# Does Bipolar Disorder Exist in Children? A Selected Review

**Anne Duffy, MD, FRCPC**[1]

Although there is increasing recognition that a substantial proportion of patients with bipolar disorder (BD) experience an onset of illness in adolescence, significant controversy remains over the validity of the diagnosis in very young children. In careful studies of adult patients dating from Kraepelin, first mood episodes not uncommonly occurred during adolescence. Some of these early-onset patients experienced subthreshold mood disturbances or predisposing temperaments earlier in childhood. Earlier onsets of BD have been reported in more recent clinical and community samples of children. Several factors possibly contributed to these earlier onsets, including exposure to psychotropics, bias in favour of a mood rather than a psychotic diagnosis, and recognition of softer-spectrum BDs. However, the validity of the diagnosis of BD in impulsive, irritable, labile, or behaviourally dysregulated children remains to be proven. Studies of high-risk children of well-characterized parents with BD have demonstrated that BD most often debuts as a depressive episode in mid to late adolescence and that activated episodes are rare prior to age 12 years. Some children manifest antecedent nonspecific psychopathology in early childhood. Therefore, as currently diagnosed, BD does not manifest as such typically until at least adolescence.

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**Highlights**

- BD, as currently diagnosed, not uncommonly has its onset during adolescence, typically debuting as a depressive episode.
- Although there may be nonspecific psychopathology or subthreshold mood disturbances early in the course, BD is rarely diagnosable in young children.
- For accurate diagnosis of BD early in the course, family history and clinical course need to be taken into account.

***Key Words**: bipolar disorder, children, early onset, diagnosis, clinical course, family history, review*

Over the past 2 decades, there has been a growing interest in BDs in general and in the early-onset subgroup in particular. This has been reflected in the number of clinical treatment trials for the various stages of the disorder, in the number of publications dedicated to BD, and in the program content of various national and international psychiatric meetings. The interest makes sense, given the relatively high and increasing prevalence of childhood psychiatric disorders[1,2] and the fact that effective treatments for BDs exist.[3–9]

Although most clinicians and researchers would agree that a substantial proportion of BD onsets occur during adolescence, whether BD manifests in young children remains controversial. In this paper, I argue that, while ill children of parents with BD show nonspecific symptoms such as anxiety, mood lability, and sleep disturbance quite early in development, BD meeting DSM-IV criteria typically does not manifest until at least adolescence. My argument will incorporate several lines of evidence, including studies of the natural

course in BD patients, longitudinal studies of children with mood disorders, and studies of the offspring of parents with BD.

## Adult Studies

### *The Natural Course of BD*

From his early systematic observations of over 900 patients suffering from "manic-depressive insanity," Kraepelin reported that "the greatest frequency of first attacks falls, however, in the period of development with its increased emotional excitability between the fifteenth and the twentieth year."[10, p 167–168] He also noted that, in the next decade, from the ages of 20 to 30 years, the frequency of first attacks remained high. In Kraepelin's sample of patients, manic-depressive attacks prior to age 10 years were rare and, if present, mild in nature. Kraepelin also observed an overrepresentation of specific temperaments in a certain proportion of patients and in family members that in some cases formed the rudimentary basis for the manifestation of full-blown manic-depressive illness and in other cases remained a "peculiarity of emotional life" or temperamental style. Kraepelin found that these temperaments were observable over the course of development through childhood and adolescence and described them as depressive, manic, irritable, and cyclothymic.

These findings have been replicated in the longitudinal prospective study[11,12] led by Angst, which described the natural course of illness in a cohort of hospitalized patients with unipolar and bipolar disorder diagnosed in accordance with DSM-III criteria. The initial observations from Angst and colleagues' multicentre, international study reported the median age of onset of BD to be 30 years, with a peak in the onset-age distribution occurring between the ages of 20 and 29 years.[11,12] Subsequent reports focusing on the Zurich-only cohort have found a median age of onset of 29 years[13] and a 10-year difference in median age of onset between BD patients with and without mood-incongruent psychotic features (age 25 years and age 35 years, respectively). In a follow-up report on this cohort,[14] Angst compared schizoaffective patients with mania and BD, demonstrating that the patients suffering from schizoaffective disorder and mania had an earlier median age of onset (age 26 years, compared with age 33 years for patients with schizoaffective disorder and BD). Angst highlighted the observation that the first manifestations of illness not uncommonly occurred during adolescence, with the earliest onset in this sample being age 13 years. These early mood disturbances had often been misdiagnosed as reactive depressions, neuroses, or personality disorders.

