1980, but study of the effects of extreme stress has a long history, primarily focused on the effects of war (e.g., shell shock in World War I) and of sexual assault against women. According to generally accepted criteria, diagnosis of PTSD requires exposure to a traumatic event that causes feelings of extreme fear, horror, or helplessness. Traumatic events are defined as experiences that involve death, serious injury, or threat of death. The consequences of this exposure are manifested in three symptom clusters required for diagnosis: involuntary reexperiencing of the trauma (e.g., nightmares, intrusive thoughts), avoidance of reminders and numbing of responsivity (e.g., not being able to have loving feelings), and increased arousal (e.g., difficulty sleeping or concentrating, hypervigilance, exaggerated startle response).

Because PTSD requires the presence of an external event and symptoms linked to this event, it differs from virtually all other psychiatric disorders and raises intriguing issues regarding the definition of trauma, the role of individuals' appraisal of and responses to the event, the implications of a single versus repeated or ongoing exposure, and the role of community- and societal-level changes in attempting to prevent PTSD.

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PREVALENCE

Results from a nationally representative study indicated that over the life course, 10% of women and 5% of men in the United States experience PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Moreover, approximately half of adults have experienced a traumatic event. In a national survey of Vietnam veterans conducted in the late 1980s, Kulka et al. (1990) estimated that 31% of males and 26% of females in this population had PTSD from their military service. Because PTSD symptoms wax and wane, especially in response to subsequent life events (not necessarily traumatic ones), many people experience *partial PTSD*, or clinically significant symptoms of PTSD that do not meet the diagnostic criteria for the disorder. Including individuals with partial PTSD resulted in an estimate of roughly 830,000 Vietnam veterans with significant posttraumatic distress or impairment approximately 20 years after service (Weiss et al., 1992).

The disparity between the 50% prevalence of exposure to trauma and the 7% lifetime prevalence of PTSD means that individual responses to trauma vary dramatically. This variability sparks what appears to be the key question in the field: Why do some people, and not others, develop PTSD? This issue has been of particular interest in recent years, leading to a search for systematic risk factors. Central questions have focused on the correlates or predictors of who develops the disorder and the strength of these effects. Current conceptualizations of PTSD symptoms provide potential explanatory frameworks for appreciating how predictors may influence the stress response and lead to differential risk for PTSD.

MODELS OF TRAUMA RESPONSE

Models Focused on Cognitive and Emotional Processes

The two most influential cognitively oriented formulations of trauma response and recovery highlight either the importance of beliefs and linked emotions about the self and the world (McCann & Pearlman, 1990) or the network of associations linking thinking about or reminders of a traumatic event to cognitive, emotional, physiological, and behavioral responses (Foa & Rothbaum, 1989). In the former formulation, a traumatic event is conceptualized as shattering the

previously held assumption that though the world is not always safe, the lack of safety affects other people only. Thus, the trauma victim's thinking about the world must be adapted to assimilate this shattered assumption and make sense of and integrate the event. The PTSD symptoms of intrusion and avoidance arise from this process, which is generally experienced as painful because it requires remembering the trauma and the accompanying distress. Recovery gradually occurs when this iterative process can be tolerated without avoidance or being overwhelmed emotionally. Thus, factors that reduce the likelihood of effective integration and assimilation would theoretically increase the likelihood of chronic stress-related symptoms and PTSD. These factors include characteristics of the individual, his or her environment, and the event itself.

In the latter cognitive formulation, the metaphor of a memory network is invoked to describe linked information about the traumatic event and subsequent cognitive, affective, physiological, and behavioral responses. Activation of one element in the network activates other aspects—almost always including fear—and this uninterrupted repetition accounts for the continuing symptoms. Recovery occurs if the strength of the associations among network components is reduced by a combination of desensitization and substitution of more adaptive associations.

Biologically Focused Models

Research on the biology of PTSD initially focused on studying psychophysiological arousal in the presence of reminders (sounds, images, or scripts) of the traumatic event. Results indicated that individuals with PTSD demonstrated heightened arousal and prolonged duration of arousal compared with control subjects (e.g., Keane et al., 1998). Recently, researchers investigating the biological substrates of PTSD have focused on the processes and structures of the brain. Research has centered on the amygdala and hippocampus, key brain areas involved in the fear response and in the consolidation of memory (e.g., LeDoux, 2000), as well as on the hypothalamic-pituitary-adrenal (HPA) axis, the parts of the neuroendocrine system that control reactions to acute stress.

Examination of parts of the brain involved in the fear response has been extensive because traumatic events usually generate fear, and because fear initiates the "flight or fight" physiological arousal associated with the hyperarousal symptoms of PTSD. Fear has also been implicated in the mechanisms establishing and maintaining traumatic memories. Research in animals has generally examined brain circuitry; research in humans has included neuroimaging studies of brain structures (Schuff et al., 1997) and processes (Rauch et al., 1996). New findings from animal studies have established direct neural pathways from sensory input to areas of the amygdala. In light of the known reciprocal neuronal connections between the hippocampus and amygdala, these findings suggest a powerful explanation for the automaticity of the fear response and the manner in which emotional memories occur and are transmitted to the hippocampus.

Careful study of individuals with PTSD indicates that they are characterized by an oversensitivity of the HPA axis. The HPA axis is involved in generating, maintaining, and shutting down increases in stress-related hormones in the face of danger, a central aspect of traumatic events. Evidence suggests that individuals with PTSD exhibit dysregulation in the activity of cortisol, a hormone regulated by the HPA axis. The destructive effects of the excessive production of cortisol are believed to be responsible for the atrophy of the hippocampus frequently found among individuals with chronic PTSD. The dysregulation in the HPA axis involves the feedback loop that puts the brakes on the arousal generated by the perception of fear (Yehuda, 1997). These findings have generated research aimed at exploring the use of medications such as beta-blockers to dampen initial arousal. With initial arousal dampened, the consolidation of emotional memories may be attenuated. The hope, therefore, is that the reduction of physiological arousal immediately after the traumatic event will interfere with the processes that lead to the development of PTSD.

PREDICTORS OF PTSD

Two major meta-analyses (statistical analyses combining the results of many studies) of the predictors of PTSD have recently been published (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003). These studies examined four categories of predictors: (a) historical or static characteristics such as family psychiatric history, intelligence, childhood trauma, and other previous trauma; (b) trauma severity; (c) psychological processes during and immediately after the trauma; and (d) social support and life stress after the traumatic event. Both meta-analyses showed that there were significant predictors of PTSD in all four categories, but that the strength of prediction varied across the categories. Those factors closer in time to the traumatic event (i.e., proximal factors) showed a stronger relationship to PTSD ($r \approx .40$) than did characteristics of the individual or his or her history that were more distant in time (i.e., distal factors; $r \approx .20$). The strongest predictor (included only in Ozer et al.) was peritraumatic dissociation. Peritraumatic dissociation refers to unusual experiences during and immediately after the traumatic event, such as a sense that things are not real, the experience of time stretching out, and an altered sense of self. Feeling that one is watching oneself in a movie or play as the event unfolds is a common description of the experience of dissociation. The strength of the relationship between such dissociation and likelihood of developing PTSD was in the moderate-to-large range.

Several important points regarding the predictors of PTSD should be highlighted. First, because largest correlations were about .40, peritraumatic dissociation and other predictors are neither necessary nor sufficient for developing PTSD. Second, the explanation for why peritraumatic dissociation is a predictor requires considering a host of differences in both the people exposed and the nature of the exposure. It may be that the severity of the traumatic event influences the likelihood of peritraumatic dissociation, either through the level of psychophysiological arousal the individual endures during the event or through more complicated relationships involving the effects of the individual's temperament, prior experience, prior psychological functioning, and other genetic or environmental factors that affect his or her capacity to regulate the emotional response. Third, level of social support following the trauma was also a strong predictor, with more social support associated with lower likelihood of later PTSD symptoms. An individual's level of social support likely relates to his or her history and functioning prior to the trauma, factors that this literature has generally not investigated and that meta-analytic approaches cannot easily summarize.

PROBLEMS AND POTENTIAL SOLUTIONS

The main limitation of the research on predictors of PTSD is the heavy reliance on self-report measures and retrospective designs. This natu-

ralistic, retrospective approach makes sense considering the general unpredictability of exposure to trauma and the obvious ethical problems of exposing research participants to extreme stress in experimental or quasi-experimental designs. Prospective studies initiated prior to the occurrence of a major disaster or trauma, however, help address this limitation. For example, recent prospective research has assessed the psychological aftermath of the September 11 terrorist attacks in the United States (Silver, Holman, McIntosh, Poulin, & Gil-Rivas, 2002). Longitudinal research with individuals in high-risk jobs, such as jobs in the military, emergency services, and police force, also provides opportunities for prospective studies of possible predictors of PTSD.

