

Sex Differences in Unipolar Depression: Evidence and Theory

Susan Nolen-Hoeksema
University of Pennsylvania

A large body of evidence indicates that women are more likely than men to show unipolar depression. Five classes of explanations for these sex differences are examined and the evidence for each class is reviewed. Not one of these explanations adequately accounts for the magnitude of the sex differences in depression. Finally, a response set explanation for the sex differences in depression is proposed. According to this explanation, men are more likely to engage in distracting behaviors that dampen their mood when depressed, but women are more likely to amplify their moods by ruminating about their depressed states and the possible causes of these states. Regardless of the initial source of a depressive episode (i.e., biological or psychological) men's more active responses to their negative moods may be more adaptive on average than women's less active, more ruminative responses.

The epidemiology of a disorder can provide important clues to its etiology. When a disorder only strikes persons from one geographical region, one social class, or one gender, we can ask what characteristics of the vulnerable group might be making its members vulnerable.

A frequent finding in epidemiological studies of mental disorders is that women are more prone to unipolar affective disorders than are men (Boyd & Weissman, 1981; Weissman & Klerman, 1977). A number of different explanations have been proposed to account for women's greater vulnerability to depression. Previous reviews of these explanations (e.g., Weissman & Klerman, 1977) have been quite brief and uncritical.

In this article, the evidence for sex differences in unipolar depression first is summarized, then the most prominent explanations proposed for these sex differences are discussed in detail. These explanations include those attributing the differences to the response biases of subjects, as well as biological, psychoanalytic, sex role, and learned helplessness explanations.

Although most of the proposed explanations for sex differences in depression have received some empirical support, not one of them has been definitively supported and not one as yet accounts for the magnitude of sex differences in depression. In the final section of this article it is suggested that differences in the ways that men and women respond to their own depressive episodes, whatever the origin of these episodes, may be an important source of the sex differences observed in depression.

This work was supported by a National Science Foundation Graduate Fellowship to Susan Nolen-Hoeksema, U.S. Public Health Service Grant MH-19604 to Martin E.P. Seligman, and U.S. Public Health Service Grant MH-40142 to Martin E.P. Seligman, Joan S. Girgus, and Susan Nolen-Hoeksema.

The author thanks Martin E.P. Seligman, Jonathon Baron, Christopher Peterson, and three anonymous reviewers for their very helpful comments on earlier drafts of this article.

Correspondence concerning this article should be addressed to Susan Nolen-Hoeksema, who is now at the Department of Psychology, Stanford University, Jordan Hall, Building 420, Stanford, California 94305.

Background on the Affective Disorders

According to the third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III)* (American Psychiatric Association, 1980), the common symptoms of depression include loss of motivation, sadness, anhedonia, low self-esteem, somatic complaints, and difficulty in concentrating. The opposite of depression is mania. Manic symptoms include greatly increased energy, racing thoughts, pressured speech, wild and extravagant behaviors, and grandiosity. Persons who suffer manic episodes typically also suffer episodes of depression, whereas the majority of persons who suffer depressive episodes never experience mania. Thus in *DSM-III*, the affective disorders are broken down into bipolar affective disorder, which is characterized by alternating episodes of mania and depression, and unipolar depression.

To date, almost all of the discussion of sex differences in depression has been concerned with sex differences in unipolar depression. It has been generally assumed that there are no sex differences in bipolar disorder (e.g., Boyd & Weissman, 1981; Weissman & Klerman, 1977). Yet in a review of the literature on bipolar disorder, Clayton (1981) showed that women predominate among persons given the diagnosis of bipolar disorder, as well as among those with unipolar depression. The only explanation that has been offered for sex differences in bipolar disorder is the suggestion of Winokur and others (see Gershon & Bunney, 1976; Winokur & Tanna, 1969) that both bipolar and unipolar affective disorder are associated with genetic abnormalities linked to the female chromosomes. (This explanation is reviewed in the section on *Biological Explanations*.) Because each of the other explanations of sex differences in depression reviewed here refers only to sex differences in unipolar depression, the data review focuses on studies of unipolar depression.

The *DSM-III* divides unipolar depression into major depressive disorder (MDD) and dysthymic disorder (DD). The diagnosis of MDD is given to patients who have the acute experience of severe depressive symptoms for a period of 2 weeks or more.

Table 1
DSM-III Categories for Unipolar Depression and the Corresponding DSM-II and ICD-9 Labels

Labels	Major depressive disorder subtypes				Dysthymic disorder
	Unspecified	With melancholia	Without melancholia	With psychotic features	
<i>DSM-II</i>	Manic-depressive illness, depressed type	Involuntional melancholia	Depressive neurosis	Involuntional melancholia; psychotic depressive reaction	Depressive neurosis
<i>ICD-9</i>	Manic-depressive reaction, depressed type	Endogenous depression; involuntional melancholia	Reactive depression	Depressive psychosis; psychotic depression; reactive depressive psychosis; psychogenic depressive psychosis	Neurotic depression

Note. *DSM-II* and *DSM-III*: *Diagnostic and Statistical Manual of Mental Disorders* (2nd ed., 3rd ed; American Psychiatric Association, 1968, 1980). *ICD-9*: *The International Classification of Diseases* (9th ed.; World Health Organization, 1980).

There are two subtypes of MDD. Major depressive disorder with melancholia is characterized by marked anhedonia, the somatic symptoms of depression (e.g., early morning wakening, psychomotor retardation), and extreme guilt. The subclass "MDD with psychotic features" is used when the patient shows gross impairment in reality testing. The diagnosis of DD is given to a person who chronically experiences moderate-to-severe depressive symptoms for at least a 2-year period. The *DSM-III* classification of depressive disorders departed in a number of ways from its predecessor, the *DSM-II*, and from the classification system used outside the United States, the *International Classification of Diseases (ICD)*; World Health Organization, 1980). So that studies using different classification systems can be more easily compared, Table 1 lists the types of depression identified by *DSM-III* and matches these labels with the most closely corresponding categories in the *DSM-II* and *ICD* systems.

Evidence for Sex Differences in Unipolar Depression

There are two sources of data on rates of depression in men and women: records of persons treated for diagnosed affective disorders and surveys of the general population in which respondents are asked about symptoms of depression they are experiencing. Many clinicians and researchers argue that clinically severe levels of depression and subclinical levels of depressive symptoms are two distinct types of depression, with different characteristics, causes, and courses (cf. Depue & Monroe, 1978). From this viewpoint, studies in which self-report questionnaires are used to detect depression in a community sample tell us nothing about the epidemiology or etiology of true depressive disorders. Others argue that clinical depression and subclinical depression are simply two points along a continuum of severity in depressive symptoms. In support of this argument, Hirschfeld and Cross (1982) found that clinical and subclinical depressions shared many of the same psychosocial risk factors, such as a high number of bad life events. Hirschfeld and Cross (1982) and Boyd and Weissman (1981) point out, however, that factors such as socioeconomic status, geographic setting (rural vs. urban residence), and sex role stereotypes can affect help-seeking behavior. From this viewpoint, then, it is im-

portant to look at both treated cases of depression and surveys of untreated depressive symptoms in any investigation of the epidemiology of depression. With regard to the explanations for sex differences in depression reviewed here, some of the explanations are more applicable either to severe depressions or to moderate levels of depression. Thus, in the present review, studies of treated cases of depression and community studies of depression are reviewed separately.

Very few of the studies described in this article were designed specifically to measure sex differences in depression. Most of the studies were general surveys of psychopathology in particular geographical areas. One goal of this review was to produce summary statistics describing the magnitude of the sex differences in depression across studies (after M.L. Smith & Glass, 1977). Rachman and Wilson (1980) have raised objections to such meta-analyses, because differences in the quality of methodologies vary greatly across studies. Thus, the conclusions regarding sex differences in depression drawn here are based only on studies that meet the following criteria for adequate methodology: (a) Standardized assessment procedures and/or standardized diagnostic systems are used to identify depression in a sample, (b) sample sizes are reasonably large (i.e., over 50), (c) the selection of the sample was reasonably random, and (d) data on unipolar depression are presented separately from data on bipolar depression. Some studies that did not meet these criteria are reviewed separately, because they provide the only available data on depression in certain countries outside the United States.¹

Treated Cases of Depression

Table 2 summarizes the data from the methodologically strong studies of treated cases of depression in the United States. Whenever possible, the rates of depression in women and men in each study were adjusted for the number of women and men who participated in the study. All but three of the ratios listed in Table 2 indicate that significantly more women than men were given a diagnosis of depression. The mean female-to-

¹The reader may obtain a list of omitted studies from the author.

Table 2
Studies of Treated Cases of Depression in the United States

Study	Diagnosis	F:M	Comments
Williams & Spitzer (1983)	Major depression	2.3**	Diagnoses given in the field trials for the <i>DSM-III</i>
	Dysthymic disorder	1.9**	
Faden (1977)	Depressive neurosis	1.4**	All admissions to inpatient psychiatric services in the United States, 1974-1975
	Psychotic depression	0.8**	
	Involuntal melancholia	1.5**	
	All affective disorders	1.3**	
Rosen, Bahn, & Kramer (1964)	Depressive reaction	1.6**	All admissions to outpatient psychiatric services in the United States, 1961-1962
	Psychotic depression	1.4**	
Pederson, Barry, & Babigian (1972)	Psychotic depression	1.8**	All admissions to inpatient and outpatient psychiatric services in Monroe County, NY, 1961-1962
Lemkau, Tietze, & Cooper (1942)	Depressive reaction	2.2	Admissions to a Baltimore hospital, 1936. Only 11 women and 5 men were given this diagnosis
S. H. Rosenthal (1966)	Depressive reaction	4.6 ^a	Admissions to a Massachusetts Mental Health Center, 1965
Weissman, Sholomskas, Pottenger, Prusoff, & Locke (1977)	Current or former depression	4.0**	Outpatients who had a Raskin Scale score of 7 or above. Sample excludes patients also given the diagnosis of alcoholism
Stangler & Printz (1980)	Major depression	0.8	Students at the University of Washington
	Dysthymic disorder	1.7*	

Note. F:M is the ratio of females to males, corrected for the number of females and males in the sample, if possible. Chi-squares were calculated to test for sex differences in the rates of disorder.

^a The chi-square could not be calculated because of insufficient data.

* $p < .05$. ** $p < .01$.

male ratio across all studies in Table 2 was 1.95:1. Thus, across all studies of treated depression in the United States reviewed here, nearly twice as many women as men have been diagnosed as depressed. A t test (R. Rosenthal, 1978) performed to test the null hypothesis that the average female-to-male ratio across studies would be 1:1 yielded a $t(14) = 3.26$ ($p < .01$), indicating that the observed sex differences in rates of depression were statistically significant.

Williams and Spitzer (1983) report the only large study to date of the rates of treated depressive disorders according to the *DSM-III* classification system. In the field trials for the *DSM-III*, clinicians in many different treatment settings across the United States used *DSM-III* criteria to diagnose their patients. The female-to-male ratios of 2.1 and 1.9 for MDD and DD, respectively, indicate a clear and significant preponderance of women among depressives (chi-square statistics for both ratios were significant at $p < .01$).

There are three studies in Table 2 in which women do not significantly preponderate among depressives. In Faden's (1977) report of all diagnoses of depression given to psychiatric inpatients in the United States in 1974-1975, significantly more men than women were diagnosed as psychotically depressed. Yet in both Rosen, Bahn, and Kramer's (1964) and Pederson, Barry, and Babigian's (1972) reports of psychiatric diagnoses in large samples, significantly more women than men were diagnosed as psychotically depressed.

Lemkau, Tietze, and Cooper (1942) reported that 11 women and 5 men out of 3,337 psychiatric patients in a Baltimore hospital in 1936 were given the diagnosis of depressive reaction. This yielded a female-to-male ratio for the diagnosis of 2.2, which appears substantial but was not statistically significant

because of the low numbers of men and women given the diagnosis.

The only other study in which women did not outnumber men among depressives was that reported by Stangler and Printz (1980). Stangler and Printz's (1980) data are from diagnoses given to University of Washington students seen in the university's psychology clinic. Nineteen of the 320 women in this sample and 14 of the 180 men were diagnosed as having MDD (the difference was nonsignificant). As we shall see later, studies of depressive symptoms reported by college students on questionnaires also have found no sex differences in the level of symptoms. However, note that in Stangler and Printz's study significantly more women than men were given the diagnosis of DD ($p < .05$).

The methodologically stronger studies of treated cases of depression outside the United States are summarized in Table 3. The mean female-to-male ratio in these studies was 2.39, $t(14) = 7.03$, $p < .01$, with all but one (Halevi, Naor, & Cochavy's [1969] study of depressive reactions in Israel) reporting significantly more women than men diagnosed as depressed.

Several of these studies report data from psychiatric registers, which are comprehensive records of all persons treated in psychiatric institutions and private practice in a large geographical area. All but one study (Gershon & Liebowitz, 1975) used the *ICD* criteria for diagnoses. Comparisons of the *ICD* and *DSM-III* systems are shown in Table 1. In Table 3, we see evidence that women significantly outnumber men among unipolar depressives in Denmark, Scotland, England, Wales, Australia, Canada, Iceland, and Israel.

Table 4 presents data from studies that only provided summary data on all affective disorders or that used either idiosyn-

Table 3
Studies of Treated Cases of Depression Outside the United States

Nation	Study	Diagnosis	F:M	Comments
Denmark	Weeke, Bille, Videbech, Dupont, & Juel-Nielsen (1975)	Depressive reaction Neurotic depression	3.8* 3.0*	Psychiatric register, 1960–1964
Scotland	Baldwin (1971)	Neurotic depression	2.4*	Scottish mental hospital admissions, 1977
England and Wales	Martin, Brotherson, & Chave (1957)	Neurotic depression	1.7*	Psychiatric register, 1949–1954
England	Dean, Walsh, Downing, & Shelley (1981)	Psychotic depression	1.8*	Psychiatric register, 1976
Australia	Berah (1983)	Neurotic depression	1.8*	State and general hospital patients, 1978–1981
	Krupinski & Stoller (1962)	Psychotic depression	3.0*	Admissions to a Victoria hospital, 1951–1952
Canada	Canadian Bureau of Statistics (1970)	Neurotic depression	2.2*	First admissions to psychiatric services, 1967
Iceland	Helgason (1977)	Psychotic depression	2.9*	Psychiatric register, 1966–1967
Israel	Halevi, Naor, & Cochavy (1969)	Reactive depression	1.3	Census of 41 psychiatric institutions, 1964
		Psychotic depression	1.8*	
		Involuntional melancholia	3.5*	
		All affective disorders	2.3*	
	Gershon & Liebowitz (1975)	Unipolar depression	2.0*	Inpatients at psychiatric hospitals
		(Feigner criteria)		

Note. F:M is the ratio of females to males, corrected for the number of females and males in the sample, if possible. Chi-squares were calculated to test for sex differences in the rates of disorder.

* The chi-square could not be calculated because of insufficient data.

* $p < .01$.

cratic criteria for diagnoses or small samples. The mean female-to-male ratio across these studies was 1.5, $t(24) = 6.26$, $p < .01$. Note that a number of the studies conducted in less modern cultures did not find significant sex differences in depression. Some of these studies had serious flaws, however. The two studies from India (Mohan, 1972; Rao, 1970) and the study of Nigeria (Ezeilo & Onyema, 1980) were conducted in hospitals built to accommodate 3–4 times more men than women. The data from Egypt (El-Islam, 1969), Iraq (Bazzoui, 1970), and Rhodesia (Buchan, 1969) were based on the impressions of one or two psychiatrists, without the use of conventional diagnostic criteria. In addition, Bazzoui (1970) and Rao (1970) point out that access to psychiatric treatment is more restricted for women than for men in many nonmodern countries. So it is not clear that the rates of depression in men and women in these studies illustrate a true absence of sex differences in depression in these countries.

As mentioned earlier, the ratio of female to male depressives varies greatly from country to country. Mazer (1967) demonstrated that in different cultures, criteria for diagnoses are differentially weighted, and certain diagnoses are more frequently applied than others. Even so, there is a consistent tendency for women to preponderate among depressives across a wide variety of nations.

The number of treated cases of a disorder does not represent the true rate of a disorder in a population, because often only the most severe cases of the disorder, or those persons most disruptive to society, or those in the upper socioeconomic classes are treated (Wing, 1976). Further, when psychiatric diagnoses are relied on, idiosyncracies in the application of diagnostic criteria from psychiatrist to psychiatrist (or country to country) can confound the data.