Pooling data from a large number of studies, Goodwin and Jamison[15] found support for the findings of Kraepelin and Angst, reporting that the weighted mean age of BD onset was 28 years and the median age was in the mid-20s. Manic episodes were found to be rare or nonexistent prior to age 13 years. As they and others[16] point out, definintion of the age of onset varies depending on the onset criteria (for example, hospitalization, treatment, or index episode). In their estimate, when age of onset is taken as the first diagnosable mood episode, the distribution replicates Kraepelin's observations: a peak distribution occurs between the ages of 15 and 19 years, closely followed by a peak of onsets between the ages of 20 and 24 years.

### *Shifts in Estimated Rates and Diagnoses*

Epidemiologic trends have documented a progressive decrease in the age of onset of both unipolar and bipolar disorders with successive birth cohorts as well as a period effect associated with the 1970s.[17–19] At the same time, there has been a well-documented diagnostic shift favouring BD over schizophrenia.[20,21] More recently, Angst[22,23] has reported the relatively high prevalence of a broader spectrum of BDs diagnosed according to varying definitions of hypomanic and depressive episodes. The net result of these factors has been a decrease in the estimated age of onset of BDs, favouring the adolescent years over later decades. For example, in the ECA study,[24] the median age of onset of BD I was 19 years. In a recalculation of the ECA data, the hazard rates for onset of mania were highest between the ages of 15 and 19 years for both sexes.[25]

In an epidemiologic sample of BD patients, Leboyer and colleagues[26,27] identified 3 subgroups on the basis of distribution of the ages of onset (defined as first mood episode). Onset-ages for these subgroups peaked at 17, 27, and 46 years and accounted for an estimated proportion of 28%, 50%, and 22% of patients, respectively. Adolescent-onset BD has been associated with higher rates of comorbid nonaffective disorders,[28,29] more frequent psychotic features,[30–33] and more mixed states.[34,35] However, other studies have concluded that there are more similarities than differences between adolescent-onset and adult-onset mania, including aspects of clinical course and outcome.[36–40] One consistent finding has been a higher risk for mood disorders among the relatives of

**Abbreviations used in this article**

| | |
|---|---|
| ADHD | attention-deficit hyperactivity disorder |
| BD | bipolar disorder |
| ECA | Epidemiologic Catchment Area |
| SD | standard deviation |

early-onset probands.[41–43] Strong genetic factors are thought to underlie the increased familial transmission.[29,44]

Finally, there has been increased interest and research into the softer-spectrum BDs conceptualized along the lines of Kraepelin's predisposing affective temperaments.[23,45–47] Specifically, Akiskal has discussed the association between cyclic depressive disorders and temperamental disturbances and BD, especially in those at familial risk. Accumulating evidence suggests that "subaffective" disturbances (for example, dysthymia, cyclothymia, and hyperthymic and depressive temperaments) in childhood and major depressive episodes in adolescence represent reliable predictors of BD,[45,48,49] especially if there is a positive family history of BD and (or) psychotic features.[50,51]

***Summary***

From the earliest systematic observations of Kraepelin to more modern studies of the natural course of illness led by Angst, the overriding conclusion is that full-blown BD does not manifest as such until at least the adolescent years. This observation has held despite factors acting to decrease the estimated age of onset, including epidemiologic trends, diagnostic shifts, and broader-spectrum diagnostic criteria. However, there are several independent research groups reporting childhood prodromal symptoms in some BD patients that include characteristic temperamental styles and episodic subthreshold mood disturbances. The most robust risk factor remains a positive family history, and children at risk warrant close follow-up if they become symptomatic.

## Child and Adolescent Studies

***Follow-Up Studies of Clinical Samples***

In a study of a large community cohort of 1709 adolescents interviewed on 2 occasions over an average 1-year interval, Lewinsohn and colleagues[52] identified a total of 18 meeting lifetime criteria for BD, yielding a lifetime prevalence of about 1% and a 1-year incidence rate of 0.13%. Further, 5.7% of the adolescent cohort met criteria for lifetime manic symptoms that fell short of diagnostic criteria for BD. Most adolescents diagnosed with a BD met criteria for cyclothymia or BD II. None had been hospitalized, and only one had received treatment with lithium. The mean (SD) age of onset of the first mood disturbance for the 18 cases of BD was 11.75 (2.96) years; this did not differ between the sexes. The age of onset distribution in this sample peaked at age 14 years for both male and female adolescents.[53] Most of the subjects with BD began their illness with either cyclothymia or a minor or major depressive episode. BD and bipolar symptoms were associated with increased risk of affective disorders in first-degree relatives, higher rates of comorbidity, significant suicidal behaviour, and substantial impairment in social, family, and school functioning, compared with subjects who had never suffered from mental illness.