Furthermore, the processes by which identified predictors may shape the development of PTSD remain largely unexamined. Systematic investigation of the ways in which these factors influence responses to trauma at multiple levels (e.g., behavioral, social, biological) could potentially inform interventions to attenuate or prevent PTSD. Future research should emphasize the more proximal mechanisms or processes—in psychological or physiological terms—that account for the relationship between PTSD symptoms and the more distal, static predictors such as prior trauma and family history of psychopathology. Evaluation of theory-based interventions with valid operationalization of critical variables could then provide data with which to evaluate current theory, an important area of study given the ethical prohibitions regarding experimental research in this field. Meta-analytic examination of the PTSD literature was useful in identifying simple, linear relationships between predictors and PTSD symptoms. It is likely, however, that some predictors influence each other in more complex ways; for example, a given predictor may strengthen the effects of another predictor on the development of PTSD (moderation) or may serve as the mechanism through which another predictor increases the likelihood of developing PTSD (mediation). Moreover, the unique meaning of exposure for a single individual may provide the most parsimonious explanation for why a person develops PTSD.

INTRIGUING ISSUES AND QUESTIONS

Definition of Traumatic Event

The definition of what constitutes a traumatic event is central to the diagnosis of PTSD and to all research regarding the disorder. Defining a traumatic event, however, is not simple; indeed, the diagnostic definition has changed over the past decade. Definitional issues raise interesting challenges for PTSD research as they call into question what kinds of experiences are traumatic and for whom. If two people experience the same event (e.g., encountering body parts) but only one reacts with fear, helplessness, or horror, has only one of them experienced a traumatic event?

Because traumatic events typically involve immediate horror and threat to survival (e.g., sexual assault at knifepoint, torture, combat), very high physiological arousal usually accompanies the experience. A broadening of the types of events that some people consider to be traumatic has led to inclusion in the PTSD literature of studies of highly distressing events (e.g., receiving a diagnosis of cancer) that may or may not invoke the same arousal that acute life-threatening situations do. The presence or absence of arousal may well become a key phenomenon that has implications for symptoms of PTSD and whether or not an event is deemed traumatic. If the subjective emotional and physiological response to the event is overlooked, research may not yield consistent findings that would perhaps emerge if arousal were required to identify an event as traumatic.

Ongoing Exposures and the Prototype of PTSD Symptoms

Early theories of trauma response and PTSD were largely based on individuals who lived in generally positive environments and experienced a discrete traumatic event or series of events within a discrete period of time (e.g., sexual assault, disaster, military service), so that the traumatic event or events signified a dramatic disruption of pretrauma life. It is unclear how well this model fits the experience of individuals subjected to pervasive traumatic stress, for example, in the contexts of chronic physical or sexual abuse, deadly civilian conflicts and genocide, or severe community violence in low-income urban areas. The impairments of such individuals, including problems in interpersonal relationships and affect and impulse regulation, may be complicated and difficult to treat (Herman, 1992). The self-perceptions of people who have experienced ongoing trauma seem to be dramatically worse than those of individuals who have experienced discrete traumatic events in the context of otherwise normal development. Some researchers have suggested that a separate term, such as "complex PTSD" or "disorders of extreme stress—not otherwise specified," should be used in place of PTSD to better describe this disorder. Much prior research did not examine whether the predictors of disorder differ depending on whether trauma is experienced as a discrete event or as an ongoing condition of life. Future research that investigates this distinction may find clearer patterns of predictive relationships than have been uncovered so far.

Prevention of PTSD

What are the implications of the research on predictors of PTSD for the prevention of the disorder? Secondary-prevention efforts that seek to reduce the likelihood of PTSD among individuals who have recently been exposed to traumatic stress could utilize these findings by developing early-intervention models that target processes associated with PTSD risk in the meta-analyses reviewed here (e.g., social support, peritraumatic dissociation if the affected individuals could be seen immediately following the event). Strategies for the primary prevention of PTSD would entail reducing the incidence of traumatic events. The most frequent types of traumatic events studied in the research literature have been combat exposure, interpersonal assaults, accidents, and disasters. Although some traumatic stressors, such as earthquakes, are beyond human control, action at the individual and community levels could clearly reduce the risk of exposure to many forms of traumatic stress and also shape the impact of even uncontrollable traumatic stressors on populations. Indeed, such efforts form the backbone of diverse disciplines and public-health policy efforts in areas including building and transportation safety, community violence prevention, domestic violence prevention, and international diplomacy.

There have been numerous investigations of the prevalence of PTSD in diverse communities that have experienced armed civil conflict or war, political repression, or other chronic violence. In such settings of collective trauma, it is particularly critical to look beyond the individual when considering both the effects of trauma and strategies for intervention and prevention. For example, severe political repression affects not just individuals but also the social

institutions and norms of a nation or community (Martin-Baro, 1994). Virtually all interventions for PTSD focus on the individual with symptoms and utilize medication or psychotherapy. Although these interventions may help alleviate individual symptoms, they are obviously inadequate for addressing the harm to social institutions or promoting long-term healing and mental health if the sources of persistent trauma are not addressed. When PTSD is a consequence of collective social and political conditions, primary prevention of this disorder involves social and political changes in the community or nation, as does repair of the social fabric. Thus, perhaps more than any other psychological disorder, PTSD forces consideration of advocacy and political action as primary (universal) prevention tools.

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Schizophrenia: A Neurodevelopmental Perspective

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Abstract

Diverse lines of research suggest that schizophrenia is a genetically influenced neurodevelopmental disorder. Family, twin, and adoption studies suggest that most cases of schizophrenia involve a genetic diathesis that is necessary but not sufficient for development of the disorder. Histological, neuroimaging, and neuropsychological findings converge in providing evidence for medial-temporal and frontal lobe dysfunction that likely predates the onset of psychosis. Behavioral phenomenology and neurobiology suggest that dopamine plays a crucial moderating role between these structural abnormalities and functional impairment. Recently, investigators have used animal models and clinical syndromes to integrate these findings into neurodevelopmental models of schizophrenia that hold great potential for yielding etiological insight.

Keywords

schizophrenia; neurodevelopment; etiology; predisposition

The past decade has seen a proliferation of research findings in the field of schizophrenia, with provocative developments in molecular genetics, neurobiology, neuroimaging, neuropsychology, and studies of high-risk individuals. Although the etiology of schizophrenia remains enigmatic, scientists are gaining ground in developing plausible models of vulnerability to this disorder. In the past, most research findings have provided insights into selected aspects of the disorder without yielding a comprehensive theory that has received broad-based approval. Recently emerging neurodevelopmental models of schizophrenia, however, are capable of accommodating diverse findings and are receiving widespread support among schizophrenia investigators.

Neurodevelopmental models propose that vulnerability to schizophrenia results from a disruption in forebrain development during the perinatal period. A brain lesion that occurs early in development is hypothesized to lie dormant until normal brain maturational events trigger the appearance of traditional diagnostic signs, typically in adolescence or early adulthood (Weinberger, 1987). Such models are supported by reports of increased intrauterine and perinatal complications among individuals with schizophrenia, as well as by demonstrations that neurological, neuropsychological, and physical abnormalities predate the onset of psychosis. Although this evidence is far from conclusive, neurodevelopmental models hold immense promise as a heuristic for bridging research in multiple domains and posing questions central to discovering the etiology of this disorder.

GENETIC AND ENVIRONMENTAL VULNERABILITY

Well-replicated findings from family, twin, and adoption studies indicate that there is a substantial genetic component to the predisposition for schizophrenia. The likeli-

hood that this genetic predisposition involves multiple genes and the possibility that different genetic variants underlie the risk for schizophrenia have made the search for genes via standard molecular techniques daunting. Although research findings have identified several chromosomal sites where there may be genes that confer susceptibility to schizophrenia (i.e., genetic linkage), a failure to replicate these findings has become the norm rather than the exception. A recent study, which examined the entire genome of individuals in families with high rates of schizophrenia, provided evidence for schizophrenia susceptibility on chromosome 1 (Brzustowicz, Hodgkinson, Chow, Honer, & Bassett, 2000). This finding is promising in that the evidence for genetic linkage was unusually strong, a factor that should facilitate the search for a specific gene on this chromosome in these families.

It is reasonable to propose that alteration of gene expression (i.e., production of proteins coded for by genes), during critical phases of early development, contributes to neurodevelopmental abnormalities seen in schizophrenia. Brain development is a delicate process that requires a precise cascade of events orchestrated by the timing and specificity of gene expression. However, until one or more schizophrenia-susceptibility genes have been identified, the link between genetic variations and neurodevelopmental abnormalities remains largely theoretical. For example, neurodevelopmental disturbances in schizophrenia may result from the improper function of proteins that regulate the movement of neurons to their final destination in the brain (neuronal migration) and the formation of neural connections (synaptogenesis).

