Introduction

Childhood and adolescence are critical periods for the early identification of psychiatric symptoms and prevention of many mental disorders, including disruptive behavior, mood, and anxiety disorders. Substantial evidence is accumulating from longitudinal birth cohort studies and nationally representative surveys to suggest that there are developmental trajectories of psychiatric problems, many of which onset at young ages. Consequently, effective interventions during early life stages have the potential to support the positive neurobiological, cognitive, and psychosocial development that is needed for successful transitions from youth to adulthood. Epidemiologic studies can help inform priorities for service delivery by identifying the extent to which symptoms remit or develop into disorders during childhood and adolescence, the disorders that may be limited to this age range versus those that persist into adulthood, and the disorders that onset in adulthood for which symptoms may have manifested earlier.

The primary purpose of this chapter is to provide an overview of the prevalence and patterns of mental disorders specifically among girls and female adolescents, with special attention to variations across age (see Chaps. 3 and 4 in this volume on the epidemiology of mental disorders in adult women and older adult women, respectively). The epidemiologic focus on one gender is warranted for three reasons: (1) gender differences in rates of mental disorders among adults likely developed at young ages; (2) the influence of gender roles and expectations on the expression and interpretation of externalizing (behavioral) and internalizing (emotional) symptoms, which are believed to be more common among boys and girls, respectively; and (3) gender as a modifier of the illness risk and protective factors that have been identified at genetic, neurobiological, and psychosocial levels. The focus on
females is needed because, as will become obvious in the rest of the chapter, the preponderance of our knowledge about mental illness in the early stages of the life course is based on samples of males only, or samples of both males and females with the results reported for both genders together.

The chapter begins with a brief description of key epidemiologic research design issues that facilitate interpreting results on children and youth, and highlights methodological concerns of special relevance for girls and female adolescents. This background information establishes the extent to which results from disparate studies can be compared to one another and, by helping evaluate the strengths and limitations of our knowledge base, reveals areas for future research.

The authors then synthesize findings from studies focused on three diagnostic categories (i.e., attention deficit and disruptive behavior, mood, and anxiety) that capture some of the highest prevalence disorders, and most disabling in terms of morbidity and mortality. A fourth category, eating disorders, is included because of its unique relevance for adolescent females. It is beyond the scope of this chapter to summarize knowledge about substance use and disorder among female youth. However, readers are directed to Chap. 9 in this volume as well as to Armstrong and Costello (2002), who review community studies of comorbidity among adolescents with substance use disorders, including studies of gender differences.

In the final portion of this chapter, the authors identify research priorities and implications for the delivery of mental health services to girls, female adolescents, and women.

**Epidemiologic Research and Sensitivity to Girls and Female Adolescents**

The primary objective of this chapter was to identify studies of nonclinical samples (i.e., household or school samples) that focused exclusively on females or reported estimates separately by gender. While there are many studies based on pediatric and adolescent mental health treatment samples that address issues for girls and female adolescents, youths in these studies are likely to have the most serious problems, and selection effects would skew the estimates upward. Because of the rarity of community-based studies of preschool-aged children, however, the authors retained studies that recruited children from general medical practices.

Given the rapid changes children and adolescents experience as they age and the multiple contexts in which development occurs, there has been increasing recognition that diagnostic schemes need to be sensitive to the ways developmental stage affects symptom manifestation. For example, in 1994 a consensus group of early childhood development and mental health experts published the first Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood, and revised it in 2005 (DC: 0–3R; Zero to Three 2005). Other research evaluates the reliability and validity of assessment tools for disorders in young children (Sterba et al. 2007), including depression (Stalets and Luby 2006),
bipolar (Biederman et al. 2003), obsessive-compulsive (Merlo et al. 2005), anxiety (Seligman et al. 2004), posttraumatic stress (Lonigan et al. 2003), and disruptive behavior (Keenan et al. 2007) disorders.

The emphasis in most measurement and assessment studies has been on identifying age-appropriate measurements, with very little attention placed on investigating the possibility that different measures may be needed for boys and girls. Currently, boys and girls are evaluated using identical criteria that have typically been normed on samples of boys. Using this approach to measure symptoms of conduct and behavior disorder may potentially capture only the most serious cases among girls, thus underestimating their rate of disorder. The opposite approach would use completely different criteria for boys and girls, based on the assumption that normative gender differences do manifest in symptoms of disorder. With this approach, a greater number of girls might be screened as having a disorder, but they would be less impaired than boys. Studies that apply impairment criteria along with diagnostic criteria to calculate prevalence estimates essentially exclude the less serious cases, as was the result when inclusion of impairment criteria significantly reduced the prevalence of any mental disorder for both male and female adolescents in a community sample (Romano et al. 2001) and in a large sample of households enrolled in a health maintenance organization (Roberts et al. 2007).

In addition to assessment complexities presented when diagnosing children and youths across different developmental age periods, it has been acknowledged that multiple informants (e.g., parents, teachers, doctors, and youths themselves) may be needed for at least two reasons. First, at very young ages, children are not able to report reliably on their symptoms, especially if they are asked to report on symptoms experienced at times other than the most recent (Angold and Costello 1995). Second, different informants may be needed to provide a comprehensive understanding of symptom presentation and associated level of impairment when children are at home versus in other social settings. However, studies have shown that different informants will yield different estimates (Romano et al. 2001; Fergusson et al. 1993), presenting an ongoing challenge for researchers who seek to capture multiple dimensions of mental illness in one prevalence estimate, and highlighting concerns about who the most reliable reporter may be.