In their longitudinal study (with an average length of follow-up of 6 years) of clinically referred children aged 8 to 14 years, Kovacs and colleagues[54,55] reported that childhood dysthymia and major depressive disorder were antecedents to subsequent recurrent affective disorders. When children with an initial diagnosis of dysthymia were compared with those having major depression, it was found that the dysthymic children tended to have an earlier age of onset. The vast majority of children with dysthymia went on to develop an index episode of major depression—76% over the follow-up period and an estimated 81% if all had been followed for a 9-year period. By the end of the follow-up period (when the subjects' mean age was 18 years and the age range was 12.5 to 24 years) 13% of the group with dysthymia and 15% of the group with depression met criteria for BD.

Latent bipolarity in subjects suffering from depression is much more common than previously thought and likely explains a substantial proportion of treatment-refractory depressive disorders.[22,56] In adults, switch rates have been estimated to be in the order of 10% to 15% in some follow-up studies,[14,48,49] or 1% to 2% yearly.[13] In follow-up studies of children suffering from depression, the switch rate estimates are higher for comparable study follow-up periods.[54,57.58] Predictors of latent bipolarity in children with depression include a positive family history of BD; clinical features that include mood-congruent psychosis, rapid onset, and psychomotor retardation; and pharmacologically induced hypomania.[59]

Several reports have described atypical mania-like symptoms in young children.[60–64] Geller and colleagues published the first prospective follow-up study of prepubertal children diagnosed with mania.[63] The baseline intake episode of mania represented the first manic episode for over 80% of the sample. Mean (SD) age of onset for the entire sample was 6.9 (3.5) years. The course was one of recovery (87%) and relapse (70%). Subjects spent over two-thirds of the follow-up period ill; mostly with either mania or hypomania. In 88% of this sample, the course was described as mixed mania and rapid cycling. Of these BD subjects, 86% were diagnosed with comorbid ADHD and 59% had psychotic features. The proportion of the sample that had prior or concurrent treatment with stimulants or antidepressants was not noted. The possibility of unmasking latent BD earlier with psychotropics has been a topic under discussion.[65–67]

Researchers describing samples of chronically ill very young children with psychotic features, agitation, irritability, labile mood, impulse-control difficulties, cognitive deficits, and major impairment in functioning have concluded that these children exhibit a subform of mania, or pediatric

mania.[34,62–64,68,69] The manic diagnostic decision appears heavily weighted on persistent and pervasive irritability and chronic overactivity. It is difficult to interpret what these children actually suffer from. As discussed by Robins and Guze[70] and others,[71,72] to validate a psychiatric diagnosis one must prove that there is a consistent clinical description, a characteristic clinical outcome in long-term follow-up, increased prevalence of the same disorder in relatives, reliable associated laboratory findings, and a clear delimitation from other disorders. Therefore, whether these very ill young children suffer from BD or from some other psychiatric illness is not known at this time. The uncertainty is compounded by reports demonstrating a lack of continuity between prepubertal depression (but not adolescent depression) and adult affective disorders[73] and, in prospective follow-up studies,[74,75] a lack of association between childhood-onset ADHD and later affective disorders.[76,77]

### *Summary*

Epidemiologic and clinical studies have confirmed that dysthymia and minor depressive episodes meeting DSM criteria do occur in childhood, with little variation from the adult syndromes. These subaffective disturbances are usually followed by major depressive episodes. A substantial proportion of the adolescents manifesting depressive disorders appear to be suffering from latent BD. However, the hypomanic or manic episodes usually do not manifest until at least mid adolescence. It may be that, in reported cases of childhood mania, early exposure to stimulants and (or) antidepressants unmasks latent bipolarity and modifies its course and presentation. At this stage, however, the existence of BD in very young children, or pediatric BD, remains to be validated. What we do know is that very young children exhibiting chronic mania-like symptoms represent a significantly disadvantaged group with complex and enduring psychopathology—the true diagnosis and natural course of which remain to be determined.