If schizophrenia were entirely due to heredity, all identical twins with schizophrenia would have co-twins who also have the disorder because identical twins share all of their genes. In fact, the co-twins of affected identical twins develop schizophrenia only about half the time. Thus, environmental factors must also influence schizophrenia's development. From a neurodevelopmental perspective, events occurring early in life are of greatest interest as potential environmental risk factors. A higher rate of obstetric complications has been found for schizophrenia patients relative to normal comparison subjects, psychiatric comparison subjects, and well siblings. A recent report suggests that the risk for schizophrenia is correlated with the number of hypoxia-associated obstetric complications (i.e., complications that can result in oxygen deprivation) an individual may have experienced (Cannon, Rosso, Bearden, Sanchez, & Hadley, 1999). The risk for schizophrenia appears to be conferred from an interaction between genetic predisposition and obstetric complications, rather than obstetric complications alone. In addition, *in utero* viral exposure has been studied as an environmental risk factor for schizophrenia because of the higher number of winter births than births in other seasons among schizophrenia patients and the increased frequency of viral epidemics in the fall. For example, an increased rate of schizophrenia was demonstrated among individuals who were exposed during their second trimester to an influenza epidemic in Helsinki in 1957 (Mednick, Machon, Huttunen, & Bonett, 1988).

NEUROLOGICAL ABNORMALITIES

The longest-held finding in support of the neurodevelopmental model is the increased size of fluid-filled spaces (lateral ventricles) in the brain that is present in first-episode schizophrenia patients and appears to remain static over time. It appears, then, that brain abnormalities are not just an index of the disorder's progression, but more likely constitute a preexisting vulnerability to the disorder. Post mortem histological studies have produced convergent evidence for neurological anomalies at the cellular level. Cellular abnormalities in the brain, such as increased neuronal spacing and altered arrangement of neuronal layers in temporal and frontal lobes areas,[2] have suggested that the predisposition for schizophrenia may involve disruption in neuronal migration. Furthermore, histological studies have failed to find signs of gliosis (a neuronal indicator of injury to a mature brain or of a neuropathological process), again suggesting the cellular deviations occurred early in life.

Neuroimaging studies have demonstrated structural and metabolic abnormalities in the medial-temporal lobe and frontal lobe of schizophrenia patients. For example, reduced frontal cerebral blood flow (hypofrontality) during tasks that require frontal activation has been observed, with the degree of frontal blood flow correlating with task performance. Although a review of recent neuroimaging findings in schizophrenia is beyond the scope of this article, there have been increasing efforts to parse out specific areas within the medial-temporal and frontal lobes that may be compromised in schizophrenia. Temporal lobe dysfunction likely contributes to positive symptoms, consisting of delusions and hallucinations, and frontal lobe dysfunction likely contributes to negative symptoms, such as impoverished thought, lack of goal-directed activities, and social withdrawal.

The performance of schizophrenia patients on neuropsychologi-

cal tasks has been used to elucidate cognitive deficits that may be secondary to brain abnormalities in these patients, as well as to develop hypotheses about the location of their neuropathology. Schizophrenia patients have been found to be impaired on a range of tasks, including ones purported to measure abstraction, sustained attention, language, and memory. Recently, Bilder et al. (2000) evaluated the performance of first-episode schizophrenia patients using a comprehensive neuropsychological test battery. They reported a large generalized deficit in schizophrenia patients with additional specific deficits in memory and executive functions. These results are not open to previous criticisms that cognitive deficits in schizophrenia merely reflect factors associated with chronic mental illness (e.g., long-term treatment) or depict global impairment. The findings are consistent with histological and neuroimaging findings that implicate temporal and frontal lobe involvement in schizophrenia.

Although it is now recognized that multiple neurotransmitters likely contribute to the etiology of schizophrenia, dopamine continues to be the primary neurotransmitter of interest.[3] The dopamine hypothesis, which originally asserted that schizophrenia results from a diffuse excess of dopamine in the brain, has been revised to suggest a dysregulation of dopamine resulting in an excess of dopamine in temporal areas and a depletion of dopamine in frontal areas. Further, it has now been proposed that the alteration in dopamine neurotransmission may not result from a primary deficit in dopamine neurons or receptors, but rather may result from abnormalities in the regulation of dopamine by limbic (medial-temporal lobe structures responsible for motivated and emotional behaviors) and frontal regions (More, West, & Grace, 1999). These are the same brain areas implicated by histological, neuroimaging, and neuropsychological studies. The dysregulation of dopamine neurotransmission that appears to occur in schizophrenia corresponds with behavioral and cognitive processes that are altered in this disorder. For example, within the frontal lobe, dopamine appears to specifically mediate aspects of working memory and motor planning that are impaired in schizophrenia (Goldman-Rakic, 1996). Dopamine and its interaction with other neurotransmitters, such as glutamate and gamma amino butyric acid (GABA), continue to be central to etiological models of schizophrenia.

Animal models[4] of schizophrenia hold promise for testing etiological theories, including neurodevelopmental models. Administration of neurotoxins in developing animals has been used to create disruptions in prenatal neuronal migration, perinatal oxygen deprivation has been used to imitate hypoxia associated with obstetric complications, and neonatal lesions to the hippocampus have been used to re-create structural brain abnormalities. To date, animal models have been able to reproduce a surprisingly broad range of neurobiological, behavioral, and cognitive aspects of schizophrenia. For example, some models have reproduced schizophrenia-like post mortem histological changes, impairment on working memory tasks, and withdrawn social behavior. In addition, animal models have demonstrated that some deficits are specific to neonatal rather than adult lesions, some symptoms show delayed emergence in adulthood, and some functions are returned to normal with the administration of drugs used to treat schizophrenia (neuroleptics). Behavioral outcomes can also vary with the genetic strain of an animal, suggesting an interaction between genes and environment. Although animal models integrate findings across research areas well, they have obvious limitations, including the fact that animal behaviors may be insufficient proxies for certain complex human behaviors.

PROSPECTIVE AND HIGH-RISK STUDIES

Neurodevelopmental models implicitly predict that signs of disorder predate the onset of florid psychosis. Indeed, research has demonstrated that individuals who later develop schizophrenia exhibit motor, cognitive, and behavioral abnormalities during childhood. In an innovative archival study, Walker and Lewine (1990) showed that preschizophrenic children could be reliably differentiated from their well siblings in home videos taken during early childhood, primarily on the basis of abnormal movements and reduced facial expression. Jones, Rodgers, Murray, and Marmot (1994) studied a British cohort of 4,746 children born in 1946, of which 30 later developed schizophrenia. The preschizophrenic individuals were more likely than control subjects to have exhibited delayed early motor development; obtained low educational test scores at ages 8, 11, and 15; preferred solitary play at ages 4 and 6; and been rated by teachers as anxious in social situations at age 15.

Researchers have also investigated abnormalities in the unaffected first-degree relatives of schizophrenia patients. These individuals are at genetic risk because they share on average half of their genes with schizophrenia patients. Healthy relatives have been observed to demonstrate both behavioral and neurobiological impairments that are similar to those seen

in affected patients. For nearly a century, higher rates of schizophrenia-related disorders, such as schizotypal personality disorder,[5] have been seen in these relatives compared with the general population. The most consistent finding in relatives has been eye movement dysfunction, a finding consistent with frontal involvement in the genetic diathesis for schizophrenia. The impaired performance of relatives on certain neuropsychological measures, such as working memory tasks, provides further convergent evidence for frontal lobe dysfunction. Relatives who are deviant on more than one of these measures may be at the greatest risk for schizophrenia and may be most informative when included in genetic studies of this disorder. Associations among schizophrenia-related disorders, cognitive task performance, and the quality of eye movements in relatives of schizophrenia patients are being investigated (for discussion, see Iacono & Grove, 1993).

VELOCARDIOFACIAL SYNDROME (VCFS) AS AN INTEGRATIVE EXAMPLE

VCFS is a congenital syndrome that affects multiple body systems and is associated with a small deletion of genetic material in a specific area of chromosome 22. The symptom profile of individuals with VCFS is variable but commonly includes facial malformations, oral palatal anomalies, nasal voice, and cardiac abnormalities. Various studies have demonstrated that the rate of schizophrenia among individuals with VCFS is approximately 25 times the rate found in the population overall (i.e., 1%), leading investigators to suggest that VCFS is a genetic subtype of schizophrenia (Bassett et al., 1998). The inverse relationship also holds, with multiple studies demonstrating that the rate of this deletion on chromosome 22 in schizophrenia patients is approximately 80 times the general-population rate of 1 in 4,000. VCFS and preschizophrenic individuals show strikingly similar developmental characteristics. Specifically, children with VCFS exhibit delayed motor development, below-average IQ, a tendency toward concrete thinking, bland affect, and lowered levels of social interaction.