Similar to the orientation toward male standards in the research on measurement and nosology, a modest amount of research has examined the possibility that informants may have their own gender biases. For example, different informants yielded different prevalence rates of disruptive and antisocial behavior for girls (Hipwell et al. 2002), and teachers’ ratings systematically identified more male students than female students with disruptive disorders (Reid et al. 2000). Additionally, Manassas (2006) has argued that fearfulness may be underreported by parents of boys because of greater social acceptability and tolerance of fearfulness expressed by girls and gender role expectations regarding femininity.

The operationalization of “age” itself is a final methodological issue that may bear on interpreting estimates from most epidemiologic studies aiming to specify gender-specific rates of disorder at different life stages. Community- and school-based samples most often use chronological age or grade level as criteria on which
to select samples or to demarcate important transition stages in analyses. However, the onset of menarche is an additional measurement of age that may be particularly important for girls. For example, the frequency of panic attacks increased after puberty among girls (Hayward and Essau 2001), and higher rates of anxiety symptoms and disorders occur among girls who reach puberty at younger ages than their peers (Caspi and Moffitt 1991). Additionally, analysis of data from the Minnesota Twin Family Study (MTFS) revealed that girls who had an early age of menarche (before age 11) had higher rates of adolescent conduct disorder (CD) symptoms than those with average (age 12–13) or late onset (after 13) menarche (Burt et al. 2006). If data from the same girls had been analyzed according to grade level alone, there may have been no discernable differences in CD symptoms for girls in seventh versus eighth grade.

Overall, substantial data collection investments have been made that are beginning to allow empirical specification of the development of mental illness during childhood and adolescence. Studies of twins and birth cohorts followed longitudinally, sometimes for decades, allow identification of trajectories and continuity, whereas large cross-sectional studies representative of different age groups inform population-based prevalence estimates. Nevertheless, it is not yet possible to provide a comprehensive picture of female-specific developmental patterns of mental illness because of the dearth of studies that focus on girls and female adolescents. For the purposes of this chapter, it was necessary to incorporate findings from studies based on mixed gender samples, and to briefly address gender differences in the epidemiology of mental disorders. However, because of unanswered questions about the potential extent of gender bias in assessments and other methodological criteria as presented here, caution is warranted in assuming that results from mixed gender samples generalize to all-female populations, or that the magnitudes of gender-based prevalence differences are stable.

Prevalence of Disorders Among Girls and Female Adolescents

In this portion of the chapter, the authors present an epidemiologic overview of four specific diagnostic categories: (1) attention-deficit and disruptive behavior; (2) anxiety disorders; (3) affective disorders; and (4) eating disorders. For each specific diagnostic category, information is presented first about age- and gender-related patterns, followed by selected information on comorbidity between the focal disorder and disorders in other diagnostic categories. The comorbidity estimates are provided with an acknowledgment of the uncertainty in measuring comorbidity among youths, given the possibility that it reflects a unique classification of mixed disorders (Angold et al. 1999a) or a disorder of multiple dysfunction (Zoccolillo 1992).

To interpret data on specific diagnoses in the sections below, it is helpful to have basic information about the prevalence of mental illness and onset patterns among youth in general. In terms of overall estimates, Costello and colleagues (1998)
compiled seven data sets to estimate rates of serious emotional disturbance (i.e., psychiatric diagnosis with significant impairment), with resulting prevalence of 5.7% and 5.6% for girls and boys, respectively. Analysis of data from the longitudinal Great Smoky Mountains Study (GSMS) indicates that male and female children who have a mental disorder before age 13 compared to those who do not are significantly more likely to have a mental disorder at age 16, but the likelihood is significantly greater for girls than for boys (Costello et al. 2003). This result is consistent with higher rates of any mental disorder among females compared to males (15.5% vs. 8.5%) in another community sample of adolescents between 14 and 17 years (Romano et al. 2001).

There are three additional points that provide a context for understanding the summaries within a developmental epidemiology framework. First, data consistently show that early stages of the life course, from birth to young adulthood, are key periods for the identification, prevention, and treatment of mental illness. Retrospectively reported survey data from the National Comorbidity Survey Replication (NCS-R) indicate that mental disorders began by age 14 for one-half of the people who ever had a mental illness, and by the age of 24 for three-quarters of them (Kessler et al. 2005). Similarly, according to respondents in a prospective longitudinal birth cohort, three-quarters of adults had a diagnosis before age 18 (Kim-Cohen et al. 2003).

Second, lengthy follow-up periods in longitudinal studies allow analysis of the long-term consequences of psychosocial risk and protective factors, as well as the persistence of internalizing and externalizing symptoms. For example, prenatal and postnatal characteristics, such as very low birth weight (LBW) compared to normal birth weight, have been shown to be associated with significantly greater psychopathology in young adulthood (Dawson et al. 2000; Hack et al. 2004). Data from the 1946 British birth cohort, followed at ages 13, 15, 36, 43, and 53, showed that delays in achieving early developmental milestones, such as standing and walking, increased the likelihood of experiencing anxiety and depression symptoms, even controlling for childhood social circumstances and life events (Colman et al. 2007).

Third, each diagnosis has its own extensive literature describing the risk and protective factors that play a role in the etiology of the disorder, with increasing attention to the multiple levels at which the factors are expressed (genetic, hormonal, biological, psychosocial, and environmental). It is beyond the scope of this chapter to provide a comprehensive review of diagnosis-specific risk profiles, but it is worth pointing out that several studies also demonstrate nonspecific associations between risk and psychopathology in general. See, for example, the review of longitudinal studies on the association between childhood trauma and psychopathology (Pine and Cohen 2002), and the twin study that demonstrates genetic and environmental influences on a majority of diagnoses (Ehringer et al. 2006).