## High-Risk Offspring Studies

### *Cross-Sectional Studies*

As mentioned, the most robust risk factor predicting the development of BD is a positive family history,[78] which reflects a strong genetic contribution to BDs.[79] Therefore, prospective longitudinal studies of the offspring of parents with confirmed BD is an important, and arguably the best, approach to studying the onset and natural course of the disorders and the role of possible modifying factors.

Several studies detailing the rate and nature of psychopathology among the offspring of parents with BD have been reported and reviewed.[80–82] Most of these studies are cross-sectional. Without exception, all have reported an increase in psychopathology in general, and mood disorders in particular, among the offspring of parents with BD. The age distribution of offspring and the rates of comorbid nonaffective disorders have varied significantly among studies. For example, in the study reported by Chang et al,[83] the mean age of the cohort was 11 years, 88% of the children diagnosed with BD also had a diagnosis of comorbid ADHD, and 8 of 9 were boys. By contrast, in the study reported by Reichart et al,[84–86] the subjects had a mean age of 16 years at baseline, 7 of 13 offspring diagnosed with BD were male, and only one of these had a diagnosis of comorbid ADHD. The differences between high-risk studies in age of onset and nature of the psychopathology can in part be attributed to differences in parental samples (for example, assortative mating and heterogeneity of BD), differential use and application of diagnostic assessments, and possibly, differential exposure to psychotropics.

### *Longitudinal Prospective Studies*

High-risk studies with a longitudinal component offer the advantage of prospectively mapping the evolution of psychopathology. Reliable descriptions of prodromal symptoms and the early course of BD are paramount to accurate diagnosis and effective early intervention. However, there are few longitudinal prospective high-risk studies. There are 3 comparable studies with standardized methodology, well-characterized parent samples, and repeated assessments of offspring over a 5- to 10-year period.[84,87,88] In all these studies, the families included were mostly intact, had limited or no assortative mating, existed in the context of relatively organized and stable communities, and had offspring with little or no exposure to stimulants or antidepressants.

In both the Dutch and Canadian studies, BD I or II almost exclusively began with a depressive episode (that is, dysthymia, depression not otherwise specified, or major depression), usually occurring in mid adolescence. In the Dutch study, 12 of 13 youths with BD began with depression, with the median age of onset of the first mood episode being 12 years (range 9 to 22 years). By comparison, in the Canadian sample, 13 of 16 youths with BD began with a depressive disturbance; the median age of onset of the first mood episode was 15.5 years (range 10 to 22 years). In both samples, several years elapsed between the onset of the depressive disorder and the first hypomanic or manic episode: in the Dutch cohort, there was a 4-year median interval (range 0 to 9 years), and in the Canadian cohort, there was a 2-year median interval (range 0 to 10 years). In both studies, the median age of onset of the first hypomanic or manic episode was the same at 18 years. No offspring meeting DSM criteria for BD I or II in either study did so prior to age 12 years. Table 1 is modelled on a similar table reported by the Dutch research group[84] and shows onset details for the Canadian cohort. In the Canadian

**Table 1 Characteristics of 16 high-risk offspring meeting lifetime DSM-IV criteria for BD I or II[a]**

| Sex | Affected parent | Familial lithium response | Index mood episode | Age of onset of index mood episode | Age of onset of first (hypo) mania[b] | BD I or II | Antidepressants[c] |
|---|---|---|---|---|---|---|---|
| Male | Father | LiR | Major depression | 16 | 18 | BD II | No |
| Female | Mother | LiR | Depression not otherwise specified | 13 | 15 | BD II | No |
| Female | Father | LiR | Depression not otherwise specified | 15 | 21 | BD I | No |
| Female | Father | LiR | Depression not otherwise specified | 17 | 18 | BD II | No |
| Female | Father | LiR | Depression not otherwise specified | 16 | 16 | BD II | No |
| Female | Mother | LiR | Depression not otherwise specified | 12 | 20 | BD II | No |
| Female | Father | LiR | Mania | 22 | 22 | BD I | No |
| Female | Mother | LiR | Major depression | 16 | 17 | BD II | Yes |
| Male | Father | LiR | Major depression | 13 | 23 | BD II | No |
| Female | Mother | LiR | Major depression | 10 | 17 | BD II | No |
| Male | Father | LiR | Major depression | 15 | 18 | BD II | No |
| Male | Mother | LiNR | Hypomania | 18 | 18 | BD II | No |
| Female | Mother | LiNR | Major depression | 15 | 16 | BD II | Yes |
| Female | Father | LiNR | Major depression | 18 | 20 | BD I | Yes |
| Female | Mother | LiNR | Major depression | 19 | 20 | BD II | No |
| Male | Father | LiNR | Hypomania | 12 | 12 | BD II | No |