To avoid these problems and others, researchers have sought to measure the rate of depression in the general population, us-

ing structured interviews or self-report questionnaires. Reviews of these studies follow.

Community Studies of Depression

Several questionnaires and interviews have been used in studies of depression in U.S. samples. These include the Beck Depression Inventory (BDI; Beck & Beck, 1972), the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977), and the Zung Self-Report Depression Scale (SDS; Zung, 1965), all of which are self-report questionnaires. The originators of each of these questionnaires have designated cutoff scores, and persons scoring above these cutoffs are considered to be seriously depressed. In addition, there are structured interviews, such as the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978) and the Diagnostic Interview Schedule (DIS; Robbins, Helzer, Croughan, & Ratcliff, 1981). Information gathered in these interviews is used to make diagnoses according to *DSM-III* criteria or the similar Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978).

Studies that have used any of these instruments to measure depression in the United States are summarized in Table 5. Most of the studies carried out in large, heterogeneous samples show a significantly greater degree of depression among women. The mean female-to-male ratio in the studies in Table 5 was 1.62, $t(17) = 6.82$, $p < .01$.

Much higher rates of moderate-to-severe symptoms of depression in both men and women are found when self-report inventories are used to detect depression than when structured interviews are used to diagnose depressive disorders. Yet both methods reveal large sex differences in depression. For example, Myers et al. (1984) report a study in which a structured interview, the DIS, was used to diagnose MDD and DD in a general community sample. Myers et al. report that 4% of the women

Table 4
Weak Studies of Treated Cases of Depression Outside the United States

Nation	Study	Diagnosis	F:M	Comments	
Canada	Weissman & Klerman (1977)	All affective disorders	1.7*	Data from the World Health Organization Collaborative Study; rates of females and males with sub-types of affective disorder were unavailable	
Czechoslovakia		All affective disorders	2.1*		
Denmark		All affective disorders	1.8*		
Finland		All affective disorders	1.3*		
France		All affective disorders	1.6*		
Norway		All affective disorders	1.5*		
Poland		All affective disorders	1.4*		
Sweden		All affective disorders	1.8*		
Switzerland		All affective disorders	1.4*		
England and Wales		All affective disorders	1.8*		
New Zealand		All affective disorders	1.8*		
England	Cooper, Kendell, Gurland, Sartorius, & Farkas (1969)	All affective disorders	1.4		Admissions to a London hospital, $n = 145$
New Zealand	Christie (1968)	All affective disorders	2.1*		Patients given diagnosis of "affective disorder"; $n = 50$; the diagnostic criteria were unclear
The Netherlands	Saenger (1968)	Psychiatric ratings of severe depression	1.3		Persons admitted to a psychiatric hospital and rated by psychiatrist, $n = 289$
Hong Kong	Yap (1965)	Affective disorder initial episode	1.1	Diagnoses given to 130 patients admitted to a hospital	
India	Rao (1970)	recurrent episodes	1.7**	One-year follow-up of 62 patients	
	Mohan (1972)	Endogenous depression	0.6*	Patients treated by the author for depression, $n = 62$	
Egypt	El-Islam (1969)	Affective psychosis	1.4	Patients institutionalized in a hospital that primarily accommodates males, $n = 140$	
		Nonpsychotic depression	1.0	Patients seen by the author, $n = 157$; the diagnostic criteria were unclear	
Iraq	Bazzoui (1970)	Depression	1.2	Hospitalized patients ($n = 42$) and private practice patients ($n = 16$); the diagnostic criteria were unclear	
Rhodesia	Buchan (1969)	Depression	1.1	Patients seen by the author, $n = 77$; the diagnostic criteria and method of patient selection were unclear	
Nigeria	Ezeilo & Onyeama (1980)	Psychotic depression	0.8	Discharge diagnosis for 969 patients; "no conventional diagnostic inventories available for use."	
		Neurotic depression	1.6*		
Kenya	Vadher & Ndeti (1980)	Nonpsychotic depression	2.3*	Patients being treated with chemotherapy for depression, $n = 30$	

Note. F:M is the ratio of females to males, corrected for the number of females and males in the sample, if possible. Chi-squares were calculated to test for sex differences in the rates of disorder.

* The chi-square could not be calculated because of insufficient data.

* $p < .05$. ** $p < .01$.

interviewed were diagnosed as having had an MDD at some time during the previous 6-month period, whereas 1.7% of the men interviewed were given the same diagnosis (ratio = 2.4, $p < .01$). Women also significantly outnumbered men among adults, with a 6-month prevalence of DD in all age groups and cities. The average percentage of women given this diagnosis was 4%, whereas the average for men was 2% (ratio = 2.0, $p < .01$).

Contrast these data with data from a study by Eaton and Kessler (1981), in which a self-report inventory of depressive symptoms called the CES-D scale was completed by a nationwide sample of 2,867 adults. Eaton and Kessler report that 11% of the men in this study and 21% of the women scored in the severe range of the CES-D (i.e., 15 or above). This is a female-to-male ratio of 1.9 ($p < .01$). Thus, even though the rates of severe depressive symptoms in this study are five times those of

diagnosed depressive disorders in men and women in Myers et al's study, the female-to-male ratios in the two studies are nearly identical.

The last four studies listed in Table 5 show no significant sex differences in depression. The samples in these studies represent subgroups of the American population, specifically, the Old Order Amish (Egeland & Hostetter, 1983), university students (Hammen & Padesky, 1977), bereaved adults (Bornstein, Clayton, Halikas, Maurice, & Robins, 1973), and elderly adults (Blazer & Williams, 1980). One of the most interesting of these studies is Egeland and Hostetter's 6-year epidemiological study of affective disorders among the Old Order Amish in Pennsylvania. The Old Order Amish are an ultraconservative Protestant religious sect, whose members maintain a closed society separated from the modern world. Egeland and Hostetter established contacts within the Amish community who would in-

Table 5
Community Studies of Depressive Symptoms in the United States

Study	Criteria for "depressed"	F:M	Comments
Myers et al. (1984)	Major depression, diagnosed with the DIS	2.4**	DIS administered to 9,000 adults in St. Louis, MO; Baltimore, MD; and New Haven, CT.
	Dysthymic disorder, diagnosed with the DIS	2.0**	
Eaton & Kessler (1981)	Scores > 15 on the CES-D	1.9**	Nationwide sample of 2,867 adults
Frerichs, Aneshensel, & Clark (1981)	Scores > 15 on the CES-D	1.8**	Los Angeles County adults, $n = 1,003$
Radloff (1975)	Scores > 15 on the CES-D	1.3*	Kansas City blacks, $n = 283$
		1.4**	Kansas City whites, $n = 876$
		1.8**	Maryland whites, $n = 1,638$
Amenson & Lewinsohn (1981)	SADS		Sample of 998 adults living in Oregon
	Current unipolar depression	2.3**	
	Past depressive episodes	1.3**	
Weissman & Myers (1978)	SADS		SADS administered to 511 adults in New Haven, CT
	Current major depression	1.6	
	Current minor depression	1.2	
	Lifetime risk, major depression	2.1**	
	Lifetime risk, minor depression	2.0*	
Blumenthal (1975)	Scores in the severe range on the Zung SDS	1.8**	Sample of 320 married adults
Egeland & Hostetter (1983)	SADS: major depression	1.0	Community of 8,186 Old Order Amish
Hammen & Padesky (1977)	Scores in the severe range on the BDI	1.1	University students, $n = 2,272$
Bornstein, Clayton, Halikas, Maurice, & Robins (1973)	Feighner criteria	1.1	Sample of 92 bereaved adults
Blazer & Williams (1980)	DSM-III criteria for major depression	1.2	Structured interview given to 997 elderly persons

Note. F:M is the ratio of females to males, corrected for the number of females and males in the sample, if possible. Chi-squares were calculated to test for sex differences in the rates of disorder. BDI = Beck Depression Inventory (Beck & Beck, 1972). CES-D = Center for Epidemiological Studies Depression Scale (Radloff, 1977). DIS = Diagnostic Interview Schedule (Robbins, Helzer, Croughan, & Ratcliff, 1981). DSM-III = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; American Psychiatric Association, 1980). SADS = Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978). SDS = Self-Report Depression Scale (Zung, 1965).

* $p < .05$. ** $p < .01$.

form them when a member of the community appeared "disturbed." This individual would then receive a SADS interview conducted by Egeland or her colleagues, with diagnoses assigned when indicated. Over a 5-year period, 21 women and 20 men were given the diagnosis of major depression, indicating no sex differences in the rates of unipolar depression in this culture.

The methods Egeland and her colleagues used to ascertain cases of mental disorder may have led to the underdetection of some disorders, however. Because the criterion for being given a SADS interview by the researchers was that an individual showed clear disruption in his or her role functioning, disorders in which the individual quietly suffers, such as depression, may have gone unnoticed. This hypothesis is supported by the fact that Egeland found rates of affective disorder that were one-half those found in other studies. In addition, Egeland found equal rates of bipolar and unipolar depression among the Amish, whereas most studies find the rates of unipolar depression to be 10 times the rates of bipolar depression (Clayton, 1981). The symptoms of mania would be more likely to disrupt role functioning than the symptoms of depression. In general, the discrepancies between Egeland's data and data from other studies suggest that unipolar depression may have been underdiagnosed. In particular, because Amish women spend most of their time in the home, detection of moderate-to-severe levels of depression in women may have been very difficult.

The survey study of university students by Hammen and Padesky (1977), which found no sex differences in depression, is in line with Stangler and Printz's (1980) data indicating no

sex differences among students treated for depression. One possible explanation for the sex differences in young adults is that depression has an earlier onset in men. That is, it may be that those men who in their lifetimes will become depressed usually do so in young adulthood. After the early 20s, however, the incidence of male depression may decline. Women, on the other hand, may be as vulnerable as men to depression in young adulthood but never show the decrease in vulnerability to depression with age that men show. Data from several studies do not support this explanation. For example, Winokur, Tsuang, and Crowe (1982) found no significant differences between men and women in the median age of onset of unipolar depression in a sample of 225 hospitalized depressives. In addition, Spicer, Hare, and Slater (1973) found that first admissions for depression in England and Wales peaked at an earlier age for women than for men. Thus, it does not appear that the absence of sex differences in depression among college students can be explained by a tendency toward earlier onset of depression in men.

Another possible explanation for the absence of sex differences in depression in college students is that only women with exceptionally good mental health (e.g., who are not depressed) go to college. On the other hand, men who go to college may be more representative of the mental health of men in general, perhaps because men are expected to go to college more than women are, so even depressed men go to college. This hypothesis is supported by data from Radloff's (1975) study on levels of depression in a group of 18- to 25-year-olds that included persons not attending college as well as persons attending col-

lege. Radloff found that the mean depression scores of women in this group were significantly higher than men's scores. Similarly, Faden (1977) found that among a group of 18- to 24-year-olds that included both college students and people not in college, significantly more women than men were treated for depression in inpatient psychiatric units in the United States. Thus, it appears that the absence of sex differences in depression in college students does not generalize to the rest of that age group not in college. This supports the hypothesis that college women are self-selected for positive mental health.

Another subgroup of the American population in which no sex differences in rates of depressive symptoms often are found is the bereaved. For example, Bornstein et al. (1973) used the Feighner criteria (Feighner et al., 1972) to diagnose depression in a sample of 65 women and 27 men recently bereaved. One month after their spouse's death, 33% of the men and 36% of the women met the criteria for depression. One year later, 19% of the men and 17% of the women continued to be depressed. In a review of the literature on the physical and mental health of bereaved men and women, Stroebe and Stroebe (1983) concluded that women do not show as much of a decline in physical and mental health after the death of a spouse as men. Stroebe and Stroebe suggest that women are trained to expect the death of their spouse, especially in old age, more than men are. Thus, bereavement may not be as much of a shock for women as for men. In addition, men are not accustomed to having to cope with the daily chores of life, so when they lose their spouses, their daily lives are more disrupted.

Finally, some studies find no sex differences in depression among older Americans. For example, Blazer and Williams (1980) administered a structured interview to 997 elderly persons and found that the rates of MDD in men and women were not significantly different. Similarly, Ensel (1982) found no sex differences in mean scores on the CES-D scale for persons over the age of 50. However, Radloff (1975) did find that women over the age of 65 had higher mean CES-D scores than men over 65. Thus these data on sex differences in depression in the elderly are mixed. In those studies that did not find sex differences, it appears that rates of depression in men increase substantially with age, whereas rates of depression in women remain the same with age or decline slightly. Similarly, in the studies of bereaved adults (described previously), the absence of sex differences in depression appears because the levels of depression in men rise with bereavement to match those of women.

In sum, seven of the studies in Table 5 indicated that women are depressed significantly more often than men, but four studies showed no sex differences in depression. These studies, however, were conducted in nonrepresentative samples of the American population, specifically: the Amish, university students, the elderly, and the bereaved. Excluding these studies, the female-to-male ratio for depressive symptoms in the general U.S. population is 1.8:1.

In Table 6, studies of depression in the general populations of nations outside the United States that used standardized measures and diagnostic criteria are summarized. Again, although the female-to-male ratio varied considerably from one study to the next, the average ratio of 2.08 (excluding Vadher & Ndetei's [1981] study) indicated that significantly more women than men are depressed, $t(12) = 4.15, p < .01$.

Note that two of the studies in Table 6 in which there clearly are no sex differences in depression, that is, Leighton et al.'s (1963) study of a tribe in Nigeria and Bash and Bash-Liechti's (1969) study of rural Iran, were conducted in nonmodern cultures. Further, the rate of depression in these nonmodern areas was much lower than the rate in urban areas of Iran or in the other African studies, which were conducted near cities. This trend might reflect a self-selection among depressives to move into the city, where they could obtain treatment. On the other hand, it could reflect negative influences of urbanization and modernization on mental health. Recall that the rate of affective disorder in Egeland and Hostetter's (1983) study of the Old Order Amish, a strictly nonmodern culture, also was much lower than the rate of depression in the general U.S. population.

Summary. Women are diagnosed as having a depressive disorder significantly more frequently than are men and, with a few exceptions, report more depressive symptoms than do men in most geographical areas of the world. If the ratios for all the stronger studies of depression (i.e., those in Tables 2, 3, 5, and 6) are averaged, the mean female-to-male ratio is 2.02, $t(57) = 8.88, p < .01$. The populations in which sex differences in depression have not been consistently found include university students, the bereaved, the elderly, the Old Order Amish, and residents of some rural, nonmodern cultures.

Explanations for the Sex Differences in Depression

Several biological and psychosocial explanations have been proposed to account for the sex differences in depression. Weissman and Klerman (1977) briefly discussed many of these explanations. In this review, the evidence for and against each explanation for the sex differences in depression is evaluated, and conclusions are reached about the level of support for each explanation. First, however, we must examine the possibility that the sex differences in depression are artifacts of differences in the socioeconomic status of men and women, or differences in the willingness of men and women to show the common symptoms of depression.

Artifact Explanations

Artifact Explanation 1: It is an income effect, not a gender effect. By most indicators, women's economic status is lower than men's (U.S. Department of Commerce, Bureau of the Census, 1985). Women also do not attain the same levels of education as men. It could be that the differences observed in rates of depression in men and women are the result of differences in socioeconomic status instead of gender differences. Radloff (1975) and Ensel (1982) tested for this possibility by comparing men's and women's mean scores on the CES-D, controlling for income level, education level, and occupation. In both of these studies, women still had more depressed mean CES-D scores than men after all these socioeconomic indicators were taken into account. These results suggest that observed sex differences in depression are not simply the result of differences in income.