Research has begun to suggest potentially shared pathophysiology for VCFS and schizophrenia that may stem from the deletion on chromosome 22. One theory proposes that both VCFS and schizophrenia are neurodevelopmental disorders that affect midline body structures, an idea consistent with the physical abnormalities seen in VCFS. It may be that the pathology also includes migration of cells destined to be midline brain structures, including medial-temporal lobe structures. VCFS and schizophrenia have been associated with similar neuropathology (e.g., enlarged ventricles and an underdeveloped cerebellum), as revealed by magnetic resonance imaging; these structural changes may play a role in predisposing individuals to psychosis (Vataja & Elomaa, 1998). Another potential mechanism stems from the observation that the chromosomal area deleted in VCFS is close to the catechol-O-methyl transferase (COMT) gene (Dunham, Collins, Wadey, & Scambler, 1992). COMT is an enzyme that metabolizes certain neurotransmitters, including dopamine. It has been proposed that a predisposition to psychosis could arise through either a decrease in the metabolism of these neurotransmitters in the brain or an increase in exposure to them during neurodevelopment. Although there are limitations to the notion that VCFS is a schizophrenia subtype, the case of VCFS illustrates how research can be integrated to handle multiple aspects of schizophrenia, including genetic predisposition to illness, presence of signs of disorder that predate psychosis, neurochemical deviations, and developmental brain abnormalities.

FUTURE DIRECTIONS

As much as schizophrenia research is yielding provocative findings, there continue to be important unanswered questions regarding etiology. To date, investigators have not been able to reliably identify susceptibility genes for schizophrenia, precluding mapping the pathway from genetic vulnerability to brain abnormalities. A better understanding of the dormancy period between early brain lesions and adult onset of the disorder is needed. Animal models have failed to support the idea that hormonal changes in puberty may trigger the appearance of symptoms, and theories suggesting that the onset of diagnostic symptoms coincides with ongoing frontal lobe development need to be more adequately investigated. The identification of environmental stressors that contribute substantially to risk for schizophrenia is required. Perinatal complications, such as obstetric complications and viral exposure *in utero*, are some of the leading risk contenders; however, they are likely insufficient to account for the 50% of identical twins who have schizophrenia but whose co-twins do not. In addition, these environmental events make different theoretical predictions based on their timing, the stage of brain development implicated, and the mechanism of action.

Although the neurodevelopmental model is making great strides in integrating diverse re-

search findings, it may be but one of several useful models that ultimately characterize schizophrenia's multiple etiologies. The lack of cohesion of some of the research evidence may be due to schizophrenia resulting from different etiologies in different individuals. Such etiological heterogeneity confounds research into the cause (or causes) of schizophrenia, as study samples likely include individuals for whom the underlying cause of the disorder is not the same. One way to obtain samples with greater etiological homogeneity would be to supplement traditional diagnostic systems with measurement of traits that are likely more direct manifestations of the biological predisposition. For example, an investigator could select study samples of individuals who not only meet current diagnostic criteria for schizophrenia, but also demonstrate signs of neurodevelopmental origin for this disorder, such as those reviewed in this article. Research using a selection procedure such as this, one that is theoretically driven and also supported by recent research findings, likely holds the greatest promise for yielding etiological insight.

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Notes

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2. The temporal lobe is located laterally in the brain, near the temples. A primary ability supported by medial-temporal lobe structures (e.g., the hippocampus) is memory. In addition, individuals with damage to the temporal lobe may experience hallucinations. The frontal lobe is located in the most anterior part of the brain. Primary abilities supported by the frontal lobe include attention, as well as higher-level planning and organizing skills sometimes referred to as executive functions. Individuals with damage to the frontal lobe may demonstrate working memory impairment (i.e., an inability to temporarily store and manipulate information needed to execute a task) and eye movement dysfunction (i.e., an inability to produce certain kinds of eye movements in experimental paradigms).

3. Dopamine is one of many identified neurotransmitters, chemicals that allow for communication between nerve cells (neurons). Neurotransmitters are typically released into the space between neurons (a synapse), where they may exert their effect by binding to specific neuroanatomical sites (receptors) of adjacent neurons. In this manner, neurotransmitters may serve to propagate electrochemical signals throughout the nervous system.

4. Animal models attempt to imitate or re-create some aspect of human functioning in animals in order to study specific processes under greater experimental control. For example, animal models of schizophrenia may create any combination of signs and symptoms of the disorder in order to gain better understanding of its etiology or treatments.

5. Schizotypal personality disorder is characterized by disturbances in interpersonal relationships, distorted thoughts or perceptions, and odd speech or behavior. These symptoms are generally believed to be similar, but subthreshold, to schizophrenia symptoms.

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Will They Do It Again? Predicting Sex-Offense Recidivism

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Abstract

This article reviews the empirical research on the prediction of reoffending among sexual offenders. The major predictors of sexual-offense recidivism are factors related to sexual deviance (e.g., deviant sexual preferences, previous sex crimes) and, to a lesser extent, criminal lifestyle (e.g., antisocial personality disorder, total number of prior offenses). The factors that predict general recidivism among sex offenders are the same as the factors that predict general recidivism among nonsexual criminals (e.g., juvenile delinquency, prior violent offenses). Given that there are special predictors of sexual recidivism, evaluators should consider separately the risk for sexual and nonsexual recidivism.

Keywords

sex offenders; recidivism; prediction

All too often, we hear about sexual offenders committing new crimes. Every sexual crime is worrisome, but we are particularly troubled when it seems that the new offense should have been predicted and could have been prevented. Perhaps the offender had a history of similar offenses or, worse, was currently under community supervision (probation or parole). The fear of recidivism (i.e., repeat offending) among sex offenders has resulted in a number of exceptional policy measures for sex offenders, such as indeterminate sentences, community notification, lifetime supervision, and postsentence detention.

All these policy initiatives assume the ability to accurately distinguish between those individuals who are at risk of committing another sexual offense and those who are not. Unfortunately, the methods most commonly used to assess recidivism risk among sex offenders have low levels of accuracy (only slightly above chance; see Hanson & Bussière, 1998). Recent research, however, has the potential of substantially improving such risk assessments.

One plausible approach to recidivism prediction is to assume that an individual offender will behave like other similar individuals. If, for example, research has found that 30% of "Type X" sex offenders commit another sexual offense, then an individual offender who matches the "Type X" profile would be expected to have a 30% chance of reoffending. Because individuals differ on any number of characteristics, evaluators need to know which characteristics are meaningfully related to recidivism. In other words, which differences make a difference?

HOW MANY SEX OFFENDERS REOFFEND?

Before considering characteristics that increase or decrease recidivism potential, it is useful to consider the expected recidivism rate for the average sex offender—the recidivism base rate. Contrary to common opinion, the observed recidivism rate of sexual offenders is relatively low. A review of 61 recidivism studies involving close to 24,000 sex offenders found that only 13.4% committed a new sexual offense within 4 to 5 years (Hanson & Bussière, 1998). Approximately 12% of sex offenders committed a new nonsexual violent offense (e.g., assault), with rapists violently reoffending more often (22%) than child molesters (10%). When recidivism was defined as any reoffense, then the rates were predictably higher (36% overall).

The observed recidivism rates are underestimates of the actual rates because many sexual offenses are never detected. The extent of the underestimation is the topic of active debate—a debate that is likely to remain active because definitive evidence is, by definition, unavailable. Nevertheless, we know that the observed recidivism rates for sexual offenses can increase to 30 to 40% as the follow-up period extends over 20 years (Hanson, Steffy, & Gauthier, 1993; Prentky, Lee, Knight, & Cerce, 1997). The inclusion of arrests and informal reports of criminal activity will also provide estimates substantially higher than those based solely on official convictions (e.g., Marshall & Barbaree, 1988; Prentky et al., 1997). However, even with long follow-up periods and thorough searches, studies rarely find sex-offense recidivism rates greater than 40%. Any recidivism is troubling, but the available evidence does not support the popular belief that sexual offenders inevitably reoffend. The overall recidivism rate of sex offenders is, on average, less than the rate for nonsexual criminals (Beck & Shipley, 1989).

RECIDIVISM RISK FACTORS

The best way to determine whether a particular characteristic is related to recidivism is to compare the recidivism rates of offenders with that characteristic (e.g., those who are single) and offenders without that characteristic (e.g., those who are married). Recently, Bussière and I reviewed 61 studies that examined 69 potential predictors of sexual-offense recidivism, 38 predictors of nonsexual violent recidivism, and 58 predictors of general (any) recidivism (total sample size of 28,972 sexual offenders). In general, the strongest predictors of sex-offense recidivism were factors related to sexual deviancy. These factors included deviant sexual preferences (according to clinical assessments); early onset of sex offending; and history of prior sex offenses, choosing strangers as victims, choosing males as victims, and committing diverse sexual crimes. The single strongest predictor of sex-offense recidivism was sexual interest in children, assessed phallometrically. Phallometric assessment involves direct monitoring of an individual's penile responses while he is viewing or listening to erotic stimuli (Launay, 1994). Apart from the sexual deviancy factors, the next most important predictors of sexual-offense recidivism were factors related to criminal lifestyle, such as antisocial personality disorder and the total number of prior offenses.