[a]Data from[88,89,103]

[b]No stimulants were used before first hypomanic or manic episode

[c]Use of antidepressants before first hypomanic or manic episode

LiR = Lithium responders, LiNR = Lithium nonresponders

study, nonspecific disorders or disorders other than mood disorders (that is, sleep, anxiety, and in some subjects, neurodevelopmental disorders) were antecedents to the onset of subsequent BD in high-risk children.[89]

In the 10-year prospective follow-up study of the offspring of Amish parents with BD I,[87,90] prodromal symptoms indicating risk for BD included anxiety or somatic complaints, distractibility or role impairment in school, excitability, hyperalertness, and mood lability. As the children grew older, more classic bipolar symptoms became significant; these included talking loudly, excessive talking, high energy, problems with thinking and concentration, and problems with sleep (that is, decreased sleep, early awakening, and initial insomnia). These symptoms tended to occur episodically. In this high-risk cohort, as in the Dutch and Canadian studies, there were no observations of diagnosable hypomania or mania below age 12 years. Further, there were no observations of chronic irritability, aggression, or continuous cycling consonant with the description of atypical pediatric mania.

### *Summary*

Although several studies have reported an elevation in psychopathology among the offspring of parents with BD, the nature and age of onset of the disorders have varied among studies, in part reflecting differences in parent samples, in methodology, and possibly, in prior treatment exposure. In addition, nonspecific symptoms and syndromes appear to characterize the early course of mood disorders in some high-risk children. In longitudinal studies of the offspring of parents with well-characterized BD, there have been no observations of diagnosable BD in children under the age of 12 years. However, there have been reports of early prodromal symptoms in these high-risk children, including anxiety, mood lability, sleep disturbances, and cognitive difficulties.

**Table 2a Course of illness in affected offspring**

| | Familial lithium response | |
|---|---|---|
| | Responders | Nonresponders |
| Clinical course | *n* (%) | *n* (%) |
| Episodic | 25 (96.2) | 3 (11.1) |
| Nonepisodic | 1 (3.8) | 24 (88.9) |

$\chi^2$ = 38.44, df 1, $P$ < 0.001

**Table 2b Course of illness between affected parent and affected offspring**

| | Parents' course | |
|---|---|---|
| | Episodic[a] | Nonepisodic[b] |
| Clinical course | *n* | *n* |
| Episodic | 25 | 4 |
| Nonepisodic | 0 | 24 |

[a]Episodic course with full remission

[b]Chronic, chronic fluctuating, or episodic with residual symptoms

$\chi^2$= 39.163, df 1, $P$ < 0.001

## Heterogeneity of BD

Heterogeneity of BD has not been taken into account in follow-up studies of clinically referred or high-risk children. Nevertheless, there is clear evidence that BD represents several different, possibly overlapping, spectra or subgroups of disorders.[8,91,92] There have been various approaches to defining more homogeneous subgroups to understand the natural course of illness, treatment response, and underlying pathophysiology. One approach to subgrouping has been based on age of onset, as already discussed[27]; another has been based on differential clinical course.[23,93,94] Our research group has focused on response to long-term mood-stabilizing treatments[8,95–101] as a way to identify more homogeneous BD subgroups.[92,102,103] Briefly, lithium responders tend to have a classic episodic remitting course, typical episodes with no or mood-congruent psychotic features, and relatives with recurrent mood disorders. By contrast, lithium nonresponders tend to have mood-incongruent psychotic features in episodes, incomplete remissions, and relatives with chronic psychiatric disorders, including schizophrenia.

In our high-risk study, we have been comparing and contrasting the offspring of 2 subgroups of parents with BD, divided on the basis of a clear response or nonresponse to long-term lithium monotherapy.[88,104,105] We have found that the offspring of lithium responders tend to develop mood disorders with an episodic and completely remitting but recurring course, whereas the offspring of lithium nonresponders tend to develop chronic or partially remitting mood disorders (Table 2a). The most striking finding has been that, although the nature of the presenting psychopathology has been varied in both high-risk groups (and included sleep and anxiety disorders and, in nonresponders, ADHD and cluster A traits), the clinical course has bred true from parent to child (Table 2b). The premorbid childhood functioning among the lithium responders was normal or gifted and did not predict which offspring would become ill, whereas, among the lithium nonresponders, those who developed mood disorders had prodromal childhood problems with functioning in school, with friendships, and with fluctuating mood and anxiety symptoms. It appears that the bipolar offspring of lithium nonresponders experience an earlier onset of complex psychopathology that follows a chronic fluctuating course. Other groups have reported an overrepresentation of similar neurodevelopmental antecedents in a subgroup having BD with very early onset.[106]