Artifact Explanation 2: Reporting biases. Some researchers have been concerned that the sex differences in depression result from men's unwillingness to admit to and seek help for

Table 6
Community Studies of Depressive Symptoms in Countries Outside the United States

Nation	Study	Criteria for "depressed"	F:M	Comments
Sweden	Essen-Moeller (1956)	ICD: affective disorder	1.8*	Structured interviews, $n = 2,550$
	Essen-Moeller & Hagnell (1961)	ICD: affective disorder	3.0**	Interviewed subjects from Essen-Moeller et al. (1956) 10 years later
Denmark	Sorenson & Stromgren (1961)	ICD: psychogenic depression and depressive neurosis	3.5**	Information taken from public records and interviews, $n = 4,876$
	T. Fremming (1961; cited in Helgason, 1961)	ICD: affective disorder	2.0**	Structured interviews, $n = 4,130$
Iceland	Helgason (1961)	ICD: current affective disorder; lifetime risk of affective disorder	1.8** 1.8**	Structured interviews, $n = 3,843$
Australia	Henderson, Duncan-Jones, Byrne, Scott, & Adcock (1979)	ICD: current depression	2.6	Structured interviews (PSE), $n = 157$
	Byrne (1980)	Scores in the depressed range on the Zung Self-Report Depression Scale	1.4*	Same sample as Henderson et al. (1979)
Uganda	Orley & Wing (1979)	ICD: affective disorder	1.6	Structured interviews (PSE), $n = 206$
Kenya	Vadher & Ndeti (1980)	ICD: affective disorder	18:0	Structured interviews (PSE), $n = 56$
Nigeria	Leighton et al. (1963)	DSM-II: neurotic depression	0.9	Structured interviews with 262 members of the Yoruba tribe
Iran				
Urban	Bash & Bash-Liechti (1974)	ICD: affective disorder	3.6**	Structured interviews, $n = 928$
Rural	Bash & Bash-Liechti (1969)	ICD: affective disorder	1.0	Structured interviews, $n = 482$

Note. F:M is the ratio of females to males, corrected for the number of females and males in the sample, if possible. Chi-squares were calculated to test for sex differences in the rates of disorder. *DSM-II* = *Diagnostic and Statistical Manual of Mental Disorders* (2nd ed.; American Psychiatric Association, 1980). ICD = *The International Classification of Diseases* (World Health Organization, 1980). PSE = Present State Examination (Wing, 1976).

* $p < .05$. ** $p < .01$.

depressive symptoms (Padesky & Hammen, 1977; Phillips & Segal, 1969). This hypothesis holds that men and women experience depressive symptoms equally frequently, and to the same degree, but because depressive symptoms are perceived as feminine (Chevron, Quinlan, & Blatt, 1978), men are less likely to admit to them.

A number of studies have failed to support this hypothesis. For example, King and Buchwald (1982) predicted that if this artifact hypothesis was true, men should be less willing than women to disclose symptoms in a public disclosure condition (e.g., an interview with the researcher), whereas fewer sex differences should be found in a private (anonymous) disclosure condition. Instead, King and Buchwald found men no less willing to disclose in the public condition than women, and neither sex was less willing to disclose symptoms in a public disclosure situation than in private. Bryson and Pilon (1984) have replicated these results. Both of these studies, however, used college students for subjects. Recall that there is no tendency toward sex differences in depressive phenomena in this population.

Clancy and Gove (1974) investigated the influence of three types of response bias on the endorsement of items on the Langner Mental Health Scale, which consists of 22 items representing psychological and physiological symptoms of distress (Langner, 1962). These three variables were "perceived desirability" of the items, "need for social approval," and the tendency to "yea-say" or "nay-say." In their random sample of 404 adults (not college students), Clancy and Gove found no significant sex differences in ratings of the undesirability of the mental health items or in need for social approval. Women, however, were more likely than men to nay-say. Women also had significantly higher scores on the Langner scale. When the

relations between the three response bias variables and the mental health scores were controlled for, the sex differences in mental health scores were even larger. These studies were replicated in similar studies by Gove and Geerken (1976) and Gove, McCorkel, Fain, and Hughes (1976).

The claim that women are more willing to seek psychotherapy for depression has also not been consistently supported in the literature. Women do go to medical professionals more often than men (Faden, 1977). In addition, Padesky and Hammen (1977) report a study of college students in which the level of depressive symptoms at which women said they would seek psychotherapy was lower than the level at which men said they would seek help. However, two studies of actual help-seeking behavior have found that men and women with similar levels of self-reported depressive symptoms were equally likely to seek psychiatric help or go to a general practitioner (Amenson & Lewinsohn, 1981; Phillips & Segal, 1969). In addition, Amenson and Lewinsohn (1981) found that men and women with equal levels of self-reported symptoms were equally likely to be diagnosed as depressed in a clinical interview.

In summary, the hypothesis that the lower rates of depression observed in men are due to men's unwillingness to admit to their depressive symptoms has not been consistently supported. Men appear to be just as likely to admit to and seek help for a given level of depression. Still, women appear to experience depression more commonly than men.

Artifact Explanation 3: Kinds of symptoms. According to this hypothesis, men and women are equally susceptible to depression, but depression in men often takes the form of "acting-out" behaviors instead of sadness, passivity, and crying, which

are symptoms commonly included in self-report inventories (Hammen & Peters, 1977). In particular, it has been suggested that the male equivalent of depression is alcoholism (Winokur & Clayton, 1967). Proponents of this argument point to statistics showing that twice as many men as women are diagnosed as alcoholics (e.g., Williams & Spitzer, 1983), and suggest that the rates of alcoholism in men make up for the absence of depression in men. This argument is boosted by evidence that in cultures in which alcohol consumption is strictly prohibited, such as among the Amish, no sex differences in depression are found (Egeland & Hostetter, 1983). In addition, many studies find high rates of depressive symptoms among alcoholic men (cf. Petty & Nasrallah, 1981).

Winokur and his colleagues (Cadoret & Winokur, 1974; Winokur & Clayton, 1967; Winokur, Rimmer & Reich, 1971) argued that depression and alcoholism are genetically linked to each other, with depressive features linked to female chromosomes and alcoholic features linked to male chromosomes. Evidence for a genetic link between depression and alcoholism comes from family history studies that show much higher rates of depression in the families of alcoholics and of alcoholism in the families of depressives than in comparison groups (Cadoret & Winokur, 1974; Cotton, 1979).

Yet there is evidence that depression is as likely a consequence as a cause of alcoholism in men. Petty and Nasrallah (1981), in a critical review of the literature on depression and alcoholism, found a much greater tendency for depression to follow alcoholism, especially in men, than for alcoholism to follow the onset of depressive symptoms. Cadoret and Winokur (1974) report that in patients suffering from both depression and alcoholism, most of the men reported becoming depressed at least 10 years after the onset of alcoholism. The alcoholics who did not become depressed tended to be periodic bingers rather than constant heavy drinkers. Cadoret and Winokur suggest that depression in male alcoholics is often due to the toxic effects of chronic alcoholism.

Even so, there is clearly some evidence that alcoholism and depression often covary within families and within individuals, and that men tend to show more alcoholism whereas women tend to show more depression. This is not, however, evidence that alcoholism and depression are the same disorder or that alcoholism is a symptom of depression. Instead, these two disorders can be considered two different maladaptive responses to difficult life circumstances. Societal restrictions against women drinking excessively may protect women who are vulnerable to alcoholism from developing the disorder. In the same vein, certain societal demands on men may protect them against depression. This is very different from saying that alcoholism is a symptom of depression. Rather, one should say that both depression and alcoholism could arise given environmental troubles, but societal demands result in sex differences in vulnerabilities to each disorder.

In summary, there apparently is little justification for dismissing the observed sex differences in depression as simply due to differences in men's and women's willingness to show the common symptoms of depression.

Biological Explanations

The pervasiveness of the sex differences in depression across cultures suggests that women's greater vulnerability to depres-

sion may be the result of biological characteristics unique to women. Two general types of biological explanations for the sex differences in depression have been proposed. The first group of explanations arises from evidence that women are particularly prone to depression during periods in which they experience significant changes in hormone levels. Several investigators have suggested that depression in women is brought about by changes in the levels of estrogen, progesterone, or other hormones.

The second group of biological explanations of the sex differences in depression attributes the differences to a greater genetic predisposition to depression in women. According to these explanations, women are more likely to inherit the disorder, because the genetic abnormality that leads to depression is somehow linked to the chromosomes that determine gender.

Hormones and moods. It is widely believed that hormonal fluctuations strongly affect moods in women. Women are believed to be more prone to depression during the premenstrual period, the postpartum period, and menopause, each of which is characterized by changes in the levels of a number of hormones. Specifically, after the onset of puberty, levels of estrogen and progesterone rise and fall sharply during the menstrual cycle (Ganong, 1984). During the first 2 weeks of the cycle, arbitrarily defined as the first 2 weeks after the onset of blood flow, levels of progesterone and estrogen remain quite low. Around the end of the first 2 weeks, the level of estrogen rises and peaks, then declines again. Near the 21st day of the cycle, the level of estrogen peaks again and the level of progesterone peaks for the first time. Then, during the last few days before the onset of the next menstrual flow, the levels of both hormones drop precipitously, and remain low until after the menstrual flow.

Levels of estrogen and progesterone also change dramatically during pregnancy, the postpartum period, and menopause. During pregnancy, both estrogen and progesterone are produced in large amounts. Then, shortly after a woman gives birth (the postpartum period), her estrogen and progesterone levels drop sharply. Similarly, during menopause estrogen and progesterone production decline to very low levels and remain there the rest of a woman's life.

If women commonly experience depression during these periods of hormonal change, this suggests that their greater vulnerability to depression in general may be the result of the negative effects of changes or imbalances in female hormones on moods. The notion that depressions during periods of hormonal fluctuation are common is not well supported, however. Menopausal depression was once thought common, and early *DSM* editions (*DSM-I* and *DSM-II*) called this type of depression involuntional melancholia. However, in the *DSM-III*, the category of involuntional melancholia was excluded from the classification of affective disorders in response to the absence of evidence for a unique, endogenous type of depression that typically arose in women during middle-to-late adulthood (Weissman, 1979; Winokur, 1973). Indeed, levels of self-reported depressive symptoms seem to drop in women as they pass through menopause (Frieze, Parsons, Johnson, Ruble, & Zellman, 1978).

Estimates of the incidence of postpartum symptoms are high, ranging from 30% to 60% (Sherman, 1971). Yet the great majority of women experiencing postpartum depressive symptoms

recover fully from them within 1 day (Pitt, 1973). O'Hara, Rehm, and Campbell (1982) and Atkinson and Rickel (1984) found that most women who remain depressed several weeks postpartum were already depressed before giving birth. Because hormone levels are quite different during pregnancy and postpartum, such data contradict the hormonal explanation for many postpartum depressions. Thus, the data on postpartum and menopausal depressions do not support the notion that hormonal fluctuations often are related to depression in women.

The rate of premenstrual depressions is estimated by some studies to be very high, perhaps as high as 90–100% (Janowsky, Gorney, & Mandell, 1967; see also Sherman, 1971). For example, Schuckit, Daly, Herrman, and Hineman (1975) administered a structured interview to 105 college women, asking about the regular occurrence of depression, anxiety, irritability, and crying during different periods of the menstrual cycle. Sixty-three percent of the students reported that they have at least one of these symptoms regularly during the premenstrual period; 33% of the students said they were often depressed during the premenstrual period.

Recently, research diagnostic criteria for premenstrual depression have been developed by Halbreich, Endicott, and Nee (1983) and Steiner, Haskett, and Carroll (1980). Halbreich et al. introduced the Premenstrual Assessment Form (PAF), a 95-item inventory of psychological and physiological symptoms. Respondents are asked to indicate, retrospectively, the severity with which they experience each symptom during the premenstrual period. Answers to the PAF can be compared to criteria for diagnosing a premenstrual major depressive syndrome; these criteria are quite similar to the *DSM-III* criteria for MDD. Halbreich et al. administered the PAF to 335 women, and found that 43% met the criteria for a premenstrual major depressive syndrome.

From the studies just cited, premenstrual depression appears to be quite common. Indeed, if 43% of the premenstrual women in any given sample, such as the sample in a questionnaire study of depression, were experiencing major depression as a result of being premenstrual, these premenstrual depressions could account for the greater rate of depression in women than men observed in such studies. We can examine this possibility with some simple calculations. Let us first consider the studies that use self-report inventories for depression. Many questionnaire studies that find significant sex differences in rates of depression report that approximately 20% of the female subjects and 10% of the male subjects score in the moderate-to-severe ranges of the depression questionnaires (*DSM-III*, American Psychiatric Association, 1980; Eaton & Kessler, 1981; Frerichs et al., 1981; Radloff, 1975). Imagine that in the absence of premenstrual influences, the true rate of depression among women would be 10%, just as it is in men. Could premenstrual depressive symptoms account for the additional 10% of the female subjects who score as depressed on the questionnaires? The premenstrual period lasts approximately 5 days in each 28-day cycle (Ganong, 1984). Thus, if we assume that the menstrual cycles of women in any given group are randomly distributed over the month, we can estimate that approximately 20% ($5/28$) of women participating in any given questionnaire study are premenstrual. In order for premenstrual depression to account for the excess

of women scoring as depressed (i.e., an additional 10% of the female sample, compared to the male sample), half (50%) of all women would have to experience moderate-to-severe depressive symptoms during the premenstrual period. That is, 20% (the average percentage of women scoring as depressed) = 10% (the assumed base rate of depression) + (20% [the percentage of women that are premenstrual in any study] \times 50%).

Recall that a number of studies have reported that the percentage of women who reported experiencing moderate-to-severe premenstrual emotional symptoms is at least 40% (Halbreich et al., 1983; Janowsky et al., 1967; Schuckit et al., 1975). This is not quite 50%, but a 40% incidence of premenstrual depressions would account for most of the excess depression in women.

However, the validity of questionnaires that ask subjects to retrospectively rate their mood levels during different phases of the menstrual cycle has been seriously questioned (Parlee, 1973). Studies using retrospective questionnaires apparently greatly overestimate the number of women who actually experience significant depressive symptoms during the premenstrual period. For example, Abplanap, Haskett, and Rose (1979) asked 33 women to complete a daily mood checklist, and once per month to complete the Moos Menstrual Distress Questionnaire (Moos, 1968). The Moos questionnaire asks subjects to rate the degree to which they experienced a number of premenstrual symptoms during different phases of their last menstrual cycle. On the retrospective Moos questionnaire, subjects reported having experienced significantly more symptoms during their last premenstrual period than at any other period in their cycle. However, Abplanap et al. found no relation between cycle phase and daily mood ratings (for similar results, see Persky, O'Brien, & Kahn, 1976). Similarly, Schuckit et al. (1975) found that of the 63% of their sample who claimed that they regularly experienced premenstrual increases in negative affect, only 7% (i.e., 4% of their overall sample) actually showed significant increases in depression during the premenstrual period. In sum, the evidence from daily mood ratings suggests that the sex differences in depression observed in community studies are probably not accounted for by the number of women reporting premenstrual depressive symptoms on depression questionnaires.

Yet if daily diaries indicate that most women do not experience significant premenstrual mood changes, why do women report on retrospective questionnaires that they feel more depressed during their premenstrual period than during other times? As Paige (1971) and Ruble and Frieze (1978) point out, even in westernized countries, societal attitudes toward menstruation are still very negative. Menstruating women are expected to hide all signs of blood flow, and the taboos against sex during menstruation are still commonly observed. It is possible that women's negative expectations about their emotional state during the premenstrual and menstrual phases represent their dread of the social inconvenience of these phases. Similarly, the actual increases in anxiety and depression in some women during the premenstrual and menstrual phases may be psychological reactions to the negative social consequences of menstruation and to the physical discomfort of menstruation (e.g., bloating and cramping), and not the direct result of biochemical fluctuations.

One way to test the notion that hormonal changes during the menstrual cycle affect women's moods and that these hormonally based mood changes contribute to the sex differences in depression would be to conduct a study in which depressive symptoms and cycle phase are measured repeatedly in a large sample. The incidence of premenstrual depression could then be established and the presence of premenstrual depression could be controlled for in comparisons of the rates of depression in men and women.

Although we do not yet have evidence that depressions caused by regular hormonal fluctuations in women contribute to the sex differences in community surveys of depression, it is still possible that abnormalities in hormonal functioning in women account for their higher rate of clinically severe depressions. Many hypotheses have been proposed for exactly how hormonal abnormalities would affect mood in women (see reviews by Janowsky & Rausch, 1985, and Rubinow & Roy-Byrne, 1984). Most of the theories of the effects of hormones on psychopathology have focused on the two ovarian hormones, estrogen and progesterone. Many of the studies of these hypotheses have used samples of women who sought treatment for severe premenstrual syndrome (PMS).

Because each of the periods during which women have been thought to be more vulnerable to depression (e.g., menopause, the premenstrual period, and the puerperium) are characterized by decreased estrogen levels, it has been proposed that estrogen withdrawal triggers depression and other premenstrual symptoms (Backstrom & Mattsson, 1975; Klaiber, Broverman, Vogel, & Kobayshi, 1979). The evidence for this hypothesis is indirect. Klaiber et al. (1979) report reductions of depressive symptoms in depressed women given estrogen therapy. In addition, some women ingesting estrogen in oral contraceptives also show decreases in depression (Bardwick, 1971; Moos, 1968).