Our review also identified a number of factors that were not related to sexual-offense recidivism. These unrelated factors included being sexually abused as a child, having low self-esteem, denying the sex offense, and lacking empathy with the victim. Many of these characteristics are difficult to assess because there are obvious social consequences to appearing victimized and remorseful; consequently, it is possible that any potential relationships could have been obscured by measurement problems. In addition, verbal statements of sex offenders may have limited predictive value. A finding that supports this interpretation is that offenders' verbal statements concerning motivation for treatment were unrelated to sex-offense recidivism, but those offenders who actually followed through with treatment reoffended less often than offenders who failed to complete treatment.

In general, the predictors of nonsexual recidivism for sexual offenders were the same factors that predict nonsexual recidivism among nonsexual criminals (see Gendreau, Little, & Goggin, 1996). These factors include antisocial personality disorder, young age, juvenile delinquency, history of prior offenses, and minority race. Rapists were more likely than child molesters to recidivate with a nonsexual offense.

COMBINING RISK FACTORS

Although the research literature demonstrates a number of factors reliably related to recidivism risk among sexual offenders, the predictive accuracy of any single factor is modest. No single factor is sufficiently diagnostic that it could be used on its own. Consequently, evaluators wishing to estimate the recidivism risk of sexual offenders need to consider a range of relevant risk factors.

When considering multiple factors, evaluators need to use valid methods for translating the offender's pattern of risk factors into a recidivism prediction. This is not an easy task. For example, consider an evaluator who rates an offender on a set of 20 valid risk factors (young age, never married, prior sex offenses, diverse sex crimes, etc.). If the offender has none of the risk factors, the offender can safely be considered low risk. If the offender has all the risk factors, then the offender can be considered high risk. But what about the typical offender who has only some of the risk factors? How does this particular pattern translate into a risk prediction? Are some risk factors more important than others?

Such concerns have stimulated efforts to produce actuarial risk scales that not only specify the risk factors to consider, but also provide explicit rules for combining individual scores into probability estimates. For many years, actuarial risk scales have been routinely used to predict general criminal recidivism among nonsexual offenders (see Andrews & Bonta, 1998). The scales designed to predict general recidivism have been effective in predicting general recidivism among sexual offenders, but they have been less successful in predicting sexual-offense recidivism. It appears that the prediction of sexual-offense recidivism requires the consideration of special factors (e.g., sexual deviance) that are not included in the risk scales designed for general criminal offenders (e.g., thieves, drug dealers).

Several different research teams—notably, those led by Epperson in Minnesota and Thornton in England—have made significant progress in constructing risk scales specifically for sexual offenders, but many of their findings have yet to be fully cross-validated and are available only as unpublished conference presentations. My own work (with Thornton) suggests that it is possible to achieve moderate accuracy in predicting sexual-offense recidivism using a relatively simple list of demographic and offense-history variables (i.e., never married, any stranger vic-

tims, any male victims, any unrelated victims, age less than 25, any noncontact sex offense, total number of prior sexual offenses, any violent offenses, total number of prior offenses; Hanson & Thornton, 2000). Offenders are assigned points for each risk factor present, and the total number of points is used to classify offenders into relative risk categories. In our validation study, the sex-offense recidivism rate for the highest-risk categories was greater than 50%, whereas the rate for the lowest-risk categories was approximately 10% after 15 years.

STATIC AND DYNAMIC (CHANGEABLE) RISK FACTORS

Almost all the available research has focused on static, unchangeable factors. Static factors are useful for identifying long-term risk potential, but they provide no information concerning when offenders are likely to reoffend or how to intervene in order to reduce the potential for recidivism. My recent research with Harris (Hanson & Harris, 2000) suggests that a number of dynamic factors can usefully contribute to risk assessment of sexual offenders under community supervision. These factors include negative social influences, attitudes tolerant of sexual assault, sexual preoccupations, uncontrolled release environment (e.g., rooming house in a high-crime neighborhood), access to victims, and lack of cooperation with supervision. Further research is required, however, before we know how best to integrate these dynamic factors into applied risk assessments for sex offenders.

One consequence of our limited knowledge of dynamic risk factors is that we have better evidence for identifying offenders who are dangerous than we have for determining when offenders are safe to be released. Not all sex offenders reoffend, and even high-risk offenders can change their ways. Unfortunately, clinicians have achieved only limited accuracy in determining when offenders have benefited from treatment.

EXPLANATIONS FOR SEX OFFENDING

Evaluating change requires an accurate model of the causes of sexual offending. As in other areas of psychology, the major theories can be divided into those that emphasize nature versus those that emphasize nurture. On the nature side are theories that consider deviant sexual preferences to be linked to genetic or hormonal anomalies. No one seriously questions that normal sexual interests are biologically based, and the recent evidence linking homosexuality to the Xq28 region of the sex chromosome (Hamer & Copeland, 1994) has lent support to the belief in a genetic predisposition for pedophilia. Researchers, however, have yet to find any biological markers associated with sex offending.

Although biomedical research may eventually provide insights, most treatment programs for sex offenders are based on some form of social learning theory. Those offenders with the most well practiced patterns of deviant behavior, and the least ability to appropriately manage their impulses, are considered to be at the highest risk for recidivism. The validity of this model is currently being tested through cognitive-behavioral treatment programs that teach offenders relapse-prevention skills in the hope of reducing their long-term recidivism risk.

DIRECTIONS FOR FUTURE RESEARCH

The ongoing research challenge of all these treatment programs is determining when offenders have changed. For sex offenders, unlike individuals with other types of behavioral problems, it is difficult to provide realistic tests of their new coping skills. Depressed patients can monitor their mood, and agoraphobics can try to go out, but clinicians can never provide sex offenders with unsupervised access to vulnerable victims. Knowledge concerning sexual offending would likely advance quickly given a reliable analogue of sex offending. Imagine, for example, creating artificial situations in which individuals at risk would feel tempted to sexually offend. The success of phallometric assessment as a recidivism risk indicator may be due to its resemblance to such an analogue stress test.

One of the ongoing controversies concerns the role of low self-esteem and subjective distress in the recidivism process. Sex offenders are characterized by considerable anxiety, depression, and low self-regard. Moreover, their recidivism risk increases when their mood deteriorates (Hanson & Harris, 2000). What is surprising, however, is that subjective distress is unrelated to long-term recidivism. Those sex offenders who are chronically unhappy commit new offenses at the same rate as other sex offenders. Treatment programs that successfully raise offenders' self-esteem appear to have no impact on recidivism rates (Hanson et al., 1993).

It is possible that low self-esteem (like being sexually abused) is linked to the initiation of sex offending but is unrelated to recidivism. In addition, the level of subjective distress may matter less than attempts to escape distress by

resorting to sexual fantasies or sexual activities (deviant or otherwise). Disentangling the factors that contribute to sexual recidivism will require detailed accounts from cooperative offenders, as well as prospective studies that monitor ongoing changes in the offenders' lives.

In conclusion, it is worth considering Grove and Meehl's (1996) caution about the use of unverified clinical opinion, which they compare to the Inquisition's methods for identifying witches. "All policymakers should know that a practitioner who claims not to need any statistical or experimental studies but relies solely on clinical experience as adequate justification, by that very claim is shown to be a nonscientifically minded person whose professional judgments are not to be trusted" (Grove & Meehl, 1996, p. 320). Not too long ago, the people who evaluate sexual offenders had little choice but to use unverified clinical opinion because the relevant research was unavailable. With recent advances, however, knowledgeable evaluators now have the potential of providing risk assessments worthy of serious consideration.

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Note

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Math Anxiety: Personal, Educational, and Cognitive Consequences

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Abstract

Highly math-anxious individuals are characterized by a strong tendency to avoid math, which ultimately undercuts their math competence and forecloses important career paths. But timed, on-line tests reveal math-anxiety effects on whole-number arithmetic problems (e.g., 46 + 27), whereas achievement tests show no competence differences. Math anxiety disrupts cognitive processing by compromising ongoing activity in working memory. Although the causes of math anxiety are undetermined, some teaching styles are implicated as risk factors. We need research on the origins of math anxiety and on its "signature" in brain activity, to examine both its emotional and its cognitive components.

