Introduction

Until four or five decades ago, the clinical consensus about childhood depression was that it did not, and perhaps could not, occur (Lefkowitz & Burton, 1978; Rie, 1966; Rochlin, 1965; Wolfenstein, 1966). In fact, prior to 1960, childhood depression was rarely mentioned in the literature (Cytryn, 2003; Tisher, 2007). A number of studies and conceptualizations have since reversed this view (Burks & Harrison, 1962; Cytryn, Cytryn, & Rieger, 1967; Glaser, 1967; Kovacs & Beck, 1977; Sandler & Jaffe, 1965; Toolan, 1962). Notably, though, some authors have conceded that childhood depression was often not directly observable. Glaser (1967) and Lesse (1974) argued that depression in childhood was present, but masked by behaviours such as oppositional behaviour, aggressiveness, and bed-wetting, which were sometimes referred to as “depressive equivalents” (also see Cytryn & McNew, 1972; Toolan, 1962). Currently, the existence of childhood depression is widely accepted (Tisher, 2007).

Since this evolution in the thinking about childhood affective disorder, a number of assessment instruments have been developed to test for childhood depression. For example, the Children’s Depressive Rating Scale (CDRS, an instrument to be used by clinicians; Poznanski, Cook, & Carroll, 1979), the Children’s Depression Scale (CDS, self- or parent report; Tisher & Lang, 1983), and the Children’s Depression Inventory (CDI, also self-report; Kovacs, 1992). Perhaps the most widely used is the CDI, which was developed as a downward extension of the well-known test for adults, the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

It seems then, that depression has been firmly located in childhood. However, there are a number of weaknesses in the underpinnings of the arguments supporting this contention.

Indicators of Depression

Many of the behaviours that are taken to be indicative of depression are not examples of depressive behaviour. An appropriate example of this notion is the set of questions that make up the CDI (Kovacs, 1992); the most widely used childhood depression test reported in the literature (Fristad, Emery, & Beck, 1997; Rivera, Bernal, & Rosselló, 2005). Table 1 shows the key phrases extracted from each item of the CDI. Clearly, many of the specific behaviours could just as easily be assigned to other conditions such as anxiety, oppositional or antisocial disorder, hopelessness, or low self-esteem. Furthermore, the above-noted arguments on masked depression and depressive equivalents are suppositions that are meant to explain why we often do not observe “depression” directly. These arguments require an assumption that the underlying condition is a polythetic construct that is not defined by a single criterion. Kovacs and Beck are among those who have adopted this view. That is, that depression is seen as a syndrome, not a specific behaviour (Kovacs & Beck, 1977). In their words:

*The distinction must be made firmly between depression as a sad, despondent mood, and depression as a clinical syndrome (a collection of symptoms). Our past experience with adult depressives consistently indicated that depression as psychopathology is definitely a syndrome, and not just a dysphoric mood state.* (p. 1)

This kind of diagnosis thus depends on a theoretical framework which, of course, might be wrong. It should be noted that a child could, as a consequence, be assigned the diagnosis of depression without displaying any deep sadness, loss of interest, or dulled affect. This notion was formalized...
in the above-noted constructs of “masked depression” and “depressive equivalents”. Gittleman-Klein (1977) has been critical of this approach:

_These behaviour patterns, labelled “masked depressions” or “depressive equivalents,” are construed to be markers for an underlying depressive disorder that is interpreted to account for a multitude of behaviour problems, even though depression itself is not clearly manifest. There is no compelling logic to the above view._ (p. 71)

To test a point closely related to Gittleman-Klein’s (1977) view, Seifer et al. (Seifer, Nurcombe, Scioli, & Grapentine, 1989) factor analyzed the Child Behavior Checklist (Achenbach & Edelbrock, 1983) and, although they were able to identify a multiple-disorder entity, they were not able to isolate a categorically distinct group of children who displayed depression in the absence of other disorders – although they could for adolescents. The authors concluded that the results did not support the existence of a depressive disorder in children.

The point to be made here is that we seem to be confusing a behavioural description of depression with theories of either its underlying cause or of its external behavioural expression. This is not to say that these explanations of childhood depression are wrong, but rather that there is ample room for other explanations of troubled behaviour among children.

**Normative Test Construction**

The fact that there have been many tests developed to assess childhood depression provides a form of face validity. But the real issue is whether a particular test identifies people who actually exhibit the condition in question. That is, how does the test compare with the “gold standard”? In the case of the advent of the assessment of depression in children a few decades ago, there was no accepted construct of childhood depression at the time, and therefore no gold standard. Furthermore, it is not clear that there is a gold standard now – although one could argue that the CDI has itself come to be seen as assuming that role.

Proper test development involves a sophisticated series of steps that involve most or all of the following: item selection, item analysis, factor analysis, development of norms, assessment of reliability, and correlations with other measures. Taken together, the description of these would fill a substantial manual and could look quite impressive. However, a list of outputs like this does not indicate validity — no matter how well its component tasks are executed or presented.