Another estrogen-related hypothesis holds that high levels of estrogen in conjunction with low levels of progesterone are what lead to tension and dysphoria. One study found that women suffering from PMS had higher estrogen/progesterone ratios than women not suffering from PMS (Backstrom & Cartensen, 1974), and two other studies found elevated levels of estrogen in women suffering from PMS (Abraham, Elsner, & Lucas, 1978; Munday, Brush, & Taylor, 1981).

Others have argued that declines in levels of progesterone, not estrogen, trigger depressive symptoms. Increases and decreases in levels of progesterone are correlated with increases and decreases in depressive symptoms (Janowsky, Fann, & Davis, 1971). Some studies have found correlations between the degree of depression in some women and the amount of progesterin in the oral contraceptives they were using (Culberg, 1972; Grant & Pryse-Davies, 1968; Kutner & Brown, 1972). In addition, several studies have found lower levels of progesterone just prior to menstruation in PMS sufferers compared to controls (Abraham et al., 1978; Backstrom & Cartensen, 1974; Munday et al., 1981; S.L. Smith, 1976).

By what mechanism would estrogen or progesterone withdrawal lead to depression? Levels of estrogen and progesterone in women have been correlated with levels of certain neurotransmitters implicated in depression. For example, in women given estrogen and progesterone therapy, increased serotonin uptake has been noted (Cone, Davis, & Coy, 1981; Ladisch,

1977). Decreased serotonin uptake has been associated with depression in some cases (Baldessarini, 1986); thus it has been suggested that women experience depression during estrogen or progesterone withdrawal because serotonin uptake decreases with the withdrawal (Hackman, Wirz-Justice, & Lichtsteiner, 1972).

Declines in estrogen and progesterone levels also have been associated in some studies with increases in the activity of monoamine oxidase (MAO; Grant & Pryse-Davies, 1968; Janowsky et al., 1971). Monoamine oxidase facilitates the breakdown of norepinephrine, and norepinephrine depletion is associated with depression (J. W. Maas, 1975).

Thus there appears to be considerable evidence that hormonal abnormalities are related to depression in women. Yet there are as many studies that do not support the hormonal theories of female depressions as there are that do (see Janowsky & Rausch, 1985; Rubinow & Roy-Byrne, 1984). For example, the premenstrual decline in estrogen is not the only decrease in estrogen that occurs during the menstrual cycle. Just after a midcycle peak, estrogen levels fall sharply, and this decline is not associated with depressive symptoms (Dalton, 1964). Premenstrual symptoms are also uncommon during that part of the menstrual cycle when estrogen/progesterone ratios are at their highest. In addition, Backstrom, Sanders, and Leask (1983) found no differences in estrogen levels between PMS sufferers and controls.

A number of studies have found that progesterone levels in women who suffer severe premenstrual tension are no different from levels in women who do not (Andersch, Hahn, Andersson, & Isaksson, 1978; Andersen, Larsen, Steenstrup, Svendstrup, & Nielson, 1977). Munday et al. (1981) found that premenstrual symptoms emerged in PMS sufferers before declines in progesterone occurred in the menstrual cycle. Several studies have found that progesterone therapy is no more effective than placebo in alleviating dysphoria and other symptoms of the premenstrual syndrome (Copen, Milne, & Outram, 1969; Jordheim, 1972; Sampson, 1979; S.L. Smith, 1976). After reviewing studies of progesterone therapy and other therapies for premenstrual depression, Rubinow and Roy-Byrne (1984) concluded that "the bulk of the evidence in support of current popular treatments is derived from uncontrolled trials, and, as is true with studies of etiology, the lack of comparability across studies at even the most fundamental levels of population definition and symptom measurement makes the uniform demonstration of any result highly unlikely" (p. 168).

Other researchers have argued that fluctuations in the mineralocorticoids during the premenstrual phase may shift the salt and water balance in the central nervous system, causing emotional symptoms (Dalton, 1964; Janowsky et al., 1967). There is evidence that aldosterone, a mineralocorticoid, may fluctuate in parallel with depressive symptoms during the menstrual cycle (Demarchi & Tong, 1972; Janowsky, Berens, & Davis, 1973). Other studies, however, have found no differences in aldosterone levels in patients exhibiting PMS and normal controls (e.g., Munday et al., 1981).

Other researchers have suggested that depressive symptoms in the premenstrual, postpartum, and menopause phases may result from excess prolactin levels (see Rubinow & Roy-Byrne, 1984) or from fluctuations in the adrenocortical hormones,

such as androgens and glucocorticoids (Vermeulen & Verdonck, 1976; M.S. Walker & McGilp, 1978). Yet, again, there is as much evidence against these hypotheses as there is for them (see Janowsky & Rausch, 1985; Rubinow & Roy-Byrne, 1984).

In sum, hypotheses that abnormalities in fluctuations of hormones or other biochemicals in women are associated with severe depression have not been consistently supported. The evidence suggesting that biochemical fluctuations lead to mood changes is indirect, open to multiple interpretations, and contradicted by an equal amount of negative evidence.

Yet it is premature to conclude that hormonal fluctuations have no effects on mood in women, because many studies of hormones and moods have serious methodological flaws. In addition, it must still be explained why sex differences in depression do not emerge until after puberty, when cyclic changes in hormones and other biochemicals begin in women.

One other biological explanation of the sex differences in depression has been proposed. According to this explanation, women's greater vulnerability to both severe and mild depression can be attributed to a genetic predisposition toward depression. A review of the evidence for this explanation follows.

Genetic factors in the sex differences in depression. Affective disorder runs in families (Gershon, 1983; Weissman, Kidd, & Prusoff, 1982). This aggregation of affective disorders within families could be due to either genetic or environmental factors shared by family members. Yet in a review of twin studies of affective illness, Allen (1976) found that in dizygotic twins, the average concordance rate for unipolar depression was 11%, but in monozygotic twins the average concordance rate for unipolar depression was 40%. The substantially higher concordance rate for monozygotic twins indicates some sort of genetic transmission of the disorder.

Could it be that the sex differences in depression are due to a greater genetic predisposition to depression in women? A number of investigators have argued that serious affective illness is the result of a mutant gene on the X chromosome (Perris, 1966; Winokur & Tanna, 1969). Because females have 2 X chromosomes, they should be more at risk for depression than men.

One way investigators have tested the X-linkage hypothesis of affective disorder is to examine the correlation between affective disorders and two abnormalities known to result from mutations on the X chromosome, red-green color blindness and the Xg blood group. If affective disorder is caused by mutations on the X chromosome that are in close proximity to the X-chromosome mutations believed to cause color blindness or the Xg blood group, then we should see significant, high correlations between the presence of affective disorder and the presence of either color blindness or the Xg blood group. A number of studies have examined the family pedigrees of persons suffering from affective disorder and have found that relatives who show affective disorder also tend to have the Xg blood group or color blindness (e.g., Mendelwicz, Fleiss, & Fieve, 1972; Reich, Clayton, & Winokur, 1969; Winokur & Tanna, 1969).

However, Gershon and Bunney (1976) note that the family pedigrees used in these studies have often been incomplete and the statistical significance of the relations observed is often marginal. In addition, these linkage studies assume that the loci for affective disorder, color blindness, and Xg blood group occur in close proximity on the X chromosome. However, a large num-

ber of studies (reviewed by Gershon & Bunney, 1976) have indicated that the loci for color blindness and the Xg blood group are not near each other.

Another way to test the X-linkage hypothesis is to examine the transmission of affective disorder from parents to children. Specifically, if a father carries the mutant gene on his X chromosome (and therefore manifests affective disorder), all of his daughters will carry the mutant gene, because the father always gives his daughters an X chromosome. Yet none of an affected father's sons will carry the mutation, because the father always gives his sons a Y chromosome. If a mother carries the mutant gene on one of her two X chromosomes, then her daughters and sons have equal chances of carrying the mutant gene. In short, if affective disorder is linked to the X chromosome, we should observe father-daughter pairs of affected individuals, but no father-son pairs (except for sons suffering from reactive depressions). We should observe equal numbers of mother-daughter and mother-son pairs.

However, most family history studies of the X-linkage hypothesis have discovered more father-son pairs of affective disorder individuals than is compatible with transmission via the X chromosome (e.g., Fieve, Go, Dunner, & Elston, 1984; Green, Goetze, Whybrow, & Jackson, 1973). Gershon and Bunney (1976) compiled data from these and several other studies and found that for 106 father-son pairs in which the father had an affective disorder, 10 sons (roughly 10%) showed affective disorder. This prevalence is higher than that in the general population. If the X-linkage hypothesis were true, the prevalence of depression in the sons of fathers with affective disorder should be considerably lower than that in the general population.

More recently, Cloninger, Christiansen, Reich, and Gottesman (1978; see also Kidd & Spence, 1976) argued that most common psychiatric disorders are unlikely to result from major chromosomal abnormalities or individual abnormal genes. Instead, such disorders are more likely the result of an aggregation of minor genetic abnormalities that interact with environmental variables to make an individual vulnerable to disorder. Cloninger et al. group all the genetic and other familial factors that influence risk for a particular disorder under the label "liability," and argue that only individuals whose liability is above a certain threshold will manifest the disorder. If one sex manifests a disorder less frequently than another, this may be because that sex has a higher threshold for the disorder. For example, men may manifest depression less frequently because they have a higher threshold for developing the disorder. However, the relative invulnerability to depression of men also may be due to nonfamilial environmental factors that protect them. Cloninger et al. argue that we can determine whether observed sex differences in a disorder are probably due to genetic/familial factors or to environmental factors by examining the rates of the disorder in the parents and siblings of affected persons. Specifically, if less depression in men is due to a lesser genetic loading (i.e., higher liability threshold), then we would expect to see more depression in the relatives of depressed men than in the relatives of depressed women. This is because depressed men are more genetically deviant than depressed women, and thus are more likely to transmit their depression to relatives (and to have had their depression transmitted from their parents). However, if the sex differences in depression are due primarily to nonfamilial

environmental factors, we should expect no differences in the rates of depression among the relatives of depressed men and women.

Merikangas, Weissman, and Pauls (1985) applied these analyses to family history data from 133 diagnosed unipolar depressives. Complete pedigrees were obtained from all probands, and diagnostic assessments were made of all living relatives of the probands on the basis of SADS interviews (Endicott & Spitzer, 1978), medical records, and family history information. Merikangas et al. found that the relatives of male and female depressives were equally likely to be diagnosed as depressed. These data indicate that the sex differences in depression are not due to genetic factors, but to environmental factors.

Summary of the biological explanations. There is no consistent evidence that the observed sex differences in serious affective disorders are due to a greater genetic predisposition to the disorder in women. The evidence for the influence of fluctuations in female hormones and other biochemicals on mood was more mixed. Some studies provided indirect support for relations between levels of particular biochemicals and moods, but many others did not. The more general notion that depression is common during the premenstrual, postpartum, and menopause periods has not been supported.

Finally, the biological explanations of sex differences in depression, as a class of explanations, do not explain the absence of sex differences in certain subgroups, such as the Amish, university students, and bereaved persons. Psychosocial factors, such as the supportiveness of the Amish culture or the greater impact of a spouse's death on men than on women, more convincingly explain the variations across groups in sex differences in depression.

Psychoanalytic Explanations

According to classic psychoanalytic theory, women are more susceptible to the depressive process than men because of the personality structure that results from women's psychosexual development (Mitchell, 1974). During the Oedipal stage, while castration anxiety is motivating a boy to develop a superego, a girl "realizes" she and all other females have been deprived of a penis and all the power and status that accompany being male. The girl's realization leads to hostility toward her mother for this deprivation, a great decline in her own self-worth, and the growth of envy of her father and all males.

A girl's fixation on her father, which is the model for later love relationships with men, is a weak one, based on narcissistic love, rather than an object attachment, because it is motivated by a desire to get back a part of herself she feels she has lost. It is this tendency toward narcissistic love relationships, driven by penis envy, that puts women at greater risk for melancholia, according to psychoanalytic theory (Mitchell, 1974). The woman looks to men to make up for her losses, but she is inevitably and frequently disappointed. After such disappointment, she is prone to turning her libidinal energy back in on herself rather than reattaching it to new objects. Her already injured ego is confronted by the hostility she feels against the object just lost, and melancholia is likely to result.

The psychoanalytic description of the development of female personality has received almost no solid experimental support

(Lee & Hertzberg, 1978; Sherman, 1971). For example, there is no support for the Oedipal shift in girls from attachment to the mother to attachment to the father (Sherman, 1971). Similarly, there is little evidence of either castration anxiety in boys or penis envy in girls, although many women acknowledge an envy of men's social power.

Later psychodynamic theorists (e.g., Horney, 1967; Menaker, 1979) downplayed notions of biological determinism in personality development, emphasizing instead the interactions between biological roles and cultural restrictions on women's behaviors. These and many more contemporary psychoanalytic writers point to the restrictions placed on female sexual expression, power, and personal freedom by patriarchal cultures as sources of frustration for women. Further, women's roles as child-bearers and child-rearers conflict with their needs for self-development and independence. To the extent that a woman renounces her role as mother, she suffers the disapproval of others, and, according to most of the neo-Freudians, she denies the primary component of female self-definition. These restrictions on female expression, the inferior status of the female role, and the conflicts between the innate desire to bear children and needs for independence all contribute to a greater tendency toward masochism in women (Horney, 1967).

The neo-Freudian accounts of female psychology are rich in their discussions of the interactions between social pressures and biological pressures. Such explanations might do well in accounting for the prevalence of sex differences in depression across many cultures. Characteristically, the theoretical richness of the psychodynamic explanations is counterbalanced by the absence of empirical support for these explanations.

However, a number of the suggestions offered by the neo-Freudians on how women's roles as passive wives and mothers affect mental health have been incorporated in contemporary sex role explanations of female psychopathology. These sex role explanations have been empirically tested, and reviews of these tests follow.

Sex Role Explanations

One sex role theory of depression, proposed by Miller (1976) and Scarf (1980), draws heavily on the work of Karen Horney, a neo-Freudian. Horney (1967) argued that many women greatly overvalue love relationships as a result of perceived rejection by their fathers. This overvaluation of love leads to obsessive quests for self-affirmation through involvement in intimate relationships, as well as internalized rage at all competitors (other women) for lovers and at the lovers themselves.

Similarly, Miller (1976) and Scarf (1980) (see also Gilligan, 1982) have argued that a central element of women's roles in society is the nurturance of relationships. Women's greater concern with relationships has been viewed by traditional psychoanalytic theory and moral development theory as an indication that women do not advance to as high a stage of moral development as do men, who are more concerned with issues of justice and rationality. Instead, Miller (1976) and Gilligan (1982) argue, women's concern with relationships simply represents a different, equally valid approach to moral issues compared to men's approach.

Yet, these theorists suggest, women's concern with relation-

ships makes them more vulnerable to despair and depression. Scarf writes, "It is in terms of highly vested and extraordinarily important loving attachments that most women's secret self-assessments and interior appraisals of self-worth are made" (p. 95). When a love relationship fails, a woman loses her self-definition.

What is the evidence that women are more concerned with relationships than are men? Gilligan (1982) has reported a series of studies of men's and women's ways of reasoning about moral issues. Gilligan presents largely anecdotal evidence indicating that women are most likely to worry about how the problem presented to them would affect relationships between the persons involved, whereas men were more likely to invoke rules of justice in solving problems. Gilligan's work has been criticized by L.J. Walker (1984), however, for the far-reaching conclusions she makes on the basis of data from unstructured interviews conducted with small samples.

In an extensive review of studies of sex differences in social behaviors and self-concept, Maccoby and Jacklin (1974) found that, although women and girls describe themselves as more socially oriented than men, their actual behaviors do not show more concern with social relationships than men's behaviors do.

In addition, it is not clear why investing one's self-worth in interpersonal relationships should be more likely to lead to depression than investing it in material and professional success, as men are said to do. Are failed love relationships more frequent events over the life span than failures at work? Why would not men's lack of investment in interpersonal relationships make them more vulnerable to feelings of loneliness and loss?