As a companion to the information presented in the specific diagnostic categories below, Table 2.1 highlights the studies that offered female-specific prevalence estimates and summarizes their key design features. The entries are listed alphabetically by author rather than by diagnosis because many studies reported on
<table>
<thead>
<tr>
<th>Study</th>
<th>Location</th>
<th>Sample size &amp; age</th>
<th>Diagnostic system &amp; instrument(s) &amp; informant(s)</th>
<th>Prevalence</th>
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<tbody>
<tr>
<td>Angold et al., 2002</td>
<td>North Carolina Schools</td>
<td>438 9–17 years</td>
<td>DSM-IV/ICD-10 CBCL/CAPA Parent/Child</td>
<td>0.9% P3M ADHD, 2.9% P3M CD, 2.8% P3M DD, 7.1% P3M AD</td>
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<tr>
<td>Bird et al., 1988</td>
<td>Puerto Rico Households</td>
<td>189 4–16 years</td>
<td>DSM-III CBCL/DISC/CGAS Parent/Teacher/Child</td>
<td>9.9% P6M OD, 9.5% P6M ADD, 5.9% P6M DD, 4.7% P6M SAD</td>
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<tr>
<td>Brown et al., 1996a</td>
<td>Western Oregon Schools</td>
<td>889 14–18 years</td>
<td>DSM-III-R K-SADS Child</td>
<td>18.4% LT MD, 8.4% LT AD, 7.3% LT DBD (At Time 1)</td>
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<tr>
<td>Brown et al., 1996a</td>
<td>Western Oregon Schools</td>
<td>889 14–18 years</td>
<td>DSM-III-R K-SADS Child</td>
<td>18.4% LT MD, 8.4% LT AD, 7.3% LT DBD (At Time 1)</td>
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<tr>
<td>Cohen et al., 1993a</td>
<td>Upstate New York Households</td>
<td>260 10–13 years</td>
<td>DSM-III-R DIS Parent/Child</td>
<td>2.3% (10–13); 7.5% (14–16); 2.7% (17–20)—MD, 8.5% (10–13); 6.5% (14–16); 6.2% (17–20)—ADD, 3.8% (10–13); 9.2% (14–16); 7.1% (17–20)—CD, 10.4% (10–13); 15.6% (14–16); 12.5% (17–20)—OD</td>
</tr>
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<td>Costello et al., 2003a</td>
<td>Great Smokey Mountains Study</td>
<td>3,005 9–16 years</td>
<td>DSM-IV CAPA Parent/Child</td>
<td>31.0% Any LT disorder, 12.1% LT AD, 11.7% LT DD, 16.1% LT DBD</td>
</tr>
<tr>
<td>Dey et al., 2004</td>
<td>United States Households</td>
<td>12,524 under 18 years</td>
<td>NHIS “If ADHD had been reported to family by doctor or other health professional”</td>
<td>4% ADHD</td>
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<tr>
<td>Deykin et al., 1987</td>
<td>Boston area</td>
<td>271 6–19 years</td>
<td>DSM-III-R DIS Child</td>
<td>6.8% LT MD</td>
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<td>Study</td>
<td>Location</td>
<td>Sample size &amp; age</td>
<td>Diagnostic system &amp; instrument(s) &amp; informant(s)</td>
<td>Prevalence</td>
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<td>Disney et al., 1999</td>
<td>Minnesota Twin Family Study</td>
<td>674 17 years</td>
<td>DSM-III-R DICA-R/CIDI Mother/Child</td>
<td>4.7% LT ADHD</td>
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<td>13.1% LT CD</td>
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<td>1.2% LT ADHD/CD</td>
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<tr>
<td>Feehan et al., 1994</td>
<td>Dunedin Multidisciplinary Health and Development Study—New Zealand</td>
<td>454 at age 18 years</td>
<td>DSM-III-R DIS Significant other/Child</td>
<td>2.6% PY GA; 1.1% PY PD</td>
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<td>14.8% PY Social Phobia; 8.8% PY Simple Phobia</td>
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<td></td>
<td>4.0% PY MD; 4.6% PY Dysthymia</td>
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<tr>
<td>Fergusson et al., 1993</td>
<td>Christchurch Health and Development Study—New Zealand</td>
<td>961 birth – 15 years</td>
<td>DSM-III-R DISC/DIS/RBPC Mother/Child/Teacher</td>
<td>9.7% Mood Disorders</td>
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<td>9.5% CD</td>
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<td>2.7% ADHD</td>
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<tr>
<td>Hipwell et al., 2002</td>