### *Summary*

Heterogeneity of BDs represents a major confounding factor not taken into account in clinical and high-risk studies of children. To a certain extent, heterogeneity may explain discrepancies in reports of the clinical course and psychopathological outcomes in children at risk for and (or) manifesting mood disorders. Specifically, in our studies, the offspring of lithium responders appear to represent a classic Kraepelian subgroup of BD patients, whereas the offspring of lithium nonresponders appear to represent a more atypical chronically ill subgroup. In both groups, it appears that presenting psychopathology and prodromal symptoms are nonspecific and that the best predictors of the likelihood and specific subtype of BD in children at risk are the family history (that is, the loading and spectra of disorders segregating in the family) and the clinical course (whether episodic or chronic).

## Closing Remarks

From the literature reviewed, I conclude the following:

- There is a lack of supporting evidence for the hypothesis that BD, as currently defined, exists in very young children. Clearly, in some cases, there are prodromal psychiatric disturbances in early childhood antecedent to the manifestation of recognizable BD.
- BD often starts in adolescence with an index episode of major depression. As described above, in some patients, there may be earlier, nonspecific prodromal symptoms, including anxiety and sleep and cognitive disturbances.

- Chronic fluctuating abnormalities of mood, overactivity, and cognition and conduct disturbances have been described in very young children. Whether this syndrome represents an early variant of BD or some other psychiatric disturbance is at this time unknown and requires further research.
- In the early stages of BD, the presenting symptoms are nonspecific and not limited to the mood spectrum. Therefore, it appears that the risk of BD is best predicted, not by the presenting psychopathology but by the clinical course and family history.

These conclusions may seem counter to the prevailing North American view that BDs do occur in very young children. The difference in conclusions reached here may reflect the fact that they were based on an integration of evidence not only available from investigations of children but also available from research on adults with BD and their offspring. As well, I have raised the point that heterogeneity, assortative mating, and early exposure to psychotropics may explain some observations of mania-like symptoms in very young children. The main point I would like to make is that accurate early identification of BD in youth must not rely on symptoms only. Symptoms are inadequate in the early stages of illness, and we must incorporate in our diagnostic decisions the clinical course and the family history.

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[1]Canada Research Chair, Child Mood Disorders, and Associate Professor, Department of Psychiatry, McGill University, Montreal Children's Hospital, Montreal, Quebec.
*Address for correspondence:* Dr A Duffy, Department of Psychiatry, McGill University, 4018 Ste-Catherine Street West, Montreal, QC H3Z 1P2; anne.duffy@mcgill.ca

**Résumé : Le trouble bipolaire existe-t-il chez les enfants? Une revue choisie**

Bien que l'on reconnaisse de plus en plus qu'une proportion substantielle de patients souffrant de trouble bipolaire (TB) voit apparaître la maladie à l'adolescence, une importante controverse subsiste à propos de la validité du diagnostic chez de très jeunes enfants. Dans des études rigoureuses de patients adultes, datant de Kraepelin, il n'était pas rare que les premiers épisodes d'humeur se manifestent à l'adolescence. Certains de ces patients à l'apparition précoce éprouvaient des perturbations de l'humeur infraliminaires ou avaient des tempéraments qui les prédisposaient plus tôt dans l'enfance. L'apparition précoce du TB a été rapportée dans des échantillons d'enfants cliniques et communautaires plus récents. Plusieurs facteurs ont pu contribuer à ces débuts précoces, dont l'exposition aux psychotropes, le biais en faveur d'un diagnostic d'humeur plutôt que de psychose, et la reconnaissance d'un spectre plus bénin des TB. Cependant, la validité du diagnostic de TB chez les enfants impulsifs, irritables, instables ou dont le comportement est déréglé reste à prouver. Les études d'enfants à risque élevé de parents bien caractérisés souffrant de TB ont démontré que le TB débute souvent par un épisode dépressif au milieu ou à la fin de l'adolescence, et que les épisodes activés sont rares avant l'âge de 12 ans. Certains enfants manifestent une psychopathologie non spécifique antécédente au début de l'enfance. Par conséquent, comme on le diagnostique actuellement, le TB ne se manifeste pas typiquement comme tel avant au moins l'adolescence.