Horney (1967), Miller (1976), and Scarf (1980) suggest that, because the desire for relationships is an inherent aspect of female personality, women who attempt to succeed in jobs will be continually faced with disturbing conflicts between their natural propensity toward relationships and demands to be independent and competitive in the job. A number of other sex role theorists have been concerned with the incompatible expectations put on a woman when she enters the marketplace (Frieze et al., 1978; Kohn, Wolfe, Quinn, & Snoek, 1965). That is, a woman may be expected to be both passive, unselfish, and supportive in line with her feminine role, and assertive, self-sufficient, and demanding if she is to achieve in her work. These dual sets of expectations are difficult because the woman may feel forced to violate one set, ignore one set, or perhaps live up to both sets of expectations at the same time (Katz, 1975). Deviance from sex role expectations may result in social rejection. For example, Costrich, Feinstein, Kidder, Marecek, & Pascale (1975) found that assertive women were rated as more unattractive and in need of psychotherapy than were assertive men.

Some support for the role conflict hypothesis comes from epidemiological studies of depressive symptoms in the general population. Aneshensel, Frerichs, and Clark (1981) administered the CES-D to 1,000 residents of Los Angeles County. They found that among persons who were married and employed, women reported significantly more depressive symptoms than men; this was not true among unmarried, employed persons, however. This pattern of results is in line with the assertion that having dual roles can be a risk factor for depression in women.

However, Radloff (1975) found that both working wives and

nonworking wives reported more depression than working men. In addition, Radloff found that nonworking wives were more depressed than working wives. Rosenfield (1980) and Ensel (1982) report similar patterns of results from data from depression questionnaires. These data do not support the role-conflict hypothesis described previously.

Gove and Tudor (1973) suggested that having two sets of duties or roles in one's life actually protects one against depression. What is wrong (in part) with the traditional female role, according to Gove and Tudor, is that it allows only one source of gratification, the family. Men may be protected against depression because when one source of gratification is taken away, either the family or work, men have that second source to fall back on. Data such as Radloff's (1975), Rosenfield's (1980), and Ensel's (1982), in which women who had both a family and a job were less depressed than nonworking wives, lend partial support to this hypothesis.

Other sex role theories focus on the subjective value of a woman's role in society compared to the value of a man's role. At a very young age, girls are more likely to say they wish they were boys than boys are to say they wish they were girls (Abel & Sahinkaya, 1962; Hartup & Zook, 1960; Parsons, 1978). This undervaluing of the female sex role relative to the male sex role has been attributed to the greater prestige, power, competence, size, and strength associated with men (Kohlberg, 1966).

Gove and Tudor (1973) argue that the traditional female role as homemaker is becoming increasingly boring and undervalued as more modern conveniences are introduced. They suggest that the sex differences in depression (and other neurotic disorders) can be attributed to the lesser value put on the female role, and the resulting lesser gratification women receive from fulfilling that role. The data cited in the present article indicating no sex differences in the Old Order Amish and in university students seem to support this explanation of the sex differences in depression. The male and female roles in Amish society, although different, are both seen as essential to the family and community (Egeland & Hostetter, 1983). In college students, the goals and life-styles of men and women are more similar than they are in noncollege populations (Hammen & Padesky, 1977). Thus, the equality in value given to the male and female roles in these two subcultures might result in the absence of sex differences observed.

Even if a woman accepts the traditional female role gladly at one point in her life, chances are her role will change dramatically several times in her life (Ginzberg, 1966; H. S. Maas & Kuypers, 1974). This role discontinuity is due to the different demands put on a woman when she becomes a wife and then a mother, when her children leave home, and when she becomes a widow. Each of these new phases in life demand major reorganizations and reorientations of time, effort, and values. Coupled with the stress of adjusting to new roles may be the loss of a particular gratification from the previous role (e.g., when children leave home; LeMasters, 1957).

These patterns of change in role obligations are said to have no parallel in a man's life cycle (H. S. Maas & Kuypers, 1974). Yet it certainly cannot be said that the roles of men do not make demands that induce stress. Indeed, when men and women are asked to indicate the number of stressful events in their lives, no sex differences are found (Uhlenhuth, Lipman, Balter, & Stern,

1974). It is not so much the number of demands put on women because of their sex role that makes them more vulnerable to low self-esteem or despair, but the conflicts or radical changes in the expectations for women and the society's devaluation of the stereotypical female role.

The biggest problem with sex role accounts of sex differences in mental health is the frequent absence in these accounts of a well-described process by which role conflict or undervaluation might lead to depression or other disorders. It is particularly difficult to explain why sex role pressures would lead some women and not others into depressions so severe they must be hospitalized. Some theorists draw on psychoanalytic notions about the need to express aggression outwardly to explain why the female sex role would predispose to depression (Bardwick, 1971; Chesler, 1972; Chodorow, 1974). That is, women are socialized to control aggressive feelings, which possibly leads to a greater tendency to introject anger. This description of a mechanism by which sex roles predispose to depression suffers from the lack of research for the psychoanalytic introjected hostility theory of depression (Sherman, 1971).

Other psychologists and sociologists suggest that the female sex role contributes to greater feelings of lack of control and helplessness, thereby leading to depression (see Radloff, 1975, for a review). This helplessness hypothesis is described in the next section.

Learned Helplessness Explanation

Learned helplessness has been defined as a set of motivational, cognitive, and affective deficits that occur when uncontrollable negative events are experienced. In humans these deficits include lowered response initiation, an inability to learn new response–outcome contingencies, sadness, and lowered self-esteem. According to the original learned helplessness theory (Maier, Seligman, & Solomon, 1969; Seligman, 1975), these deficits are the result of the expectation that outcomes will be uncontrollable, because no response can be found to control them. In the best-known application of learned helplessness theory, Seligman (1975) pointed out the similarities between depressive symptoms and helplessness deficits, and suggested that some depressions may be due to the expectation that one has no control over important events.

A reformulation of the original helplessness model was proposed by Abramson, Seligman, and Teasdale (1978) to account for inadequacies in the original model in explaining the generality, chronicity, self-esteem loss, and individual differences in human helplessness. The focus of the reformulated model was on the explanations people make for events. People who tend to explain bad events by causes that influence many areas of their lives (global causes), instead of causes that influence only one area (specific causes), will expect to be helpless in many areas of their lives. People who explain bad events by causes that are stable rather than unstable in time will expect to be helpless in the future. People who blame themselves instead of others for bad events will experience a loss of self-esteem. Thus, according to Abramson et al., people who habitually tend to explain bad events by internal, stable, and global causes (and explain good events by external, unstable, and specific causes) will be more vulnerable to depression than people with the opposite style.

Radloff and Monroe (1978) asserted that the epidemiological trends in the incidence of depression in different groups may be best explained by the learned helplessness model. High rates of depression are found among the poor, the undereducated, nonwhites, the unemployed, the ill, and of course, women. In each of these groups other than women, it is easy to see how one might expect his or her actions to be ineffectual in bringing about good events and avoiding bad events. How does learned helplessness explain the preponderance of women among depressives?

First, women may receive more "helplessness training" over their lifetimes than men (LeUnes, Nation, & Turley, 1980). In an extensive review of childrearing practices, Maccoby and Jacklin (1974) found that one of the few practices in which parents consistently treated girls and boys differently was in their attention to girls' and boys' actions. Whereas boys' behaviors and misbehaviors were praised or criticized accordingly, girls' behaviors were largely ignored. Similarly, when boys and girls misbehave in class, teachers are more likely to respond to the misbehavior of boys than that of girls (Dweck, Davidson, Nelson, & Enna, 1978; Serbin, O'Leary, Kent, & Tonick, 1973). Girls seem to have many opportunities to learn that their responses do not control outcomes. Cross-cultural studies indicate that in many cultures, boys are trained to be more self-reliant and active, whereas girls are encouraged to be dependent on others (Maccoby & Masters, 1970; Whiting & Whiting, 1975).

As adults, women can also expect that their actions will be less successful at generating desired outcomes than men's. In several experimental studies, men's performances at laboratory tasks were rated higher than equally good female performances (Deaux & Taynor, 1973; Pheterson, Kiesler, & Goldberg, 1971). Successful women are often rated as less acceptable and likable than unsuccessful women or successful men (Feather & Simon, 1975; Horner, 1968). Goldberg (1968; see also Lavach & Lainer, 1975) found that the achievements of women were rated as less valuable than the achievements of men. Outside of the laboratory, women are less likely to be promoted than men given similar job performances (Rosen & Jerdee, 1974).

On another level, women have more difficulty having their ideas taken seriously by groups than do men (Unger, 1978; Wahrman & Pugh, 1974). In addition, when a woman attempts to gain power over a situation by asserting that she has greater expertise, greater information, greater authority, or a greater right to make a decision than a man, she is often seen as being "out of place" (Johnson, 1976).

Besides experiencing more events that could give them a sense of helplessness, women may also be more prone to the maladaptive explanatory style the reformulated helplessness theory identifies as a risk factor for depression. A number of studies of children and college students have found that women are more likely to attribute success on academic tasks and other positive events to luck or the favors of others, and to attribute failures to a lack of ability and other stable, global factors (Breen, Vulcano, & Dyck, 1979; Dweck, 1975; Dweck & Repucci, 1973; Nicholls, 1975; Wieggers & Frieze, 1977). Such attribution patterns are associated with a greater tendency to become helpless after failing at tasks.

Although a wide range of studies have supported the hypothe-

sis that a maladaptive explanatory style is a risk factor for depression (for a review see Peterson & Seligman, 1984), most of the studies of the reformulated model have used populations in which sex differences in levels of depression are usually not found (e.g., university students, children, and hospitalized depressives). There have been no studies using a nonstudent adult sample to test whether sex differences in explanatory style account for sex differences in depression.

Of course, it is neither necessary nor sufficient under the helplessness theory to have a maladaptive explanatory style in order to become depressed. It is sufficient that one expects to be helpless in controlling the environment. As discussed previously, there is considerable evidence that women in many areas of the world are trained to have this expectation. Thus, learned helplessness theory could explain the preponderance of women among depressives across cultures. Direct tests of the helplessness theory account of sex differences in depression have not been done, however.

One test of this explanation of sex differences in depression would include measuring the number of uncontrollable events in the lives of a sample of (nonstudent) women and men, and assessing their explanatory styles and their levels of depression. The learned helplessness explanation of sex differences in depression would be supported if it was shown that a greater degree of uncontrollability and a more maladaptive explanatory style in women account for any sex differences observed in depression in the sample. A more important study would be a longitudinal study in which explanatory style, uncontrollable life events, and depression are first assessed in a large group of children and then reassessed frequently as the children go through puberty, adolescence, young adulthood, and older adulthood. The learned helplessness theory would predict that the emergence of sex differences in depression after puberty should be preceded by a divergence in men's and women's explanatory styles and uncontrollable life experiences, with women developing more maladaptive explanatory styles and beginning to experience more uncontrollable events than men. Similarly, if women become less vulnerable to depression in older adulthood (cf. Frieze et al., 1978), then this change in vulnerability should be preceded by improved explanatory style and a reduced number of uncontrollable events.

Summary

Five different explanations of the sex differences observed in depression have been reviewed. From the evidence currently available, not one of these explanations has been strongly supported.

In the final section of this article, a new explanation for the sex differences in depression is proposed. It is argued that, regardless of the initial source of a depressive episode, how an individual responds to his or her own depressed state may contribute to the severity, chronicity, and recurrence of an episode. Specifically, it is argued that the men's responses to their dysphoria are more behavioral and dampen their depressive episodes, whereas women's responses to their depressive episodes are more ruminative and amplify them.

Response Sets for Depressive Episodes

Most people occasionally experience mild-to-moderate episodes of depressive symptoms. For some of us, these episodes last only a few hours or days. Yet we somehow bring ourselves out of such episodes before we reach the point where we would say we are depressed on a questionnaire or would seek professional help. For others, however, initially mild depressive symptoms often become more severe, until they are moved to acknowledge it to those who ask or even to seek therapy.

There is reason to believe that the ways we respond to or cope with our moods affect the severity and chronicity of those moods (Rippere, 1977). In particular, persons who tend to respond to their own episodes of depression by engaging in activities designed to distract them from their mood appear to recover faster from depressive episodes than those who tend to be inactive and to ruminate about the causes and implications of their depressed moods (cf. Beck, Rush, Shaw, & Emery, 1979; Teasdale, 1985; Zullo, 1984).

Evidence is emerging from a number of studies that men and women show different patterns of responding to their own feelings of depression. The general results of these studies, which are described in more detail in the following sections, are that men, when depressed, tend to engage in activities designed to distract themselves from their mood. Women, when depressed, tend to be less active and to ruminate more about the possible causes of their mood and the implications of their depressive episodes.

Some may want to argue that such differences reduce the sex differences in depression to artifacts of response bias (cf. Funabiki, Bologna, Pepping, & Fitzgerald, 1980). I propose, however, that women's response tendencies toward depression are actually a *cause* of their greater tendency toward depression, whereas men's response tendencies actually lessen their rates of depression. That is, the more ruminative response style of women amplifies and prolongs their depressive symptoms, whereas the more active response style of men dampens and shortens their depressive symptoms. Indeed, men and women may not even need to have different vulnerabilities to the onset of depression for the sex differences in rates of depression to emerge. The sex differences in response tendencies for depressed moods and the effects of these differential responses on the severity and chronicity of depressed moods in themselves could account for the observed sex differences in rates of depression.

First, let us examine the evidence for sex differences in response tendencies for depressive episodes.

Sex differences in responses to depressive symptoms. In order to investigate the possible sex differences in responses to depressed moods, Nolen-Hoeksema (1986) presented college students with a "list of things people do when depressed" and asked them to rate how likely they would be to engage in the behaviors or thoughts described when depressed. The subjects were instructed to be sure to "rate these items according to what you think you *would* do, not what you *should* do." The men in the sample had significantly higher scores than women on 4 of 37 items: "I avoid thinking of reasons why I'm depressed," "I do something physical," "I play a sport," and "I take drugs." Each of these responses would tend to distract the

individual from his depressed mood. The women in the sample scored significantly higher than the men on "I try to determine why I'm depressed," "I talk to other people about my feelings," and "I cry to relieve the tension." These responses tend to focus and maintain the individual's attention on her mood.

Quite similar sex differences in responses to depressed mood have been found in other studies (Chino & Funabiki, 1984; Funabiki et al., 1980; Kleinke, Staneski, & Mason, 1982). For example, Kleinke et al. (1982) found that male college students were more likely than female students to say they coped with depression by thinking about other things, ignoring their problem, or engaging in physical activity. Female students were more likely to say they would cut down on responsibilities and activities when depressed, confront their feelings, and blame themselves for being depressed. Again, the men's responses to their mood tended to be active and designed to relieve the mood by distraction, whereas the women's responses tended to be less active and more likely to focus attention on their mood.

These sex differences in activity levels during depression may emerge during childhood. In a study of self-reported depressive symptoms in 168 children from 8 to 11 years old, Nolen-Hoeksema, Girgus, and Seligman (1986) found that the depressive symptoms distinguishing depressed girls from nondepressed girls were different from those distinguishing depressed boys from nondepressed boys. Discriminant function analyses of the children's answers to the Children's Depression Inventory (CDI; Kovacs, 1980) revealed that depressed boys more often endorsed items indicating misbehavior, such as "I never do what I am told," "I do bad things," and "I get into fights all the time." Depressed girls did not tend to have high scores on these misbehavior items. Instead, the depressed girls endorsed items indicating negative self-evaluation, self-preoccupation, and loneliness, such as "I hate myself," "I'll never be as good as other kids," and "I feel alone." (Both boys and girls scoring in the depressed range on the CDI had high scores on items indicating sadness, pessimism, and indecision.) These sex differences in most prominent depressive symptoms do not translate directly into sex differences in responses to depressed affect. Yet they do indicate that even in childhood, depressed boys tend to be active, whereas depressed girls tend to be more contemplative or self-focused.

Dweck and Gilliard (1975) provide observational evidence to support the more general notion that when stressed, men respond actively but women are more contemplative, even in childhood. They gave boys and girls experiences with unsolvable tasks and asked the children to state their expectancies for future success after each trial or only after the first and last trials. Their results indicate that asking for expectancy statements after each trial of failure was associated with decreases in persistence at later trials and in expectations for success at future trials in both boys and girls. When the experimenter asked for expectancy statements only after the first trial, the boys did not show decreased persistence on later trials. However, the girls showed decreases in persistence that were as large as those in the condition in which expectancy statements were asked for after every trial. That is, the girls appeared to engage naturally in self-evaluation and rumination about the future, regardless of whether or not they were asked to, whereas boys did not.