<td>Pittsburgh, PA Households</td>
<td>2,451 5–8 years</td>
<td>DSM-IV CGAS/CSI/SRA/AS Parent/Teacher/Child</td>
<td>5.4% (5); 4.1% (6); 5.1% (7); 4.3% (8)—PY ADHD</td>
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<td>4.3% (5); 3.5% (6); 4.7% (7); 4.0% (8)—PY ODD</td>
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<tr>
<td>Jensen et al., 1995</td>
<td>US Military Households</td>
<td>135 6–17 years</td>
<td>DSM-III-R DISC/CBCL/CES-D/CDI/RCMAS Parent/Child</td>
<td>25.2% P6M AD 4.3% P6M DD</td>
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<td>20.0% P6M ADHD</td>
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<td>5.2% P6M ODD</td>
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<td></td>
<td>1.9% P6M CD</td>
</tr>
<tr>
<td>Kashani et al., 1987</td>
<td>Columbia, MO Schools</td>
<td>75 14–16 years</td>
<td>DSM-III DICA Parent/Child</td>
<td>8% CD</td>
</tr>
<tr>
<td>Kessler and Walters, 1998</td>
<td>The National Comorbidity Survey—US Households</td>
<td>900 15–24 years (unweighted N)</td>
<td>DSM-III-R CIDI Parent/Child</td>
<td>8.0% PM MD</td>
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<td>16.1% PY MD</td>
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<td></td>
<td>20.6% LT MD</td>
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<td>Study</td>
<td>Location</td>
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<td>Diagnostic system &amp; instrument(s) &amp; informant(s)</td>
<td>Prevalence</td>
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<tr>
<td>Kilpatrick et al., 2003</td>
<td>National Survey of Adolescents—US Households</td>
<td>1,904 12–17 years (unweighted N)</td>
<td>DSM-IV &amp; NWS Child</td>
<td>6.3% P6M PTSD</td>
</tr>
<tr>
<td>Lahey et al., 2000</td>
<td>MECA New Haven, CT Puerto Rico Atlanta, GA Westchester, NY Households</td>
<td>604 9–17 years</td>
<td>DSM-III-R DISC Parent/Child</td>
<td>5.2% (9–11); 3.5% (12–14); 1.0% (15–17)—PY ODD 1.4% (9–11); 4.5% (12–14); 3.6% (15–17)—PY CD</td>
</tr>
<tr>
<td>Leaf et al., 1996</td>
<td>MECA New Haven, CT Puerto Rico Atlanta, GA Westchester, NY Households</td>
<td>1,285 9–17 years</td>
<td>DSM-III-R DISC/CGAS Parent/Child</td>
<td>5.6% (9–12); 7.4% (13–17)—Any P6M DISC diagnosis</td>
</tr>
<tr>
<td>Lewinsohn et al., 2000</td>
<td>Oregon Adolescent Depression Project—Schools</td>
<td>T1 = 891 (high school or below age 24) T2 = 810 (high school or below age 24) T3 = 538 (age 24)</td>
<td>DSM-III-R/DSM-IV K-SADS/CES-D Child</td>
<td>1.3% (T1); 2.3% (T2); 4.0 (T3)—PY AN or BN</td>
</tr>
<tr>
<td>Offord et al., 1987</td>
<td>Ontario, Canada Households</td>
<td>1,345 4–16 years 721 4–11 years 624 12–16 years</td>
<td>DSM-III CBCL Parent/Child/Teacher</td>
<td>1.8% (4–11); 4.1% (12–16)—P6M CD 3.3% (4–11); 3.4% (12–16)—P6M Hyperactivity</td>
</tr>
<tr>
<td>Pine et al., 2001+a</td>
<td>Upstate New York Households</td>
<td>388 9–18 years 380 11–20 years 358 17–26 years</td>
<td>DSM-III-R DISC Parent/Child</td>
<td>4.7% (9–18); 10.1% (11–20); 17.8% (17–26)—MD 4.2% (9–18); 12.6% (11–20); 8.2% (17–26)—Social Phobia 11.5% (9–18); 9.5% (11–20); 32.1% (17–26)—Specific Phobia</td>
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<tr>
<td>Study</td>
<td>Location</td>
<td>Sample size &amp; age</td>
<td>Diagnostic system &amp; instrument(s) &amp; informant(s)</td>
<td>Prevalence</td>
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<tr>
<td>Romano et al., 2001</td>
<td>Quebec, Canada Community sample</td>
<td>578 14–17 years</td>
<td>DSM-III-R DISC Mother/Child</td>
<td>15.5% Any DSM-III-R Diagnosis</td>
</tr>
<tr>
<td>Stice et al., 2004a</td>
<td>Southwest US Schools</td>
<td>496 11–15 years</td>
<td>DSM-IV EDE/K-SADS Child</td>
<td>1.6% (T1); 2.4% (T2); 1.8% (T3)—PY BN; 8.1% (T1); 9.7% (T2); 6.5% (T3)—PY MD</td>
</tr>
<tr>
<td>Wu et al., 1999</td>
<td>MECA New Haven, CT Puerto Rico Atlanta, GA Westchester, NY Households</td>
<td>604 9–17 years</td>
<td>DSM-III-R DISC/CGAS Parent/Child</td>
<td>4.3% DD 5.0% DBD 2.6% DD &amp; DBD</td>
</tr>
</tbody>
</table>