Keywords

anxiety; mental arithmetic; math competence; working memory; problem solving

My graduate assistant recently told me about a participant he had tested in the lab. She exhibited increasing discomfort and nervousness as the testing session progressed, eventually becoming so distraught that she burst into tears. My assistant remarked that many of our participants show some unease or apprehension during testing—trembling hands, nervous laughter, and so forth. Many ask, defensively, if their performance says anything about their overall intelligence. These occasionally extreme emotional reactions are not triggered by deliberately provocative procedures—there are no personally sensitive questions or intentional manipulations of stress. Instead, we merely ask college adults to solve elementary-school arithmetic problems, such as 46 + 18 = ? and 34 − 19 = ?

The reactions are obvious symptoms of anxiety, in this case math anxiety induced by ordinary arithmetic problems presented in timed tasks. On the one hand, it is almost unbelievable that tests on such fundamental topics can be so upsetting; knowing that 15 − 8 = 7 ought to be as basic as knowing how to spell "cat." On the other hand, U.S. culture abounds with attitudes that foster math anxiety: Math is thought to be inherently difficult (as Barbie dolls used to say, "Math class is hard"), aptitude is considered far more important than effort (Geary, 1994, chap. 7), and being good at math is considered relatively unimportant, or even optional.

In this article, I discuss what has been learned about math anxiety across the past 30 years or so, and suggest some pressing issues to be pursued in this area. An important backdrop for this discussion is the fact that modern society is increasingly data and technology oriented, but the formal educational system seems increasingly unsuccessful at educating students to an adequate level of "numeracy," the mathematical equivalent of literacy (Paulos, 1988).

MATH ANXIETY DEFINED AND MEASURED

Math anxiety is commonly defined as a feeling of tension, apprehension, or fear that interferes with math performance. The first systematic instrument for assessing math anxiety was the Mathematics Anxiety Rating Scale (MARS), published by Richardson and Suinn (1972). In this test, participants rate themselves on the level of anxiety they would feel in various everyday situations, such as trying to refigure a restaurant bill when they think they have been overcharged or taking a math test. My co-workers and I use a shortened version of the test, which yields scores that correlate well with scores obtained using the original test and also has very acceptable test-retest reliability (i.e., an individual who takes the test on different occasions generally receives similar scores). We have also found that for a quick determination, one can merely ask, "On a scale from 1 to 10, how math anxious are you?" Across at least a half-dozen samples, responses to this one question have correlated anywhere from .49 to .85 with scores on the shortened MARS.

There is a rather extensive literature on the personal and educational consequences of math anxiety, summarized thoroughly in Hembree (1990). Perhaps the most pervasive—and unfortunate—tendency is avoidance. Highly math-anxious individuals avoid math. They take fewer elective math courses, both in high school and in college, than people with low math anxiety. And when they take math, they receive lower grades. Highly math-anxious people also espouse negative attitudes toward math, and hold negative self-perceptions

about their math abilities. The correlations between math anxiety and variables such as motivation and self-confidence in math are strongly negative, ranging between −.47 and −.82. It is therefore no surprise that people with math anxiety tend to avoid college majors and career paths that depend heavily on math or quantitative skills, with obvious and unfortunate consequences.

Interestingly, math anxiety is only weakly related to overall intelligence. Moreover, the small correlation of −.17 between math anxiety and intelligence is probably inflated because IQ tests include quantitative items, on which individuals with math anxiety perform more poorly than those without math anxiety. The small correlation (−.06) between math anxiety and verbal aptitude supports this interpretation. However, math anxiety is related to several other important characteristics. As conventional wisdom suggests, it is somewhat higher among women than men. The gender difference is rather small, may be particularly apparent in highly selected groups (e.g., college students), and may be partly attributable to a greater willingness on the part of women to disclose personal attitudes. Nonetheless, when we recruited participants for research on math anxiety, we found fewer men than women at high anxiety levels, but just the reverse at low levels (Ashcraft & Faust, 1994).

Individuals who are high in math anxiety also tend to score high on other anxiety tests. The strongest interrelationship is with test anxiety, a .52 correlation. Despite the overlap among kinds of anxiety, however, the evidence is convincing that math anxiety is a separate phenomenon. For instance, intercorrelations between alternative assessments of math anxiety range from .50 to .70, but intercorrelations of math anxiety with other forms of anxiety range from .30 to .50. In a particularly clear display of the specificity of math anxiety, Faust (1992) found physiological evidence of increasing reactivity (e.g., changes in heart rate) when a highly math-anxious group performed math tasks of increasing difficulty. When the same participants performed an increasingly difficult verbal task, there was hardly any increase in their reactivity (e.g., Ashcraft, 1995, Fig. 6), and participants with low math anxiety showed virtually no increase in either task.

MATH ANXIETY AND MATH COMPETENCE

An obvious but unfortunate consequence of the avoidance tendency is that compared with people who do not have math anxiety, highly math-anxious individuals end up with lower math competence and achievement. They are exposed to less math in school and apparently learn less of what they are exposed to; as a result, they show lower achievement as measured by standardized tests (e.g., Fennema, 1989). The empirical relationship is of moderate strength (a correlation of −.31 for college students), but sufficient to pose a dilemma for empirical work. That is, when highly math-anxious individuals perform poorly on a test, their poor performance could in fact be due to low competence and achievement rather than heightened math anxiety. If the relationship between anxiety and competence holds for all levels of math difficulty, then variations in competence will contaminate any attempt to examine math performance at different levels of math anxiety.

Fortunately, there are ways out of this dilemma. One is to test additional samples of participants on untimed, pencil-and-paper versions of the math problems studied in the lab. For example, we (Faust, Ashcraft, & Fleck, 1996) found no anxiety effects on whole-number arithmetic problems when participants were tested using a pencil-and-paper format. But when participants were tested on-line (i.e., when they were timed as they solved the problems mentally under time pressure in the lab), there were substantial anxiety effects on the same problems.

We have also taken a second approach (see Ashcraft, Kirk, & Hopko, 1998). In brief, we administered a standard math achievement test to individuals with low, medium, or high math anxiety, and replicated the overall result reported by Hembree (1990; i.e., math achievement scores decrease as math anxiety increases). But we then scored the achievement test to take advantage of the line-by-line increases in difficulty. With this scoring method, we found that there were no math-anxiety effects whatsoever on the first half of the test, which measured performance on whole-number arithmetic problems. Anxiety effects were apparent only on the second half of the test, which introduced mixed fractions (e.g., 10 1/4 − 7 2/3), percentages, equations with unknowns, and factoring. For these problems, there was a strong negative relationship between accuracy and math anxiety. Thus, individuals with high levels of math anxiety do not have a global deficit in math competence, and they can perform as well as their peers on whole-number arithmetic problems. Investigations of higher-level arithmetic and math, though, do need to take the competence-anxiety relationship into account.

There is still reason to be somewhat suspicious of this relationship between anxiety and competence, however. Effective treatments for math anxiety (see Hembree, 1990,

Table 8) have resulted in a significant improvement in students' math achievement scores, bringing them nearly to the level shown by students with low math anxiety. Because the treatments did not involve teaching or practicing math, the improvement could not be due to a genuine increase in math competence. We suspect instead that these students' original (i.e., pretreatment) math competence scores were artificially low, depressed by their math anxiety. When the anxiety was relieved, a truer picture of their competence emerged.

COGNITIVE CONSEQUENCES OF MATH ANXIETY

Our original studies were apparently the first to investigate whether math anxiety has a measurable, on-line effect on cognitive processing, that is, whether it actually influences mental processing during problem solving. In our early studies (Ashcraft & Faust, 1994; Faust et al., 1996), we found that math anxiety has only minimal effects on performance with single-digit addition and multiplication problems. One anxiety effect we did find, however, was in a decision-making process sensitive to "number sense" (Dehaene, 1997)—when making true/false judgments, highly math-anxious individuals made more errors as the problems became increasingly implausible (e.g., 9 + 7 = 39), whereas low-anxiety participants made fewer errors on such problems.

Arithmetic problems with larger numbers (e.g., two-column addition or multiplication problems), however, showed two substantial math-anxiety effects. First, participants at high levels of anxiety routinely responded rapidly to these problems, sometimes as rapidly as participants with low anxiety, but only by sacrificing considerable accuracy. This behavior resembles the global avoidance tendency characteristic of highly math-anxious individuals, but at an immediate, local level: By speeding through problems, highly anxious individuals minimized their time and involvement in the lab task, much as they probably did in math class. Such avoidance came at a price, however—a sharp increase in errors.

Second, the results showed that addition problems with carrying were especially difficult for highly math-anxious individuals. In particular, the time disadvantage for carry versus no-carry problems was three times larger for participants with high anxiety (753 ms) than for those with low anxiety (253 ms), even aside from the difference in accuracy between the two groups. Our interpretation was that carrying, or any procedural aspect of arithmetic, might place a heavy demand on working memory, the system for conscious, effortful mental processing. In other words, we proposed that the effects of math anxiety are tied to those cognitive operations that rely on the resources of working memory.