In summary, there is evidence that in both children and

adults, the responses of males to stress and feelings of sadness tend to be active and often designed to distract them from their mood. The responses of females to their episodes of depression tend to be inactive and likely to focus their attention back on the mood and the self. These sex differences in responses to one's own affective state contribute to the sex differences observed in rates of depression. That is, men's active response style toward depression dampens the severity of their depressed mood and shortens the episodes of depression. Yet women's ruminative, inactive response style amplifies and prolongs their episodes of depression.

Effects of response style on depressed moods. There are at least three mechanisms by which a ruminative, inactive response set for depression should amplify and prolong an episode, whereas an active response set for depression should dampen and shorten an episode. First, rumination interferes with attention, concentration, and the initiation of instrumental behaviors (Diener & Dweck, 1978; Heckhausen, 1980; Kuhl, 1981; Sarason, 1975). Kuhl (1981) has argued that these difficulties lead to increased failures and a greater sense of helplessness in controlling one's environment, and thereby contribute to depression. On the other hand, engaging in active behaviors when depressed increases the individual's chances for controlling the environment and obtaining positive reinforcers, thereby dampening an existing depressed mood.

Support for this argument comes from a study by Kuhl (1981). He classified subjects as either "state oriented" or "action oriented" on the basis of their responses to a questionnaire asking what they would do in response to a variety of bad events. Then he exposed these subjects to uncontrollable failures at cognitive tasks (i.e., the standard learned helplessness training). The state-oriented subjects showed more helpless behaviors on subsequent tasks than did the action-oriented subjects. Kuhl found that the state-oriented subjects did not appear to become helpless during training because they came into the training with a generalized belief that they could not succeed at the tasks. Rather the state-oriented subjects' excessive rumination about their failures during the helplessness training appeared to interfere with learning in subsequent tasks, thereby leading to poor performance on these tasks, compared to the action-oriented subjects. With regard to depression, Kuhl argues that state-oriented persons may be more likely to focus on their mood state when depressed and that this focus interferes with instrumental behavior. The individual then experiences increased failure and loss of control, which act as helplessness training.

A second mechanism by which a tendency to become ruminative when depressed would amplify depression, whereas a tendency to become active would dampen it, is suggested by Bower (1981) and Teasdale (1983, 1985). Bower, Teasdale, and others have demonstrated that mood state has a powerful effect on an individual's recall of past events, social perceptions, and ability to learn new material. Persons who are depressed or who have been made sad by affect induction procedures (cf. Velten, 1968) show greater access to negative memories and easier learning of negatively toned new material. For example, Teasdale and Fogarty (1979) induced a happy or sad mood in college students by having the subjects read a series of self-referent statements appropriate in tone and content to the mood to be

induced. Self-reports of mood by subjects confirmed that the procedures did induce the intended affect. Then the subjects were given a series of positively toned or negatively toned stimulus words and were asked to tell the experimenter what past experience each stimulus word brought to mind as quickly as the experience was recalled. The latency of retrieval for pleasant and unpleasant memories was measured. The time required to retrieve pleasant memories, relative to that needed to retrieve unpleasant memories, was significantly longer for the subjects with induced depression than for the subjects with induced happiness. Teasdale and Fogarty argued that these results indicate that mood state affects the accessibility of pleasant and unpleasant memories, with depressed mood enhancing the accessibility of unpleasant memories and impairing the accessibility of pleasant memories.

Bower (1981) and Teasdale (1985) describe a vicious cycle between mood and memory that would maintain and deepen a depressed mood. The depressed mood activates a storehouse of negative memories, which amplify the current depressed mood and lead one to interpret current events in light of memories of past failures and losses. The depressed mood is thereby exacerbated and extended.

An individual who tends to be inactive and ruminative in response to a depressed mood should be more likely to become caught in the vicious cycle between mood and memory described by Teasdale and Bower. This cycle would both amplify and maintain the depressed mood. An individual who tends to respond to a depressed mood by becoming active should be more likely to distract him- or herself from the mood and negative cognitions, thereby breaking the cycle and dampening the depressive episode.

Finally, ruminative response sets during depressive episodes may increase the likelihood that an individual will consider depressogenic explanations for current negative events (Diener & Dweck, 1981; Kuhl, 1981; Zullo, 1984), thereby increasing expectations of helplessness and hopelessness. That is, ruminating about one's current state generates depressing explanations that increase depression (e.g., "I am depressed because I really blew it at the meeting today"). Such explanations, according to the reformulated helplessness theory (Abramson et al., 1978), increase the individual's expectations that he or she will continue to have problems in the future. Another type of vicious circle is set up, in which the individual's expectations of uncontrollability lead to decreases in positive, goal-oriented behavior, and the resulting failures enhance the individual's sense of helplessness and depression (Radloff & Rae, 1979).

Support for this mechanism comes from Diener and Dweck's (1978) study with children. They gave children solvable or unsolvable puzzles and asked them to vocalize whatever they were thinking while working on the puzzles and after each puzzle. Diener and Dweck found that some children had many thoughts about their past and future performances and others did not seem to think much at all about explanations and expectations. Both the ruminating and the nonruminating children expressed some depressive explanations for their failures. Yet the ruminating children invoked these depressing explanations more frequently than the nonruminators. In addition, after trials with unsolvable puzzles, the ruminators showed more helplessness on future tasks than did the nonruminators. Diener and

Dweck argued that the more frequent invocation of depressing explanations by the ruminating children led to the greater helplessness deficits seen in them. Additional evidence for this argument comes from a study by Zullo (1984), who found that college students who showed both a ruminative style and a tendency to explain events in pessimistic terms (cf. Abramson et al., 1978) were more prone to depression than were students who only showed rumination or a tendency toward pessimistic explanations.

In sum, I have argued that a ruminative response set for depression may amplify depressive episodes, relative to an active response set for depression, by (a) interfering with instrumental behavior, thereby increasing failures and a sense of helplessness; (b) increasing the accessibility of negative memories; and (c) increasing the chances that an individual will consider depressing explanations for his or her depression. Women appear to engage in more rumination and less distracting activity than men during depressive episodes. The sex differences in rates of depression arise because women's ruminative response styles amplify and prolong their depressive episodes by the mechanisms described previously, whereas men's active response styles dampen their depressive episodes.

Origins of response styles. Why would women be more ruminative and men more active in their responses to depressed moods? Being active and ignoring one's moods are part of the masculine stereotype. Being emotional and inactive are part of the feminine stereotype. From a very young age, children describe themselves and others in terms of sex role stereotypes, even before their actual behavior conforms to the stereotype (Brown, 1956; Nadelman, 1974; Schell & Silber, 1968). Parents reinforce behaviors consistent with these stereotypes; parents seem particularly concerned that boys not show feminine or sissy behaviors (Maccoby & Jacklin, 1974). Thus, the active response style of men toward their depressed moods may result simply from conformity to the sanctions against emotionality in men. Rumination in women may not be encouraged directly by parents or others; parents and teachers do not appear to reward girls for passivity and contemplation—they simply do not reward them as much for activity as they do boys (Dweck et al., 1978; Serbin et al., 1973). In addition, because women are told that they are naturally emotional, they may come to believe that depressed moods are unavoidable and cannot be easily dismissed when present. Such an attitude would decrease the probability of women taking simple actions to distract themselves from their moods.

Implications for interventions and the prevention of depression in women. If a ruminative response style amplifies women's vulnerability to depression, then the recommended interventions for depressed women should be ones that help to distract them from their mood and increase activity. In addition, it would be important to educate the depressed client about the distorting effects of mood on thinking and memory so that she realizes the difficulties involved in thinking clearly about problems and situations while depressed. Several of the interventions suggested by cognitive-behavioral therapies for depression (i.e., Beck et al., 1979; Teasdale, 1985) would be particularly appropriate for the treatment of depression in women. These interventions provide the client with exercises to disentangle herself from the effects of mood on thinking as well as a

structured approach to problem solving that helps her deal with existing problems in the most rational way possible. A ruminating depressive would be encouraged to engage in some activity when depressed in order to distract herself from the mood. She could also be discouraged from allowing herself to worry constantly. Beck, Emery, and Greenberg (1985) and others have recommended setting aside a half-hour at the end of the day to do one's worrying. The ruminator often finds her worries diminished in size and number by the time the "worry hour" arrives. In addition, the depressive should be encouraged never to make a decision or try to solve a problem when she is depressed, because she has particular access to negative memories and depressing attributions for events. Instead, she should do something to distract herself to relieve the mood state, then go back to the decision or problem to think about it.

To help prevent depression when grown, girls should be encouraged to be as active in response to their moods as boys are encouraged to be. This does not imply that women should be encouraged to become cold or unfeeling, or that they should fill their lives with distractions to avoid thinking about their real problems. I agree with Hammen and Peters's (1977) concern that sex role demands might prevent a seriously depressed man from seeking the help he needs. In addition though, sex role expectations for women might encourage them to pay too much attention to their own hedonics. Seriously depressed women may be more likely to be detected than seriously depressed men. Yet mildly depressed women may be more likely to become moderately or seriously depressed than mildly depressed men.

In addition, it is clear that sometimes feelings of depression should not be ignored. Often such feelings are indications that something is seriously wrong in one's life. Denying the signs of existing difficulties can be maladaptive. However, constant attention to one's own hedonics can also be maladaptive.

Conclusion

Many of the explanations for sex differences in depression reviewed here are part of the popular mythology about the mental health of women. This is especially true of the notions that hormonal fluctuations strongly affect moods in many women and that women have a natural preoccupation with interpersonal relationships. However, this review has revealed that most of the explanations for depression in women have little empirical support. Suggestions for future research on several of the existing explanations have been offered throughout this review. In addition, a new response set explanation for the sex differences in depression was proposed. This new explanation also requires further investigation.

One laboratory test of the new response set explanation would be to measure the tendency toward a ruminative versus an active response set for depression in a sample of men and women; then standard procedures (e.g., Velten, 1968) could be used to induce a depressed mood in the subjects. The chronicity and severity of the induced mood in the men and women would then be compared. According to the proposed response set explanation, women should ruminate more than men, and this ruminative response set should be related to more chronic and severe depressed moods in women.

Another way of testing the response set explanation would be

to conduct a longitudinal field study in which the response sets for depression and depressive symptoms are measured repeatedly over an extended period of time in a nonstudent sample. The response set explanation would predict that women should show more rumination during depression and that this style would be related to more frequent, more chronic, and more severe depressive episodes in women as compared to men.

One large study could be done that would simultaneously test several of the proposed explanations of sex differences in depression. This study would be longitudinal, beginning with a large sample of prepubescent children. The response sets for depression, explanatory style, uncontrollable life events, and depressive symptoms would be measured repeatedly (perhaps semiannually) and information concerning the onset of menses and the menstrual cycle phase would be gathered from the girls once they reached puberty. These variables then could be compared for their ability to predict the depressive episodes that women and men experience over their life spans and the emergence of sex differences in depression.

References

- Abel, H., & Sahinkaya, R. (1962). Emergence of sex and race friendship preferences. *Child Development, 33*, 939-943.
- Abplanap, J. M., Haskett, R. F., & Rose, R. M. (1979). Psychoendocrinology of the menstrual cycle. I. Enjoyment of daily activities and moods. *Psychosomatic Medicine, 41*, 587-604.
- Abraham, G. E., Elsner, C. W., & Lucas, L. A. (1978). Hormonal and behavioral changes during the menstrual cycle. *Senologia, 3*, 33-38.
- Abramson, L. Y., Seligman, M. E. P., & Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87*, 49-74.
- Allen, M. G. (1976). Twin studies of affective illness. *Archives of General Psychiatry, 33*, 1476-1478.
- Amenson, C. S., & Lewinsohn, P. M. (1981). An investigation into the observed sex differences in prevalence of unipolar depression. *Journal of Abnormal Psychology, 90*, 1-13.
- American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- Andersch, B., Hahn, L., Andersson, M., & Isaksson, B. (1978). Body water and weight in patients with premenstrual tension. *British Journal of Obstetrics and Gynaecology, 85*, 546-551.
- Andersen, A. N., Larsen, J. F., Steenstrup, O. R., Svendstrup, B., & Nielson, J. (1977). Effect of bromocriptine on the premenstrual syndrome. *British Journal of Obstetrics and Gynaecology, 84*, 370-374.
- Aneshensel, C. S., Frerichs, R. R., & Clark, V. A. (1981). Family roles and sex differences in depression. *Journal of Health and Social Behavior, 22*, 379-393.
- Atkinson, A. K., & Rickel, A. U. (1984). Postpartum depression in primiparous patients. *Journal of Abnormal Psychology, 93*, 115-119.
- Backstrom, T., & Cartensen, H. (1974). Estrogen and progesterone in plasma in relation to premenstrual tension. *Journal of Steroid Biochemistry, 5*, 257-260.
- Backstrom, T., & Mattsson, B. (1975). Correlations of symptoms in premenstrual tension to oestrogen and progesterone concentrations in blood plasma. *Neuropsychobiology, 1*, 80-86.
- Backstrom, T., Sanders, D., & Leask, R. (1983). Mood, sexuality, hormones and the menstrual cycle: II. Hormone levels and their relationship to the premenstrual syndrome. *Psychosomatic Medicine, 45*, 503-507.
- Baldessarini, R. J. (1986). A summary of biomedical aspects of mood