Sample size is reported for girls and female adolescents only. When multiple informants are noted, the prevalence rates reflect the estimates derived from parents’ reports. Unless otherwise specified, the reporting period for diagnoses was not reported. If a study used impairment criteria, estimates are reported based on diagnostic criteria only.

**Abbreviations of instruments:** AS = Antisocial Behavior Scale; CAPA = Child and Adolescent Psychiatric Assessment; CBCL = Child Behavior Checklist; CES-D = Center for Epidemiologic Studies Depression Scale; CDI = Children’s Depression Inventory; CGAS = Children’s Global Assessment Scale; CIDI = Composite International Diagnostic Interview; CSI = Child Symptom Inventory; DICA = Diagnostic Interview for Children and Adolescents; DIS = Diagnostic Interview Schedule; DISC = Diagnostic Interview Schedule for Children; EDE = Eating Disordered Examination; K-SADS = Schedule for Affective Disorders and Schizophrenia for School-Age Children; NWS = National Women’s Study; RBPC = Revised Behavior Problems Checklist; RCMAS = Revised Children’s Manifest Anxiety Scale; SRA = Self-Reported Antisocial Behavior Scale

**Abbreviations of reporting periods:** PM = past month; P3M = past 3 months; P6M = past 6 months; PY = past year; LT = lifetime

**Abbreviations of diagnoses:** AD = anxiety disorders; ADD = attention deficit disorder; ADHD = attention deficit hyperactivity disorder; AN = anorexia nervosa; BN = Bulimia Nervosa; CD = conduct disorder; DBD = disruptive behavior disorder; DD = depressive disorder; GA = generalized anxiety; MD = major depression; OCD = obsessive-compulsive disorder; OD = oppositional disorder; ODD = oppositional defiant disorder; PD = panic disorder; PTSD = posttraumatic stress disorder; SAD = separation anxiety disorder

* Longitudinal study
more than one disorder. A brief review of the table underscores the wide variability in age ranges and populations represented, assessment tools used, time frame for diagnostic reporting (e.g., past month, lifetime), informants (e.g., parents, teachers, children) and, if longitudinal, in follow-up periods. In particular, there is notable variation in the recall periods used for reporting prevalence estimates, including lifetime, past year, past 6 months, and past 3 months. Some of the variability is to be expected given the age ranges of the study respondents. For example, “lifetime” and “recent” may be a clinically meaningful distinction for an older adolescent, whereas current status (i.e., past month or past 3 months) may be the only meaningful period for a preschooler. The estimates included in the remaining sections of the chapter are “current” prevalence rates, unless otherwise stated. In addition, the chronological ages that correspond to different life stages are not entirely consistent, but in general, “early childhood” covers ages 0–3 years, “preschool” ages 3–5 years, “childhood” ages 5–11 years, and “adolescence” ages 12–18 years.

**Attention-Deficit and Disruptive Behavior Disorders**

Disruptive behavior disorders include attention deficit/hyperactivity disorder (ADHD), which is marked by inattention and impulsivity, CD, and oppositional defiant disorder (ODD; Rapoport and Ismond 1996). A CD diagnosis is based on persistent patterns of violations of rules, including aggression toward people and animals and destruction of property, whereas an ODD diagnosis speaks of negative, hostile, or defiant behavior. Notably, an ODD diagnosis is not given if a child or adolescent meets criteria for CD.

According to a systematic review of 102 general population and school-based studies of ADHD prevalence around the world, the pooled prevalence estimate for respondents 18 years or younger was 5.3% (Polanczyk et al. 2007). A higher prevalence rate (8.7%) for the USA has been reported based on data from the 8–15-year-old participants, assessed with DSM-IV criteria, in the cross-sectional 2001–2004 National Health and Nutrition Examination Surveys (Froehlich et al. 2007).

ADHD symptoms most likely onset before school age and rates of diagnosis increase as children age, although prospective studies of nonclinical preschool-aged populations are sufficiently rare that these conclusions should be treated tentatively. According to one of the few studies of disorder among a preschool population, the combined estimate for boys and girls between ages 3 and 5 years was 2% (Lavigne et al. 1996). Estimates between 3 and 5% in studies of elementary school students (Shepard et al. 2000) and between 6 and 9% for adolescents (Anderson et al. 1987; Bird et al. 1988) have been reported.

Rates of CD and ODD among very young children are difficult to find. The Environmental Risk Longitudinal Twin Study estimated a CD prevalence rate of 6.6% among 4.5 to 5-year olds (Kim-Cohen et al. 2005). Symptoms of CD occur before adolescence, between 5 and 8 years for both boys and girls (Lahey et al. 1998), with the onset of disorder in late childhood for boys and in early adolescence for girls
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(Cohen et al. 1993). CD prevalence estimates for female adolescents have ranged from 4% (Cohen et al. 1993) to 9% (Zoccolillo 1993).

The CD median onset age reported by respondents aged 18 years and older in the NCS-R was 11.6 years, with lifetime CD prevalence of 7.1% among females and 12.0% among males (Nock et al. 2006). Given the mixture of cross-sectional studies of adults retrospectively reporting symptoms of disorder, and longitudinal studies of children of all ages, it is challenging to determine whether early onset CD persists and additional cases occur as people age, or if the early onset cases remit and are replaced by people at older ages, all of whom are new cases. On the other hand, modest evidence about the persistence of externalizing disorders, measured as an aggregate category, comes from a study of children (aged 4–9 years) recruited from pediatric primary care. The disorder persisted for approximately one-third of the girls with a disorder at baseline (Briggs-Gowan et al. 2003). Additionally, there was no change in ODD prevalence in a pediatric sample recruited at ages 2–5 years and reinterviewed 4–6 years later (Lavigne et al. 2001). By adulthood, the lifetime prevalence of ODD is 9.2% among females and 11.2% among males (Nock et al. 2007).

Studies that provide insight into gender-based patterns in these diagnoses show that ADHD is between two and three times more common in boys than in girls. According to Polanczyk et al. (2007), the pooled ADHD prevalence estimate for females was close to 5% and 10% for males, with substantial variability in both estimates depending on the age ranges studied and diagnostic systems used. A review of research on CD and ODD reports that, like ADHD, rates of CD tend to be higher among boys compared to girls, whereas rates of ODD are roughly comparable (Loeber et al. 2000).