In an investigation of this possibility, Kirk and I (Ashcraft & Kirk, 2001) tested one- and two-column addition problems, half requiring a carry. We embedded this test within a dual-task procedure, asking our participants to do mental math, the primary task, while simultaneously remembering random letters, a secondary task that taxes working memory. Two or six letters were presented before each addition problem, and after participants gave the answer to the problem, they were asked to recall the letters in order. We reasoned that as the secondary task became more difficult (i.e., when more letters had to be held in working memory), performance on the primary task might begin to degrade, in either speed or accuracy. If that happened, we could infer that the primary task indeed depended on working memory, and that the combination of tasks began to exceed the limited capacity of working memory.

When the addition problem involved carrying, errors increased substantially more for participants with high math anxiety than for those with low anxiety (Ashcraft & Kirk, 2001, Experiment 2). Moreover, as we predicted, this was especially the case when the secondary task became more difficult, that is, with a six-letter memory load. On carry problems (e.g., 6 + 9, 27 + 15), highly anxious individuals made 40% errors in the heavy-load condition, compared with only 20% errors for individuals with low anxiety in the high-load condition and 12% errors for both groups in the light-load condition. In the control conditions, with each task performed separately, the comparable error rates were only 16% and 8%. These results could not be attributed to overall differences in working memory. That is, we examined the participants' working memory spans (the amount of information they were able to remember for a brief amount of time) and found no differences between the groups when spans were assessed with a verbal task. But span scores did vary with math anxiety when they were assessed with an arithmetic-based task.

These results are consistent with Eysenck and Calvo's (1992) model of general anxiety effects, called processing efficiency theory. In this theory, general anxiety is hypothesized to disrupt ongoing working memory processes because anxious individuals devote attention to their intrusive thoughts and worries, rather than the task at hand. In the case of math anxiety, such thoughts probably involve preoccupation with one's dislike or fear of math, one's low self-confidence, and the like. Math anxiety lowers

math performance because paying attention to these intrusive thoughts acts like a secondary task, distracting attention from the math task. It follows that cognitive performance is disrupted to the degree that the math task depends on working memory.

In our view, routine arithmetic processes like retrieval of simple facts require little in the way of working memory processing, and therefore show only minimal effects of math anxiety. But problems involving carrying, borrowing, and keeping track in a sequence of operations (e.g., long division) do rely on working memory, and so should show considerable math-anxiety effects. Higher-level math (e.g., algebra) probably relies even more heavily on working memory, so may show a far greater impact of math anxiety; note how difficult it will be, when investigating high-level math topics, to distinguish clearly between the effects of high math anxiety and low math competence.

GAPS IN THE EVIDENCE

Math anxiety is a bona fide anxiety reaction, a phobia (Faust, 1992), with both immediate cognitive and long-term educational implications. Unfortunately, there has been no thorough empirical work on the origins or causes of math anxiety, although there are some strong hints. For instance, Turner et al. (2002) documented the patterns of student avoidance (e.g., not being involved or seeking help) that result from teachers who convey a high demand for correctness but provide little cognitive or motivational support during lessons (e.g., the teacher "typically did not respond to mistakes and misunderstandings with explanations," p. 101; "he often showed annoyance when students gave wrong answers . . . . He held them responsible for their lack of understanding," p. 102). Turner et al. speculated that students with such teachers may feel "vulnerable to public displays of incompetence" (p. 101), a hypothesis consistent with our participants' anecdotal reports that public embarrassment in math class contributed to their math anxiety. Thus, it is entirely plausible, but as yet undocumented, that such classroom methods are risk factors for math anxiety.

Other gaps in the evidence involve the cognitive consequences of math anxiety, including those that interfere with an accurate assessment of math achievement and competence. My co-workers and I have shown that the transient, on-line math-anxiety reaction compromises the activities of working memory, and hence should disrupt performance on any math task that relies on working memory. The mechanisms for this interference are not yet clear, however. It may be that intrusive thoughts and worry per se are not the problem, but instead that math-anxious individuals fail to inhibit their attention to those distractions (Hopko, Ashcraft, Gute, Ruggiero, & Lewis, 1998).

Finally, as research on mathematical cognition turns increasingly toward the methods of cognitive neuroscience, it will be interesting to see what "signature" math anxiety has in brain activity. The neural activity that characterizes math anxiety should bear strong similarities to the activity associated with other negative affective or phobic states. And our work suggests that the effects of math anxiety should also be evident in neural pathways and regions known to reflect working memory activity.

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Do Negative Cognitive Styles Confer Vulnerability to Depression?

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Abstract

According to the cognitive-vulnerability hypothesis of depression, negative cognitive styles confer vulnerability to depression when people confront negative life events. In this article, we present evidence that negative cognitive styles do indeed confer vulnerability to clinically significant depressive disorders and suicidality and discuss possible developmental antecedents of cognitive vulnerability to depression. We consider the issue of stability versus change in cognitive vulnerability to depression and discuss the broader implications of the cognitive-vulnerability findings for mental and physical health.

Keywords

depression; vulnerability; negative cognitive styles

Consider two women who are fired from their jobs at the same firm. One may become seriously depressed, although the other suffers only mild discouragement. Why is it that some people are vulnerable to depression whereas others never seem to become depressed? According to biological theories of depression, abnormal genetic or biochemical processes predispose some individuals to depression. Alternatively, the cognitive perspective suggests that the way people typically interpret or understand events in their lives (i.e., their cognitive styles) has an important effect on their vulnerability to depression.

NEGATIVE COGNITIVE STYLES AND VULNERABILITY TO DEPRESSION

Unlike biological models, cognitive theories of depression attempt to explain individual differences in

responses to stressful life events in terms of a set of maladaptive thinking patterns. For example, according to the hopelessness theory (Abramson, Metalsky, & Alloy, 1989), people who characteristically infer that negative life events are produced by stable (persisting over time) and global (widespread) causes ("It will last forever and affect everything I do"), catastrophize about the consequences of negative life events, and believe that the occurrence of a negative event means that they are flawed or worthless possess maladaptive cognitive styles. Compared with people who do not exhibit these negative cognitive styles, such people are hypothesized to be more likely to develop episodes of depression—particularly a subtype known as hopelessness depression—when they experience stressful events. Similarly, in Beck's (1987) theory, individuals who hold certain dysfunctional attitudes—that their worth depends on being perfect ("If I fail partly, it is as bad as being a complete failure") or on others' approval ("I am nothing if a person I love doesn't love me")—are hypothesized to develop depression, particularly a subtype of reactive depression (a subtype triggered by stressors), when they encounter negative events that impinge on their cognitive vulnerability.

Do negative cognitive styles, in fact, increase people's vulnerability to depression? Recent prospective studies have obtained considerable support for this cognitive-vulnerability hypothesis (Alloy et al., 1999). One such study is our own Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project (Alloy et al., 1999). At the outset of this study, nondepressed college freshmen, with no other mental disorders, were identified as being at high risk (HR) or low risk (LR) for depression based on the presence versus absence of negative cognitive styles. Follow-up assessments of these freshmen were conducted every 6 weeks for 2.5 years and then every 4 months for an additional 3 years; the study included self-report and structured interview assessments of stressful life events, cognitions, and psychopathology.

More than half of the CVD Project sample had no prior history of clinical depression. Among these participants, the HR freshmen were more likely than the LR freshmen to develop a first onset of major depressive disorder (17% vs. 1%), minor depressive disorder (39% vs. 6%), and hopelessness depression (41% vs. 5%) during the first 2.5 years of follow-up, and these differences between groups remained even in an analysis controlling for whether the students had depressive symptoms when they were initially selected for the study. What about those participants who, though nondepressed at the outset of the study, did have a prior history of clinical depression? This subsample allows a test of whether the cognitive-vulnerability hypothesis holds for recurrences of depression, a question that is important given that depression is often a recurrent disorder (Judd, 1997). Among participants with past depression, HR freshmen were more likely than LR freshmen to develop recurrences of major (27% vs. 6%), minor (50% vs. 26.5%), and hopelessness (52% vs. 22%) depression, and these differences also remained in an analysis controlling for depressive symptoms at the start of the study. Thus, negative cognitive styles provided risk for both recurrences and first onsets of clinically significant depression, suggesting that similar processes may, at least in part, underlie the first and subsequent episodes of depression.

Among the entire CVD Project sample, HR participants were also more likely than LR participants (28% vs. 12.6%) to develop suicidality (a constellation of behaviors ranging from suicidal thinking to actual suicide attempts) during the follow-up, even when prior history of suicidality was controlled statistically (Abramson et al., 1998). Moreover, the association between cognitive vulnerability and the development of suicidality was completely mediated by hopelessness. That is, only those participants who became hopeless about their futures developed suicidality during the follow-up period.