- disorders. In J. C. Coyne (Ed.), *Essential papers on depression* (pp. 459–492). New York: New York University Press.
- Baldwin, J. A. (1971). *The mental hospital in the psychiatric service: A case register study*. New York: Oxford University Press.
- Bardwick, J. M. (1971). *The psychology of women: A study of bio-cultural conflicts*. New York: Harper & Row.
- Bash, K. W., & Bash-Liechti, J. (1969). Studies on the epidemiology of neuro-psychiatric disorders among the rural population of the province of Khuzestan, Iran. *Social Psychiatry, 4*, 137–143.
- Bash, K. W., & Bash-Liechti, J. (1974). Studies on the epidemiology of neuro-psychiatric disorders among the population of the city of Shiraz, Iran. *Social Psychiatry, 9*, 163–171.
- Bazzoui, W. (1970). Affective disorders in Iraq. *British Journal of Psychiatry, 117*, 185–203.
- Beck, A. T., & Beck, R. W. (1972). Screening depressed patients in a family practice: A rapid technique. *Postgraduate Medicine, 52*, 81–85.
- Beck, A. T., Emery, G., & Greenberg, R. L. (1985). *Anxiety disorders and phobias: A cognitive perspective*. New York: Basic Books.
- Beck, A. T., Rush, J., Shaw, B., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.
- Berah, E. F. (1983). Sex differences in psychiatric morbidity: An analysis of Victorian data. *Australian and New Zealand Journal of Psychology, 17*, 266–273.
- Blazer, D., & Williams, C. D. (1980). Epidemiology of dysphoria and depression in an elderly population. *American Journal of Psychiatry, 137*, 439–444.
- Blumenthal, M. D. (1975). Measuring depressive symptomatology in a general population. *Archives of General Psychiatry, 32*, 971–978.
- Bornstein, P. E., Clayton, P. J., Halikas, J. A., Maurice, W. L., & Robins, E. (1973). The depression of widowhood after 13 months. *British Journal of Psychiatry, 122*, 561–566.
- Bower, G. H. (1981). Mood and memory. *American Psychologist, 36*, 129–148.
- Boyd, J. H., & Weissman, M. M. (1981). Epidemiology of affective disorders: A re-examination and future directions. *Archives of General Psychiatry, 38*, 1039–1046.
- Breen, L. J., Vulcano, B., & Dyck, D. B. (1979). Observational learning and sex roles. *Psychological Reports, 44*, 135–144.
- Brown, D. G. (1956). Sex-role preference in young children. *Psychological Monographs, 70* (14, Whole No. 421).
- Bryson, S. E., & Pilon, D. J. (1984). Sex differences in depression and the method of administering the Beck Depression Inventory. *Journal of Clinical Psychology, 40*, 529–534.
- Buchan, T. (1969). Depression in South African patients. *South African Medical Journal, 43*, 1055–1058.
- Byrne, D. G. (1980). The prevalence of symptoms of depression in an Australian general population. *Australian and New Zealand Journal of Psychiatry, 14*, 65–71.
- Cadoret, R., & Winokur, G. (1974). Depression in alcoholism. *Annals of the New York Academy of Sciences, 23*, 34–39.
- Canadian Bureau of Statistics. (1970). *Mental health statistics: Vol. 1. Institutional admissions and separations, 1967*. Ottawa: The Queen's Printer.
- Chesler, P. (1972). *Women and madness*. Garden City, NY: Doubleday.
- Chevron, E. S., Quinlan, D. M., & Blatt, S. J. (1978). Sex roles and gender differences in the expression of depression. *Journal of Abnormal Psychology, 87*, 680–683.
- Chino, A. F., & Funabiki, D. (1984). A cross-validation of sex differences in the expression of depression. *Sex Roles, 11*, 175–187.
- Chodorow, N. (1974). Family structure and feminine personality. In M. Z. Rosaldo & L. Lamphere (Eds.), *Women, culture, and society* (pp. 43–66). Stanford, CA: Stanford University Press.
- Christie, K. M. (1968). *A first assessment of costs and benefits associated with drug usage in New Zealand mental hospitals*. Auckland: New Zealand Institute of Economic Research.
- Clancy, K., & Gove, W. (1974). Sex differences in mental illness: An analysis of response bias in self-reports. *American Journal of Sociology, 80*, 205–216.
- Clayton, P. J. (1981). The epidemiology of bipolar affective disorder. *Comprehensive Psychiatry, 22*, 31–43.
- Cloninger, C. R., Christiansen, K. O., Reich, T., & Gottesman, I. (1978). Implications of sex differences in the prevalence of antisocial personality, alcoholism, and criminality for familial transmission. *Archives of General Psychiatry, 35*, 941–951.
- Cone, R. I., Davis, G. A., & Coy, R. W. (1981). Effects of ovarian steroids on serotonin metabolism within grossly dissected and microdissected brain regions of the ovariectomized rat. *Brain Research Bulletin, 7*, 639–644.
- Cooper, J. E., Kendell, R. E., Gurland, B. J., Sartorius, N., & Farkas, T. (1969). Cross-national study of diagnosis of the mental disorders: Some results from the first comparative investigation. *American Journal of Psychiatry, 125*(Suppl.), 21–29.
- Copen, A. J., Milne, H. B., & Outram, D. H. (1969). Dytide, norethisterone, and placebo in the premenstrual syndrome: A double-blind comparison. *Clinician Trials, 6*, 33–36.
- Costrich, N., Feinstein, J., Kidder, L., Marecek, J., & Pascale, L. (1975). When stereotypes hurt: Three studies of the penalties for sex role reversals. *Journal of Experimental and Social Psychology, 11*, 520–530.
- Cotton, N. S. (1979). The familial incidence of alcoholism: A review. *Journal of Studies on Alcohol, 40*, 89–116.
- Culberg, J. (1972). Mood changes and menstrual symptoms with different gestagen-estrogen combinations. *Acta Psychiatrica Scandinavica* (Suppl. 236), 1–86.
- Dalton, K. (1964). *Premenstrual syndrome*. Springfield, IL: Charles C Thomas.
- Dean, G., Walsh, D., Downing, H., & Shelley, E. (1981). First admissions of native-born and immigrants to psychiatric hospitals in south-east England, 1976. *British Journal of Psychiatry, 139*, 506–512.
- Deaux, K., & Taynor, J. (1973). Evaluation of male and female ability: Bias works two ways. *Psychological Reports, 32*, 261–262.
- Demarchi, G. W., & Tong, J. E. (1972). Menstrual, diurnal, and activation effects on the resolution of temporally paired flashes. *Psychophysiology, 9*, 362–367.
- Depue, R. A., & Monroe, S. M. (1978). Learned helplessness in the perspective of the depressive disorders: Conceptual and definitional issues. *Journal of Abnormal Psychology, 87*, 3–20.
- Diener, C. I., & Dweck, C. S. (1978). Analysis of learned helplessness: Continuous changes in performance, strategy, and achievement cognitions following failure. *Journal of Personality and Social Psychology, 36*, 451–462.
- Dweck, C. S. (1975). The role of expectations and attributions in the alleviation of learned helplessness. *Journal of Personality and Social Psychology, 31*, 674–685.
- Dweck, C. S., Davidson, W., Nelson, S., & Enna, B. (1978). Sex differences in learned helplessness: II. The contingencies of evaluative feedback in the classroom and III. An experimental analysis. *Developmental Psychology, 14*, 268–276.
- Dweck, C. S., & Gilliard, D. (1975). Expectancy statements as determinants of reactions to failure: Sex differences in persistence and expectancy change. *Journal of Personality and Social Psychology, 32*, 1077–1084.
- Dweck, C. S., & Repucci, N. D. (1973). Learned helplessness and reinforcement responsibility in children. *Journal of Personality and Social Psychology, 25*, 109–116.
- Eaton, W. W., & Kessler, L. G. (1981). Rates of symptoms of depression in a national sample. *American Journal of Epidemiology, 114*, 528–538.

- Egeland, J. A., & Hostetter, A. M. (1983). Amish study: I. Affective disorders among the Amish, 1976-1980. *American Journal of Psychiatry*, *140*, 56-61.
- El-Islam, M. F. (1969). Depression and guilt: A study at an Arab psychiatric clinic. *Social Psychiatry*, *4*, 56-58.
- Endicott, J., & Spitzer, R. L. (1978). A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, *35*, 837-844.
- Ensel, W. M. (1982). The role of age in the relationship of gender and marital status to depression. *Journal of Nervous & Mental Disease*, *170*, 536-543.
- Essen-Moeller, E. (1956). Individual traits and morbidity in a Swedish rural population. *Acta Psychiatrica Neurologica Scandinavica* (Suppl. 100), 1-160.
- Essen-Moeller, E., & Hagnell, O. (1961). The frequency and risk of depression within a rural population group in Scania. *Acta Psychiatrica Scandinavica* (Suppl. 162), 28-32.
- Ezeilo, B. N., & Onyema, W. (1980). Marital status and psychiatric illness: A Nigerian perspective. *Psychopathologie Africaine*, *16*, 309-319.
- Faden, V. B. (1977). *Primary diagnoses of discharges from nonfederal general hospital psychiatric inpatient units, U.S., 1975* (Mental Health Statistical Note 137). Rockville, MD: Department of Health, Education, and Welfare Publications.
- Feather, N. T., & Simon, J. G. (1975). Reactions to male and female success and failure in sex-linked cultures. *Journal of Personality and Social Psychology*, *31*, 20-31.
- Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, *26*, 57-63.
- Fieve, R. R., Go, R., Dunner, D. L., & Elston, R. (1984). Search for biological/genetic markers in a long-term epidemiological and morbid risk study of affective disorders. *Journal of Psychiatric Research*, *18*, 425-445.
- Frerichs, R. R., Aneshensel, C. S., & Clark, V. A. (1981). Prevalence of depression in Los Angeles County. *American Journal of Epidemiology*, *113*, 691-699.
- Frieze, I. H., Parsons, J. E., Johnson, P. B., Ruble, D. N., & Zellman, G. L. (1978). *Women and sex roles: A social psychological perspective*. New York: Norton.
- Funabiki, D., Bologna, N. C., Pepping, M., & Fitzgerald, K. C. (1980). Revisiting sex differences in the expression of depression. *Journal of Abnormal Psychology*, *89*, 194-202.
- Ganong, W. F. (1984). Physiology of reproduction and pregnancy. In R. C. Benson (Ed.), *Current obstetric and gynecologic diagnosis and treatment* (pp. 60-87). Los Altos, CA: Lange Medical Publications.
- Gershon, E. S. (1983). The genetics of affective disorders. In L. Grinspoon (Ed.), *Psychiatry update* (pp. 434-457). Washington, DC: American Psychiatric Press.
- Gershon, E. S., & Bunney, W. E. (1976). The question of X-linkage in bipolar manic-depressive illness. *Journal of Psychiatric Research*, *13*, 99-117.
- Gershon, E. S., & Liebowitz, J. H. (1975). Sociocultural and demographic correlates of affective disorders in Jerusalem. *Journal of Psychiatric Research*, *12*, 37-50.
- Gilligan, C. (1982). *In a different voice*. Cambridge, MA: Harvard University Press.
- Ginzberg, E. (1966). *Life styles of educated women*. New York: Columbia University Press.
- Goldberg, S. (1968). Are women prejudiced against women? *Transactions*, *5*, 28-30.
- Gove, W. R., & Geerken, M. R. (1976). Response bias in surveys of mental health: An empirical investigation. *American Journal of Sociology*, *82*, 1289-1317.
- Gove, W., McCorkel, J., Fain, T., & Hughes, M. (1976). Response bias in community surveys of mental health: Systematic bias or random noise? *Social Science and Medicine*, *10*, 497-502.
- Gove, W. R., & Tudor, J. F. (1973). Adult sex roles and mental illness. *American Journal of Sociology*, *78*, 812-835.
- Grant, E. C. G., & Pryse-Davies, J. (1968). Effect of oral contraceptives on depressive mood changes and endometrial monoamine oxidase and phosphates. *British Medical Journal*, *3*, 777-780.
- Green, R., Goetze, V., Whybrow, P., & Jackson, R. (1973). X-linked transmission of manic-depressive illness. *Journal of the American Medical Association*, *223*, 1289.
- Hackman, E., Wirz-Justice, A., & Lichtsteiner, M. (1972). The uptake of dopamine and serotonin in the rat brain during progesterone decline. *Psychopharmacologia*, *32*, 183-191.
- Halbreich, U., Endicott, J., & Nee, J. (1983). Premenstrual depressive changes. *Archives of General Psychiatry*, *40*, 535-542.
- Halevi, H. S., Naor, E., & Cochavy, Z. (1969). *Census of mental inpatients, 1964*. Jerusalem: Ministry of Health.
- Hammen, C. L., & Padesky, C. A. (1977). Sex differences in the expression of depressive responses on the Beck Depression Inventory. *Journal of Abnormal Psychology*, *86*, 609-614.
- Hammen, C. L., & Peters, S. D. (1977). Differential responses to male and female depressive reactions. *Journal of Consulting and Clinical Psychology*, *45*, 994-1001.
- Hartup, W. W., & Zook, E. A. (1960). Sex-role preferences in three- and four-year-old children. *Journal of Consulting Psychology*, *24*, 420-426.
- Heckhausen, H. (1980). Task-irrelevant cognitions during an exam: Incidence and effects. In H. W. Krohne & L. Laux (Eds.), *Achievement, stress, and anxiety*. Washington, DC: Hemisphere.
- Helgason, T. (1961). The frequency of depressive states in Iceland as compared with other Scandinavian countries. *Acta Psychiatrica Scandinavica* (Suppl. 162), 81-90.
- Helgason, T. (1977). Psychiatric services and mental illness in Iceland: Incidence study (1966-1967) with a 6-7 year follow-up. *Acta Psychiatrica Scandinavica* (Suppl. 268), 1-140.
- Henderson, S., Duncan-Jones, P., Byrne, D. G., Scott, R., & Adcock, S. (1979). Psychiatric disorder in Canberra: A standardized study of prevalence. *Acta Psychiatrica Scandinavica*, *60*, 355-374.
- Hirschfeld, R. M. A., & Cross, C. K. (1982). Epidemiology of affective disorders: Psychosocial risk factors. *Archives of General Psychiatry*, *39*, 35-46.
- Horner, M. S. (1968). *Sex differences in achievement motivation and performance in competitive and non-competitive situations*. Unpublished doctoral dissertation, University of Michigan, Ann Arbor.
- Horney, K. (1967). *Feminine psychology*. New York: Norton.
- Janowsky, D. S., Berens, S. C., & Davis, J. M. (1973). Correlations between mood, weight, and electrolytes during the menstrual cycle: A renin-angiotensin-aldosterone hypothesis of premenstrual tension. *Psychosomatic Medicine*, *35*, 143-154.
- Janowsky, D. S., Fann, W. E., & Davis, J. M. (1971). Monoamines and ovarian hormone-linked sexual and emotional changes: A review. *Archives of Sexual Behavior*, *1*, 205-218.
- Janowsky, D. S., Gorney, R., & Mandell, A. J. (1967). The menstrual cycle. Psychiatric and ovarian-adrenocortical hormone correlates: Case study and literature review. *Archives of General Psychiatry*, *17*, 459-469.
- Janowsky, D. S., & Rausch, J. (1985). Biochemical hypotheses of premenstrual tension syndrome. *Psychological Medicine*, *15*, 3-8.
- Johnson, P. (1976). Women and power: Toward a theory of effectiveness. *Journal of Social Issues*, *32*, 99-110.
- Jordheim, O. (1972). The premenstrual syndrome: Clinical trials of treatment with a progestogen combined with a diuretic compared with both a progestogen alone and with a placebo. *Acta Obstetrica Gynecologica Scandinavica*, *51*, 77-80.
- Katz, M. (1975, August). *Sex role training and coping behavior in a role*