There are also gender-specific patterns in the symptoms that young males and females are likely to experience, although more research in this area appears to have been devoted to ADHD symptoms compared to CD or ODD symptoms. Results of a meta-analysis suggest that girls with ADHD had lower ratings on hyperactivity, inattention, impulsivity, and externalizing problems compared to boys with ADHD, but greater intellectual impairments and more internalizing problems (Gershon 2002). A few exceptions to this pattern have been noted. For example, Reid et al. (2000) reported a no-difference finding in ADHD symptoms among students aged 5–18 years. Greater impairment among girls compared to boys was also found in a small study (Rucklidge and Tannock 2001); among youths between ages 13 and 16 with ADHD, girls were more impaired than boys on the basis of self-reported anxiety, distress, depression, locus of control, and vocabulary scores.

It is somewhat unclear when gender differences emerge, although distinct ADHD trajectories have been noted as starting around age 6 years or upon entrance to school (Shepard et al. 2000), and higher CD prevalence among boys was consistent across 5–15-year olds in the cross-sectional British Child Mental Health Survey (Maughan et al. 2004). Similarly, there is ambiguity about the degree to which male–female disruptive behavior disorder rates continue to diverge during adolescence, or possibly converge during the transition from childhood to adolescence (Loeber et al. 2000). When boys and girls who had subthreshold conduct-related problems at ages
5 through 10 years were compared at ages 8 through 13, boys (25%) were more likely than girls (7%) to have CD (Messer et al. 2006).

Very high rates of comorbidity among the disruptive behavior disorders have been documented. Newcorn and Halperin (2000) estimated that between 40 and 70% of children with ADHD have ODD or CD. All three diagnoses (ADHD, ODD, and CD) are highly comorbid with anxiety disorders, with some variation in strength depending on the specific externalizing–anxiety pair (Marmorstein 2007). For example, approximately one-quarter of youth with ADHD have an anxiety disorder (Biederman et al. 1991). In a sample restricted to female twins, depression was a prevalent diagnosis among those with CD (Marmorstein and Iacono 2001). As expected, given gender differences in prevalence rates of disruptive disorders, comorbidity patterns vary as well (Boylan et al. 2007; Loeber et al. 2000).

Longitudinal studies help specify the temporal patterns among these comorbidity patterns. Early onset and high levels of disruptive behaviors in elementary school girls predicted CD in adolescence (Coté et al. 2001). Similarly, based on the 25-year longitudinal study of a New Zealand birth cohort, conduct problems at ages 7–9 years were associated with higher rates of substance dependence for males and females (Fergusson et al. 2005). Additionally, the MTFS showed that for boys and girls between ages 11 and 14, any symptom of ADHD or CD was a risk for substance use disorder at age 18 (Elkins et al. 2007).

Studies that emphasize girls’ comorbidity patterns show that CD increases later risk of anxiety disorder (Zoccolillo 1993), and among girls between ages 6 and 18 recruited from doctors’ offices, those with ADHD compared to those without ADHD are more likely to have depression, ODD, substance use disorder, and anxiety 5 years later (Biederman et al. 2006). The GSMS longitudinal survey of children from 9 to 16 years old showed that ODD is not associated with later CD in girls as it was for boys (Rowe et al. 2002). However, patterns among girls showed that there were strong associations between earlier ADHD and later ODD, and earlier CD and later substance abuse (Costello et al. 2003).

**Anxiety Disorders**

Aggregate anxiety disorder estimates from mixed gender samples of youth in community settings range from 2.4 to 17.7% (Ollendick et al. 2001). Another review of published studies of anxiety disorders in children below 12 years found a range of 2.6–41.2%, and separation anxiety disorder (SAD) appeared to be most prevalent (Cartwright-Hatton et al. 2006). The broad range is due in part to the types of anxiety disorders that a given study evaluated, and usually include some combination of SAD, generalized anxiety disorder (GAD), specific phobia, social phobia, panic disorder (PD), posttraumatic stress disorder (PTSD), and obsessive-compulsive disorder (OCD). Impairment criteria tend to have been applied in studies of anxiety disorder more than in studies of other types of mental disorder. For example, Kashani and Orvaschel (1988) reported an anxiety disorder prevalence of 17.3% without...
impairment criteria and 8.7% with it. Romano et al. (2001) showed that impairment criteria have the greatest impact on simple and social phobia estimates.

Across studies that examine specific anxiety disorders among youth, estimates are not consistently reported by age or gender. SAD has been estimated as the most prevalent anxiety disorder among children with rates between 3 and 5%, but only 0.01–2.4% among adolescents (Eisen et al. 2001). Rates of both social phobia (Sweeney and Rapee 2001) and PD have been estimated at about 1–2% of adolescents (Hayward and Essau 2001; Wittchen et al. 1998). These rates tend to be higher among adolescents than children and higher among adults than adolescents (Silverman and Carter 2006). Estimates of panic attacks are higher than those for PD, and they range more widely, leading to questions about the relationship between attacks and the course and severity of PD across childhood and through adolescence. Prevalence of OCD in adolescence has been reported between 3 and 4% (Douglas et al. 1995; Thomsen 2001; Valleni-Basile et al. 1994; Zohar 1999), whereas rates of specific phobias have been reported as high as 12.7% among 13–18-year olds (Essau et al. 2001). According to the review by Schechter and Tosyali (2001), the estimates of PTSD in community studies range from 1.2 to 6.3%, with much higher rates found in samples of children and adolescents who have been exposed to specific traumatic events such as witnessing violence, natural disasters, and physical or sexual abuse.