According to the cognitive theories of depression, people with negative cognitive styles are vulnerable to depression in part because they perceive and recall information about stressful events that has negative implications for themselves. Thus, we (Alloy, Abramson, Murray, Whitehouse, & Hogan, 1997) examined whether our nondepressed HR participants did, in fact, process information about themselves more negatively than LR participants. A task battery administered at the outset of the CVD Project included judgments of whether positive and negative adjectives were self-descriptive, response times for making these judgments, predictions of future behavior, and memory for the adjectives. The task battery yielded results consistent with the prediction: Relative to LR participants, HR participants showed greater endorsement, faster processing, and better recall of negative depression-relevant adjectives involving themes of incompetence, worthlessness, and low motivation. HR participants also showed less endorsement, slower processing, and worse recall of positive depression-relevant adjectives (e.g., "successful," "lovable") than LR participants.

Similar negative biases in information processing about the self have been obtained among nondepressed individuals who have recovered from a past depression when their cognitive vulnerability

is activated by a negative mood state (Ingram, Miranda, & Segal, 1998). Our findings (Alloy et al., 1997) are significant because they indicate that negatively biased information processing previously shown to be characteristic of depressed individuals (Ingram et al., 1998) also occurs among cognitively vulnerable nondepressed individuals. Moreover, such negatively biased information processing also predicted onsets of depressive episodes during the first 2.5 years of the CVD Project, alone and in combination with cognitive HR status (Alloy et al., 1999).

The CVD Project results are important because they provide the first demonstration that negative thinking patterns and information processing—or for that matter, any psychological factor—confer vulnerability to full-blown, clinically significant depressive episodes. This is noteworthy because a criticism of the cognitive theories of depression is that they apply only to mild depression. In the case of the participants with no prior history of depression, these findings provide especially strong support for the cognitive-vulnerability hypothesis because they are based on a truly prospective test, uncontaminated by prior history of depression.

However, although the CVD Project demonstrated that negative cognitive styles predict depression prospectively, it did not establish a causal link between cognitive style and depression. Indeed, demonstrating such causality is a key issue for the cognitive theories of depression. Such a demonstration would require showing that manipulations of cognitive vulnerability lead to corresponding changes in the likelihood of depression onset. In a study consistent with a potential causal role for cognitive vulnerability to depression, DeRubeis and Hollon (1995) reported that decreases in depressed patients' negative cognitive styles following cognitive therapy for depression, a treatment designed to ameliorate such styles, predicted corresponding reductions in relapse of depression. Specifically, depressed patients successfully treated with cognitive therapy were less likely than depressed patients treated with antidepressant medication to suffer relapses of depression, and the reduced relapse rate was mediated by the cognitive therapy's effect on decreasing patients' stable and global styles for inferring causes of negative events.

Similarly, Gillham, Reivich, Jaycox, and Seligman (1995) provided schoolchildren with a 12-week cognitive therapy–based preventive intervention that was designed to teach the children to adopt more adaptive beliefs about themselves and to replace negative explanations for their successes and failures with more optimistic ones. At 1-year follow-up, only about 7% of the children in the prevention group reported high levels of depressive symptoms, compared with nearly 30% of the control group. Inasmuch as disagreement exists about whether cognitive therapy works by remediating negative cognitive styles or by providing compensatory skills for overriding the effects of such styles (DeRubeis & Hollon, 1995), future studies must find a way to directly manipulate cognitive styles in order to more clearly test their causal role for depression onset.

DEVELOPMENTAL ORIGINS OF COGNITIVE VULNERABILITY TO DEPRESSION

If negative cognitive styles do confer vulnerability to depression, then it is important to understand how these styles develop. In the CVD Project, we also studied HR and LR participants' parents, as well as the participants' early developmental experiences (Alloy et al., 1999). Our preliminary findings suggest several potential antecedents of negative cognitive styles. Mothers of HR participants were more likely to have a history of depression and had more episodes of depression in their lifetime than mothers of LR participants. This association could have been due to shared genetic risk for depression or to the participants' learning negative cognitive styles from their parents. Children may directly model their parents' cognitive styles or be taught through parental feedback to make negative inferences about stressful events in their lives. Both the modeling and the feedback hypotheses are consistent with the finding that parents of HR participants had more dysfunctional attitudes and provided more negative feedback about causes and consequences of events in their child's life than parents of LR participants.

A history of maltreatment, particularly emotional abuse, may also contribute to cognitive vulnerability because the abuser by definition supplies negative cognitions to the victim (e.g., "Of course you didn't get invited to the prom—you're ugly"; Rose & Abramson, 1998). Self-reports of participants in the CVD Project are consistent with this formulation: HR participants were more likely to report a history of emotional (but not physical or sexual) abuse than LR participants. Moreover, a history of childhood emotional abuse predicted onsets of depressive episodes in CVD Project participants during follow-up, a relationship that was mediated by their negative cognitive styles (Alloy et al., 1999). These correlational findings cannot establish that the associations between parental depression, cognitive styles, feedback, abuse, and children's cognitive risk for depres-

sion are causal; however, they encourage future prospective tests of the role of developmental history in the origins of cognitive risk for depression.

STABILITY OF COGNITIVE VULNERABILITY TO DEPRESSION

An unresolved issue is whether a vulnerability factor for depression must exhibit stability, never changing over time (e.g., Just, Abramson, & Alloy, in press; Persons & Miranda, 1992). This issue has both empirical and practical significance. If cognitive vulnerability is an immutable trait, it could be measured at any time and would be expected to predict a constant level of risk for depression; however, it would be difficult to remediate. Alternatively, if cognitive vulnerability can change and the conditions and mechanisms promoting such change are discovered, it may be possible to devise effective therapeutic and preventive interventions for depression. Moreover, one could predict when an individual is likely to be relatively immune from depression and when he or she is at greatest risk—the best time to intervene.

An example from medicine illustrates that a vulnerability factor for a disorder need not show traitlike stability and, instead, may show transient or long-term fluctuations (Just et al., in press). A person's vulnerability to a host of diseases, ranging from influenza to cancer, is in part a function of the integrity of the immune system. Yet the integrity of the immune system can change over time. For example, acute stress can produce relatively transient changes in immune functioning, and chronic stress can produce longer–term changes. The critical point is that medical researchers conceptualize compromised immune function as an important vulnerability factor for many diseases, yet no medical researcher would suggest that immune functioning necessarily should be stable. The hopelessness theory allows for the possibility that the negative cognitive styles hypothesized to confer vulnerability to depression do not possess traitlike stability and, in fact, may change over time as a function of intervening life experiences, the occurrence of depressive episodes themselves, and therapeutic interventions. All that is required by the hopelessness theory is that at any given moment, a person's cognitive styles confer the predicted amount of vulnerability to depression.

Recent evidence suggests that people's reported cognitive styles do change as a function of their depression status (Just et al., in press), with styles more negative when people are in a depressed episode than when they have recovered from the episode. However, most of these studies involved patients who were treated for their depression, and the treatment may have ameliorated, deactivated, or otherwise reduced the likelihood of reporting negative cognitive styles. In contrast, studies of people who have not been treated have generally found stability of cognitive styles over time, even as individuals developed and recovered from depression (Just et al., in press). For example, in the CVD Project, participants' cognitive styles remained stable over the first 2.5 years of follow-up, regardless of intervening depressive episodes (Alloy et al., 1999). Future research must identify the conditions and mechanisms that promote change in cognitive styles once they have developed and address whether change in cognitive styles does, in fact, lead to corresponding change in the likelihood of depression.

COGNITIVE VULNERABILITY IN CONTEXT

Work on cognitive vulnerability to depression has shown that negative cognitive styles and negatively biased self-referent information processing confer increased risk for first onsets and recurrences of clinically significant depressive disorders. This work not only has provided the first demonstration of a psychological vulnerability to depression, but also suggests that purely biological approaches to understanding depression are likely to fall short. Indeed, our recent research indicates that even bipolar spectrum mood disorders (manic depression), which have traditionally been viewed as almost entirely genetic in origin and biologically mediated, may also be influenced by cognitive styles for interpreting life events. Both manic and depressive symptoms among individuals with manic-depressive disorder were predicted prospectively by negative cognitive styles and information processing in combination with the occurrence of stressful life events (Reilly-Harrington, Alloy, Fresco, & Whitehouse, in press).

More broadly, the work on cognitive vulnerability to depression suggests that the content of one's thinking and, in particular, one's appraisals of life experiences may profoundly affect one's health. The notion that mental contents influence physical health has been highly controversial. The present findings add to the growing body of research indicating that pessimistic thinking predicts, and possibly contributes to, poor health (and similarly that optimistic thinking predicts good health) through emotional (depression), behavioral (health habits), interpersonal (social support), and physiological

(immune system functioning) routes (Peterson, Maier, & Seligman, 1993). Perhaps, in broadest context, the findings on cognitive vulnerability to depression teach us all that rather than being passive victims of life's insults, through our cognitive appraisals we can profoundly influence our mental and physical reactions to these experiences.

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