- conflict situation: *Homemaking-career conflicts*. Paper presented at the 83rd Annual Convention of the American Psychological Association, Chicago, IL.
- Kidd, K. K., & Spence, M. A. (1976). Genetic analyses of pyloric stenosis suggesting a specific maternal effect. *Journal of Medical Genetics, 13*, 290-294.
- King, D. A., & Buchwald, A. M. (1982). Sex differences in subclinical depression: Administration of the Beck Depression Inventory in public and private disclosure situations. *Journal of Personality and Social Psychology, 42*, 963-969.
- Klaiber, E. L., Broverman, D. M., Vogel, W., & Kobayshi, V. (1979). Estrogen therapy for severe persistent depressions in women. *Archives of General Psychiatry, 6*, 550-554.
- Kleinke, C. L., Staneski, R. A., & Mason, J. K. (1982). Sex differences in coping with depression. *Sex Roles, 8*, 877-889.
- Kohlberg, L. (1966). A cognitive-developmental analysis of children's sex-role concepts and attitudes. In E. E. Maccoby (Ed.), *The development of sex differences* (pp. 82-172). Stanford, CA: Stanford University Press.
- Kohn, R. L., Wolfe, D. M., Quinn, R. P., & Snock, J. D. (1965). *Organizational stress: Studies in role conflict and ambiguity*. New York: Wiley.
- Kovacs, M. (1980). Rating scales to assess depression in school-aged children. *Acta Paedopsychiatria, 46*, 305-315.
- Krupinski, J., & Stoller, A. (1962). Survey of institutionalized mental patients in Victoria, Australia, 1882-1959. II. Analyses in terms of diagnoses. *Medical Journal of Australia, 1*, 1314-1315.
- Kuhl, J. (1981). Motivational and functional helplessness: The moderating effect of state versus action orientation. *Journal of Personality and Social Psychology, 40*, 155-170.
- Kutner, S. J., & Brown, W. L. (1972). Types of oral contraceptives, depression and premenstrual symptoms. *Journal of Nervous & Mental Disease, 55*, 153-162.
- Ladisich, W. (1977). Influence of progesterone on serotonin metabolism: A possible causal factor for mood changes. *Psychoneuroendocrinology, 2*, 257-266.
- Langner, T. (1962). A twenty-two item screening score of psychiatric symptoms indicating impairment. *Journal of Health and Human Behavior, 3*, 269-276.
- Lavach, J. F., & Lainer, H. B. (1975). The motive to avoid success in 7th, 8th, 9th, and 10th grade high achieving girls. *Journal of Educational Research, 68*, 216-218.
- Lee, D., & Hertzberg, J. (1978). Theories of feminine personality. In I. H. Frieze, J. E. Parsons, P. B. Johnson, D. N. Ruble, & G. L. Zellman (Eds.), *Women and sex roles: A social psychological perspective* (pp. 28-44). New York: Norton.
- Leighton, A. H., Lambo, T. A., Hughes, C. C., Leighton, D. C., Murphy, J. M., & Macklin, D. B. (1963). *Psychiatric disorder among the Yoruba*. Ithaca, NY: Cornell University Press.
- LeMasters, E. E. (1957). Parenthood as crisis. *Marriage and Family Living, 19*, 352-355.
- Lemkau, P., Tietze, C., & Cooper, M. (1942). Complaint of nervousness and the psychoneuroses: An epidemiological viewpoint. *American Journal of Orthopsychiatry, 12*, 214-223.
- LeUnes, A. D., Nation, J. R., & Turley, N. M. (1980). Male-female performance in learned helplessness. *Journal of Psychology, 104*, 255-258.
- Maas, H. S., & Kuypers, J. A. (1974). *From thirty to seventy: A forty-year longitudinal study of changing lifestyles and personal development*. San Francisco, CA: Jossey-Bass.
- Maas, J. W. (1975). Biogenic amines and depression. *Archives of General Psychiatry, 32*, 1357-1361.
- Maccoby, E. E., & Jacklin, C. N. (1974). *The psychology of sex differences*. Stanford, CA: Stanford University Press.
- Maccoby, E. E., & Masters, J. (1970). Attachment and dependency. In P. Mussen (Ed.), *Carmichael's manual of child psychology* (Vol. 2, pp. 1-101). New York: Wiley.
- Maier, S. F., Seligman, M. E. P., & Solomon, R. L. (1969). Pavlovian fear conditioning and learned helplessness. In B. A. Campbell & R. A. Church (Eds.), *Punishment and aversive behavior* (pp. 299-343). New York: Appleton-Century-Crofts.
- Martin, F. M., Brotherson, J. H. F., & Chave, S. P. W. (1957). Incidence of neurosis in a new housing estate. *British Journal of Preventative and Social Medicine, 11*, 196-202.
- Mazer, M. (1967). Psychiatric disorders in general practice: The experience of an island community. *American Journal of Psychiatry, 124*, 609-615.
- Menaker, E. (1979). The therapy of women in the light of psychoanalytic theory and the emergence of a new view. In L. Lerner (Ed.), *Masochism and the emergent ego: Selected papers of Esther Menaker*. New York: Human Services Press.
- Mendlewicz, J., Fleiss, J. L., & Fieve, R. R. (1972). Evidence for X-linkage in the transmission of manic-depressive illness. *Journal of the American Medical Association, 222*, 1624.
- Merikangas, K. R., Weissman, M. M., & Pauls, D. L. (1985). Genetic factors in the sex ratio of major depression. *Psychological Medicine, 15*, 63-69.
- Miller, J. B. (1976). *Toward a new psychology of women*. New York: Beacon Press.
- Mitchell, J. (1974). *Psychoanalysis and feminism*. New York: Random House.
- Mohan, B. (1972). *Social psychiatry in India: A treatise on the mentally ill*. Calcutta: The Minerva Association.
- Moos, R. H. (1968). The development of a menstrual distress questionnaire. *Psychosomatic Medicine, 30*, 853-867.
- Munday, M., Brush, M. G., & Taylor, R. W. (1981). Correlations between progesterone, oestradiol and aldosterone levels in the premenstrual syndrome. *Clinical Endocrinology, 14*, 1-9.
- Myers, J. K., Weissman, M. M., Tischler, G. L., Holzer, C. E., Leaf, P. J., Orvaschel, H., Anthony, J. E., Boyd, J. H., Burke, J. D., Kramer, M., & Stoltzman, R. (1984). Six-month prevalence of psychiatric disorders in three communities. *Archives of General Psychiatry, 41*, 959-967.
- Nadelman, L. (1974). Sex identity in American children: Memory, knowledge, and preference tests. *Developmental Psychology, 10*, 413-417.
- Nicholls, J. G. (1975). Causal attributions and other achievement-related cognitions: Effects of task outcome, attainment value, and sex. *Journal of Personality and Social Psychology, 31*, 379-389.
- Nolen-Hoeksema, S. (1986). Unpublished data, University of Pennsylvania, Philadelphia.
- Nolen-Hoeksema, S., Girgus, J. S., & Seligman, M. E. P. (1986). *Sex differences in depressive symptoms in children*. Unpublished manuscript, University of Pennsylvania, Philadelphia.
- O'Hara, M. W., Rehm, L. P., & Campbell, S. B. (1982). Predicting depressive symptomatology: Cognitive-behavioral models and postpartum depression. *Journal of Abnormal Psychology, 91*, 457-461.
- Orley, J., & Wing, J. K. (1979). Psychiatric disorders in two African villages. *Archives of General Psychiatry, 36*, 513-520.
- Padesky, C., & Hammen, C. (1977). *Help-seeking for depression: Sex differences in college students*. Unpublished manuscript, University of California, Los Angeles.
- Paige, K. E. (1971). Effects of oral contraceptives on affective fluctuations associated with the menstrual cycle. *Psychosomatic Medicine, 33*, 515-537.
- Parlee, M. B. (1973). The premenstrual syndrome. *Psychological Bulletin, 80*, 454-465.
- Parsons, J. E. (1978). Classic theories of sex-role socialization. In I. H. Frieze, J. E. Parsons, P. B. Johnson, D. N. Ruble, & G. L. Zellman

- (Eds.), *Women and sex roles: A social psychological perspective* (pp. 95–113). New York: Norton.
- Pederson, A. M., Barry, D. J., & Babigian, H. M. (1972). Epidemiological considerations of psychotic depression. *Archives of General Psychiatry*, 27, 193–197.
- Perris, C. (1966). A study of bipolar (manic-depressive) and unipolar recurrent depressive psychosis. *Acta Psychiatrica Scandinavica* (Suppl. 194), 1–89.
- Persky, H., O'Brien, C. P., & Kahn, M. R. (1976). Reproductive hormone levels, sexual activity and moods during the menstrual cycle. *Psychosomatic Medicine*, 38, 62–63.
- Peterson, C., & Seligman, M. E. P. (1984). Causal explanations as a risk factor for depression: Theory and evidence. *Psychological Review*, 91, 347–374.
- Petty, F., & Nasrallah, H. A. (1981). Secondary depression in alcoholism: Implications for future research. *Comprehensive Psychiatry*, 22, 587–595.
- Pheterson, G. I., Kiesler, S. B., & Goldberg, P. A. (1971). Evaluation of the performance of women as a function of their sex, achievement, and personal history. *Journal of Personality and Social Psychology*, 19, 114–118.
- Phillips, D. L., & Segal, B. E. (1969). Sexual status and psychiatric symptoms. *American Sociological Review*, 34, 58–72.
- Pitt, B. (1973). Maternity blues. *British Journal of Psychiatry*, 122, 431–433.
- Rachman, S. J., & Wilson, G. T. (1980). *The effects of psychological therapy: Second enlarged edition*. New York: Pergamon Press.
- Radloff, L. (1975). Sex differences in depression: The effects of occupation and marital status. *Sex Roles*, 1, 249–265.
- Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Journal of Applied Psychological Measurement*, 1, 385–401.
- Radloff, L. S., & Monroe, M. K. (1978). Sex differences in helplessness with implications for depression. In L. S. Hansen & R. S. Rapoza (Eds.), *Career development and the counseling of women* (pp. 199–221). Springfield, IL: Charles C. Thomas.
- Radloff, L. S., & Rae, D. S. (1979). Susceptibility and precipitating factors in depression: Sex differences and similarities. *Journal of Abnormal Psychology*, 88, 174–181.
- Rao, A. V. (1970). A study of depression as prevalent in south India. *Transcultural Psychiatric Research Review*, 7, 166–167.
- Reich, T., Clayton, P., & Winokur, G. (1969). Family history studies: V. The genetics of mania. *American Journal of Psychiatry*, 125, 1358–1369.
- Rippere, V. (1977). "What's the thing to do when you're feeling depressed?"—A pilot study. *Behavior Research and Therapy*, 15, 185–191.
- Robbins, L. N., Helzer, J. E., Croughan, J., & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry*, 38, 381–389.
- Rosen, B. M., Bahn, A. K., & Kramer, M. (1964). Demographic and diagnostic characteristics of psychiatric clinic outpatients in the U.S.A., 1961. *American Journal of Orthopsychiatry*, 34, 455–468.
- Rosen, B., & Jerdee, T. H. (1974). Effects of applicant's sex and difficulty of job on evaluations of candidates for managerial positions. *Journal of Applied Psychology*, 59, 511–512.
- Rosenfield, S. (1980). Sex differences in depression: Do women always have higher rates? *Journal of Health and Social Behavior*, 21, 33–42.
- Rosenthal, R. (1978). Combining results of independent studies. *Psychological Bulletin*, 85, 185–193.
- Rosenthal, S. H. (1966). Changes in a population of hospitalized patients with affective disorders, 1945–1965. *American Journal of Psychiatry*, 123, 671–681.
- Rubinow, D. R., & Roy-Byrne, P. (1984). Premenstrual syndromes: Overview from a methodologic perspective. *American Journal of Psychiatry*, 141, 163–172.
- Ruble, D., & Frieze, I. (1978). Biosocial aspects of reproduction. In I. H. Frieze, J. E. Parsons, P. B. Johnson, D. N. Ruble, & G. L. Zellman (Eds.), *Women and sex roles: A social psychological perspective* (pp. 191–209). New York: Norton.
- Saenger, G. (1968). Psychiatric outpatients in America and the Netherlands: A transcultural comparison. *Social Psychiatry*, 3, 149–164.
- Sampson, G. A. (1979). Premenstrual syndrome: A double-blind controlled trial of progesterone and placebo. *British Journal of Psychiatry*, 135, 209–215.
- Sarason, I. G. (1975). Anxiety and self-preoccupation. In I. G. Sarason & C. D. Spielberger (Eds.), *Stress and anxiety* (pp. 27–44). Washington, DC: Hemisphere.
- Scarf, M. (1980). *Unfinished business: Pressure points in the lives of women*. New York: Ballantine Books.
- Schell, R. E., & Silber, J. W. (1968). Sex role discrimination among young children. *Perceptual & Motor Skills*, 27, 379–389.
- Schuckit, M. A., Daly, V., Herrman, G., & Hineman, S. (1975). Premenstrual symptoms and depression in a university population. *Diseases of the Nervous System*, 36, 516–517.
- Seligman, M. E. P. (1975). *Helplessness: On depression, development and death*. San Francisco: Freeman.
- Serbin, L. A., O'Leary, K. D., Kent, R. N., & Tonick, I. J. (1973). A comparison of teacher response to the preacademic and problem behavior of boys and girls. *Child Development*, 44, 796–804.
- Sherman, J. A. (1971). *On the psychology of women: A survey of empirical studies*. Springfield, IL: Charles C. Thomas.
- Smith, M. L., & Glass, G. V. (1977). Meta-analysis of psychotherapy outcome studies. *American Psychologist*, 32, 752–760.
- Smith, S. L. (1976). The menstrual cycle and mood disturbances. *Clinical Obstetrics and Gynecology*, 19, 391–397.
- Sorenson, A., & Stromgren, E. (1961). The Samsø investigation. *Acta Psychiatrica Scandinavica* (Suppl. 162), 63–68.
- Spicer, C. C., Hare, E. H., & Slater, E. (1973). Neurotic and psychotic forms of depressive illness: Evidence from age-incidence in a national sample. *American Journal of Psychiatry*, 130, 535–541.
- Spitzer, R. L., Endicott, J., & Robins, E. (1978). Research diagnostic criteria: Rationale and reliability. *Archives of General Psychiatry*, 35, 773–782.
- Stangler, R. S., & Printz, A. M. (1980). DSM-III: Psychiatric diagnosis in a university population. *American Journal of Psychiatry*, 137, 937–940.
- Steiner, M., Haskett, R. F., & Carroll, B. J. (1980). Premenstrual tension syndrome: The development of research diagnostic criteria and new rating scales. *Acta Psychiatrica Scandinavica*, 62, 177–190.
- Stroebe, M. S., & Stroebe, W. (1983). Who suffers more? Sex differences in health risks of the widowed. *Psychological Bulletin*, 93, 279–301.
- Teasdale, J. D. (1983). Negative thinking in depression: Cause, effect or reciprocal relationship? *Advances in Behavior Research and Therapy*, 5, 3–26.
- Teasdale, J. D. (1985). Psychological treatments for depression: How do they work? *Behavior Research and Therapy*, 23, 157–165.
- Teasdale, J. D., & Fogarty, S. J. (1979). Differential effects of induced mood on retrieval of pleasant and unpleasant events from episodic memory. *Journal of Abnormal Psychology*, 88, 248–257.
- Uhlenhuth, E. H., Lipman, R. S., Balter, M. B., & Stern, M. (1974). Symptom intensity and life stress in the city. *Archives of General Psychiatry*, 31, 759–764.
- Unger, R. K. (1975). *Status, power, and gender: An examination of parallelisms*. Paper presented at the conference on New Directions for Research on Women, Madison, WI.
- U.S. Department of Commerce, Bureau of the Census. (1985). *Statistical abstracts of the United States, 1985*. Washington, DC: Author.

- Vadher, A., & Ndeti, D. M. (1981). Life events and depression in a Kenyan setting. *British Journal of Psychiatry*, *139*, 134-137.
- Velten, E. (1968). A laboratory task for induction of mood states. *Behavior Research and Therapy*, *6*, 473-482.
- Vermeulen, A., & Verdonck, L. (1976). Plasma androgen levels during the menstrual cycle. *American Journal of Obstetrics and Gynecology*, *125*, 491-494.
- Wahrman, R., & Pugh, M. D. (1974). Sex, non-conformity, and influence. *Sociometry*, *37*, 137-147.
- Walker, L. J. (1984). Sex differences in the development of moral reasoning: A critical review. *Child Development*, *55*, 677-691.
- Walker, M. S., & McGilp, S. (1978). Excretory patterns of urinary free 11-hydroxycorticosteroids and total oestrogens throughout the normal menstrual cycle. *Annals of Clinical Biochemistry*, *15*, 201-202.
- Weeke, A., Bille, M., Videbech, T., Dupont, A., & Juel-Nielsen, N. (1975). Incidence of depressive syndromes in a Danish county. *Acta Psychiatrica Scandinavica*, *51*, 28-41.
- Weissman, M. M. (1979). The myth of involuntional melancholia. *Journal of the American Medical Association*, *242*, 742-744.
- Weissman, M. M., Kidd, K. K., & Prusoff, B. A. (1982). Variability in rates of affective disorders in relatives of depressed and normal probands. *Archives of General Psychiatry*, *39*, 1397-1403.
- Weissman, M. M., & Klerman, G. L. (1977). Sex differences in the epidemiology of depression. *Archives of General Psychiatry*, *34*, 98-111.
- Weissman, M. M., & Myers, J. K. (1978). Affective disorders in a United States community: The use of research diagnostic criteria in an epidemiological survey. *Archives of General Psychiatry*, *35*, 1304-1311.
- Weissman, M. M., Sholomskas, D., Pottenger, M., Prusoff, B. A., & Locke, B. Z. (1977). Assessing depressive symptoms in five psychiatric populations: A validation study. *American Journal of Epidemiology*, *106*, 203-214.
- Whiting, B. B., & Whiting, J. W. M. (1975). *Children of six cultures: A psychocultural analysis*. Cambridge, MA: Harvard University Press.
- Wieggers, R. M., & Frieze, I. H. (1977). Gender, female traditionality, achievement level, and cognitions of success and failure. *Psychology of Women Quarterly*, *2*, 125-137.
- Williams, J. B. W., & Spitzer, R. L. (1983). The issue of sex bias in DSM-III. *American Psychologist*, *38*, 793-798.
- Wing, J. K. (1976). A technique for studying psychiatric morbidity in inpatient and outpatient series and general population samples. *Psychological Medicine*, *6*, 667-671.
- Winokur, G. (1973). Depression in the menopause. *American Journal of Psychiatry*, *130*, 92-93.
- Winokur, G., & Clayton, P. (1967). Family history studies: II. Sex differences and alcoholism in primary affective illness. *British Journal of Psychiatry*, *113*, 973-979.
- Winokur, G., Rimmer, J., & Reich, T. (1971). Alcoholism: IV. Is there more than one type of alcoholism? *British Journal of Psychiatry*, *118*, 525-531.
- Winokur, G., & Tanna, V. L. (1969). Possible role of X-linked dominant factor in manic-depressive disease. *Diseases of the Nervous System*, *30*, 89-93.
- Winokur, G., Tsuang, M. T., & Crowe, R. R. (1982). The Iowa 500: Affective disorder in relatives of manic and depressed patients. *American Journal of Psychiatry*, *139*, 209-212.
- World Health Organization. (1980). *The international classification of diseases* (9th ed.). Geneva: Author.
- Yap, P. M. (1965). Phenomenology of affective disorders in Chinese and other cultures. In A. V. S. deReuch & R. Porter (Eds.), *Transcultural psychiatry* (pp. 84-105). Boston: Little, Brown.
- Zullo, H. (1984). *The interaction of rumination and explanatory style in depression*. Unpublished master's thesis, University of Pennsylvania, Philadelphia.
- Zung, W. W. K. (1965). A self-rating depression scale. *Archives of General Psychiatry*, *12*, 63-70.

Received September 3, 1985

Revision received April 30, 1986 ■