Few gender differences have been reported in rates of most anxiety disorders among children. Among adolescents, however, rates have been reported to be up to three times higher among females compared to males with regard to aggregated categories of anxiety symptoms and disorders (Anderson et al. 1987; Hayward and Essau 2001; Silverman and Carter 2006; Southam-Gerow 2001), as well as specific phobia (Milne et al. 1995) and social phobia (Wittchen et al. 1999). This pattern was confirmed in one of the few epidemiological studies to compare rates of mental disorder by gender and ethnicity: Hispanic females had significantly higher rates of anxiety than other females and than Hispanic males (McLaughlin et al. 2007). OCD presents an exception, with higher rates reported among boys and male adolescents than girls and female adolescents (Geller et al. 1998). Studies of gender differences in PTSD among adolescents in community samples have been inconsistent (Foy et al. 1996; Schechter and Tosyali 2001). However, with data from the National Survey of Adolescents (aged 12–17 years), Kilpatrick and colleagues (2003) estimated that the rate of PTSD was 6.3% for girls and 3.7% for boys; but for both girls and boys with PTSD, rates of comorbidity were high.

Longitudinal studies have documented that externalizing behaviors among youths (recruited between ages 4 and 16 years) were associated with anxiety disorders in adulthood (reinterviewed at ages 18 through 30; Roza et al. 2003), and anxiety disorders in childhood (before age 13) predicted a range of mental disorders in adolescence (age 13–19; Bittner et al. 2007). Based on retrospective reports from respondents of the National Epidemiological Survey on Alcohol and Related Conditions, anxiety disorder before age 19 increases the risk of bipolar disorder later in life (retrospective reports from Goldstein and Levitt 2007). The relationship between anxiety and depression has been studied quite extensively in longitudinal
Mood Disorders

Many symptoms of mood disorders, including bipolar disorder and depression, may begin in early childhood, although it is uncertain how many children could be considered to have a disorder. More certain is the high risk that childhood symptoms and diagnosis pose for depression throughout the life course, including adolescence (Costello et al. 2002; Roza et al. 2003), young adulthood (Dekker et al. 2007; Fergusson and Woodward 2002), and middle age (Clark et al. 2007).

Examinations of gender-specific patterns suggest that prior to adolescence there are differences in the types of symptoms experienced by boys and girls. In a study of public school children between ages 10 and 12, girls identified more internalizing symptoms and negative self-esteem, and boys identified more externalizing symptoms and more school problems (Bailey et al. 2007). Despite these different symptom patterns, the rate of depression does not appear to differ substantially between boys and girls prior to puberty. However, several studies have found that unipolar depression rates double or triple around the age of 13 for females but not for males (Angold et al. 1999b; Costello et al. 2002). For example, according to data from the Oregon Adolescent Depression Project (Lewinsohn et al. 1993), adolescent females had higher rates than males of major depressive disorder (25% vs. 12%) and dysthymic disorder (4% vs. 2%). One year later, the rate of depression among the females was 32%. When gender differences in nine symptoms were examined, the investigators found approximately 75% of the girls reported weight or appetite problems, and 82% reported feelings of worthlessness or guilt, both more prevalent than among boys with depression (Lewinsohn et al. 1998). Compared to information about depression, there is much less information on bipolar disorder in childhood, although studies show onset ages around 12 years of age, a prevalence of about 1% during adolescence (ages 14–18), and few gender differences (Costello et al. 2002).

A considerable amount of research has aimed to understand the mechanisms behind the substantial gender difference in depression that manifests around the age of puberty. Based on their analysis of data from females in the Virginia twin registry, Silberg et al. (2001) hypothesized that depression that onsets before age 14 may be an etiologically different disorder from depression that onsets after age 14. When male and female twins from that registry were compared, negative life events had a significant effect on depression for both boys and girls, but a strong genetic component was identified only for female adolescent-onset depression (Silber et al. 1999). Experiences of negative life events during adolescence are potentially important risk factors for males and females, but possibly only in conjunction with other genetic risks or the timing of hormonal changes (Cyranowski et al. 2000). Moreover, the importance of early identification of biologic or physiologic risks
has been highlighted by studies of the long-term consequences of LBW. Costello et al. (2007) found that LBW was associated with depression but not other diagnoses, in adolescence but not in childhood, and among girls but not boys. By age 16, girls’ rates of depression were 38.1% and 8.4% given LBW and normal birth weight, respectively.

Comorbidity among youths with depression is high. It is estimated that between one-third and three-quarters of children and adolescents with depression have an additional disorder (Lewinsohn et al. 1993). The patterns of co-occurring disorder vary with age; ADHD and CD are prevalent among children with depression, and eating disorders and substance abuse are prevalent among adolescents with depression.

According to one review, between one-quarter and one-half of youth with depression have a comorbid anxiety disorder, and approximately one-tenth of youth with anxiety have depression (Axelson and Birmaher 2001). In an adolescent community sample, adolescent girls had higher rates of co-occurring anxiety and depressive disorders than boys, while boys had higher rates of co-occurring ADHD and CD (Romano et al. 2005). Studies that examine the relative timing of the onset ages of these disorders appear to assume similar patterns for males and females, with predictive models including controls for gender rather than separate analyses being run for males and females, or interactions tested between gender and other risk factors.

There is compelling evidence for a relationship between prior anxiety or anxiety symptoms and later onset of mood disorders. Longitudinal community studies in the USA (Avenevoli et al. 2001) and other countries indicate that among youth who have both anxiety and mood disorders, the anxiety disorders typically onset first (Beesdo et al. 2007; Reinherz et al. 2003; Roza et al. 2003). Moreover, in models predicting risk, adolescents (ages 13–14) who reported a greater number of fears had a higher risk of depression when they were in their early twenties compared to adolescents with fewer fears (Pine et al. 2001), and adolescents with anxiety disorders were at increased risk of developing bipolar symptoms in early adulthood (Johnson et al. 2000) as well as depression (Beesdo et al. 2007).

Some nuances to the relationship between anxiety and depression have been identified in other longitudinal studies. Specifically, depression at age 14 increased OCD risk at age 18 (Douglas et al. 1995). Both the GSMS and the Dunedin study suggest that there may be an equally strong relationship between prior depression and later anxiety (Costello et al. 2003; Moffitt et al. 2007).

**Eating Disorders**

Estimates based on community samples indicate that rates of eating disorders are low relative to the other disorders presented in this chapter. Prevalence rates of anorexia nervosa, bulimia nervosa, and binge eating disorder were 0.04%, 0.3%, and 1.9%, respectively, in a study of middle and high school aged female students.
Reports of eating disturbances were as high as 14.7% in another study of girls between ages 9 and 13, but most were of mild severity (Colton et al. 2007). Despite the low prevalence, mortality rates associated with eating disorders overall and anorexia in particular are higher than those associated with most other mental disorders, according to a review of research in this area (Striegel-Moore and Bulik 2007).

It is suspected that most eating disturbances are resolved after adolescence. However, few longitudinal studies include measures of eating disturbances, and few span the peak period of disorder onset (Keel et al. 2007). Retrospectively reported data from the NCS-R suggest that eating disorders onset between the ages of 19 and 25 (Hudson et al. 2007). Thus, developmental trajectories of problematic eating behaviors and their association with eating disorders in adults have not yet been well-specified.

Arguments have been made for the development of better measurement instruments as additional research develops. Diagnostic classifications may be useful in clinical studies but there is concern about their low specificity and positive predictive value in epidemiologic studies (Ackard et al. 2007; Colton et al. 2007). Moreover, concerns about cultural differences in the presentation of eating disorder symptoms have been identified (Striegel-Moore et al. 2005), along with the need to evaluate eating disorder criteria that may be more appropriate for Latinas (Alegria et al. 2007) and African Americans (Taylor et al. 2007).

There is limited epidemiologic data on comorbidity between eating disorders and other mental disorders. Youth with eating disorders tend to have higher than expected rates of anxiety (Swinbourne and Touyz 2007), especially OCD and social phobia (Kaye et al. 2004), depression and substance use disorders (Baker et al. 2007), and bipolar disorder (McElroy et al. 2005). Studies of the temporal ordering of eating disorder onset ages relative to other diagnoses have not yielded consistent results. For additional information on eating disorders, please see Chap. 7 in this volume.

Implications for Women’s Mental Health

To date, female-specific studies appear to have been conducted on an ad hoc rather than systematic basis. On the basis of this chapter presentation, it is recommended that future research attend to girls and female adolescents in a deliberate way. For example, as developmental trajectories, built upon existing data, are used to specify the genetically complex and heterogeneous pathways that seem to be operant, analyses should be run separately for both genders, or theoretically relevant interactions should be tested (i.e., history of childhood maltreatment and gender). Assessments in longitudinal studies, whether samples comprised both genders or only one, should be as frequent as possible to more accurately capture exposure to and timing of risk factors across different age periods, when vulnerability and resilience may be most salient. Moreover, while highlighting the unique influence of gender, it is important
to consider that there are likely to be racial–ethnic differences in prevalence and the mechanisms that explain the development of mental disorders. Cultural influences are particularly important for the definitions of the degree and type of symptoms that are considered “abnormal” and the way gender influences those definitions. As Silverman and Carter (2006) observed with regard to anxiety disorders, very limited research has examined the influence of gender across different age periods and for different ethnic and sociodemographic groups.

From a public health perspective, one should not lose sight of social stratification and community level influences upon mental health, and, in fact, this is a potentially critical area for future research. Without consideration of these influences, the range of potential interventions may be increasingly limited to pharmacological treatments. Several recent studies argue for a balance of attention to individual and environmental influences. First, in a study of families and work that included data on children from birth to third grade, socioeconomic status moderated the effect of child and family risks on symptom severity (Essex et al. 2006). Second, a comparison of adolescents (aged 11–17) representing different ethnic groups (i.e., European Americans, African Americans, and Mexican Americans) showed that economic disadvantage rather than ethnic status was a factor in disorder prevalence (Roberts et al. 2006). Third, neighborhood differences accounted for differences in internalizing behavior problem scores in a longitudinal study of children (aged 5–11 years) recruited from 80 neighborhoods in Chicago (Xue et al. 2005).

The last several years have provided advances in knowledge about the epidemiology and etiology of mental illness, particularly regarding the developmental sequencing of symptoms and disorders across childhood and adolescence. Together with sophisticated methods and analytic approaches, these advances hold substantial promise for the development of interventions that may alter illness trajectories, either by early identification and prevention of the onset of mental disorder or by minimizing its severity and chronicity. As the research develops further, issues of particular importance for girls and female adolescents warrant specific attention. Otherwise, interventions will be developed based on findings that to a great extent are gender-neutral or biased toward the experiences of boys and male adolescents, potentially compromising their effectiveness.

References