It is important to note here that the CDI is a good test. It has been given an “A” rating by the California Evidence-Based Clearinghouse for Child Welfare (2009), has been translated into at least 20 languages (National Center on Child Abuse Prevention Research, 2005), shows good reliability (e.g. Finch, Saylor, Edwards, & McIntosh, 1987), and, as just noted, is the most frequently used child-depression assessment instrument in the literature (Fristad et al., 1997; Rivera et al., 2005). Such findings seem to indicate validity, but all we really know is that the test measures what it measures with consistency and that it correlates with some interesting and relevant attributes. Indeed, while the CDI has been shown to differentiate between clinical and non-clinical samples (Fristad, Weller, Weller, & Teare, 1987; Saylor, Finch, Spirito, & Bennett, 1984), there is evidence that it does not discriminate well between depression and other clinical diagnoses, such as anxiety (Comer & Kendall, 2005; Saylor et al., 1984). Having said that, it clearly measures something very well that appears to be important. The only argument here is that it might not be depression.

**Clinical Course**

Many of the papers in the 1960s and 1970s that presented the view that depression did not exist in childhood were based on clinical experiences or descriptive clinical studies, not on controlled experimental investigations or epidemiological surveys. The general view was that the nervous system during early childhood was too immature to support the complexity of thinking that was thought to be required for the expression of depression. For example, Rochlin (1965) expressed the belief that depression is not possible during childhood because the superego was not yet mature enough to produce the key elements of depression (guilt within this psychoanalytic framework). Mahler (1961) also emphasized the immature personality structure of the child when

### Table 1. Abstracted meaning from the 27 CDI items

<table>
<thead>
<tr>
<th>Item</th>
<th>Meaning</th>
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<tbody>
<tr>
<td>I am sad</td>
<td>unmotivated</td>
</tr>
<tr>
<td>nothing works</td>
<td>trouble sleeping</td>
</tr>
<tr>
<td>always wrong</td>
<td>always tired</td>
</tr>
<tr>
<td>no fun</td>
<td>poor appetite</td>
</tr>
<tr>
<td>I am bad</td>
<td>aches and pains</td>
</tr>
<tr>
<td>terrible things</td>
<td>feel alone</td>
</tr>
<tr>
<td>hate myself</td>
<td>school no fun</td>
</tr>
<tr>
<td>my fault</td>
<td>no friends</td>
</tr>
<tr>
<td>kill myself</td>
<td>school-mark drop</td>
</tr>
<tr>
<td>crying</td>
<td>others are better</td>
</tr>
<tr>
<td>bother me</td>
<td>nobody loves me</td>
</tr>
<tr>
<td>avoid people</td>
<td>disobedient</td>
</tr>
<tr>
<td>can’t decide</td>
<td>I fight</td>
</tr>
<tr>
<td>I look ugly</td>
<td></td>
</tr>
</tbody>
</table>

Table 1. Abstracted meaning from the 27 CDI items
claiming that “systematic affective disorders” are unknown in childhood. However, the subsequent decline in the popularity of psychoanalytic theory, concerns about deductions drawn from clinical observations, and the advent of behavioural research that supported the notion of childhood depression have caused such opinions to fade away from contemporary consideration. Having said that, I must note that I cannot escape my own clinical observations of many troubled children with a variety of presentations; there was nothing there that appeared like adult depression. Furthermore, recent experimental work has suggested that there is some evidence that affective disorders show meaningful changes over the developmental course, particularly across puberty.

In this regard, the first issue to consider is that scientists and clinicians have long wrestled with the relationship between anxiety disorders and depression. The two most widely used diagnostic systems, the International Classification of Diseases (ICD) and the Diagnostic and Statistical Manual (DSM) have treated them separately, but those currently preparing revisions (e.g. for the DSM-V and ICD-11) have been under pressure to treat them as one entity, called affective disorder (Goldberg, 2008; Watson, 2005). This is based on evidence indicating that it is not always a simple matter to separate anxiety and depression based on large-scale surveys, and many therapeutic medications developed for depression also work for anxiety (e.g. panic disorder) and vice versa.

Secondly, there is a growing body of literature showing that anxiety disorders tend to appear much earlier in life than depression (e.g. Thompson & Bland, 1989; Wittchen, Beesdo, Bittner, & Goodwin, 2003), and a number of longitudinal studies have indicated that anxiety is a risk factor for major depression in later life, whether it is measured in childhood (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000) or adolescence (Pine, Cohen, & Brook, 2001).

Furthermore, there is evidence from longitudinal studies that child depression, so called, appears to have different origins than adolescent and adult depression (Duggal, Carlson, Sroufe, & Egeland, 2001; Harrington, Rutter, & Fombonne, 1996) and is qualitatively different (Duggal et al., 2001; Harrington et al., 1996; McGee & Williams, 1988; McGee, Feehan, Williams, & Anderson, 1992; Seifer et al., 1989), with the adolescent and adult forms being relatively similar (Duggal et al., 2001; McGee et al., 1992). Notably, childhood depression was found to be associated with adverse family environments and relationships (Duggal et al., 2001; Harrington et al., 1996), suggesting that whatever construct that “childhood depression” actually refers to is related to disrupted attachment earlier on in life. Adolescent depression was associated with maternal depression only, possibly reflecting a genetic influence. These findings do implicate early childhood attachment difficulties, and certainly disruptions in attachment have been shown to predict depression. Duggal et al. (2001) found that early-life anxious attachment was associated with depression in adolescence, and Ingram (2003) has viewed attachment as being integral to the development of a cognitive vulnerability to depression that in turn, would appear later in life.

Finally, if we were to take measurements of childhood depression at face value, we see that the widespread finding that females show much higher rates than males, does not hold for pre-pubertal samples. Angold and Rutter (1992) found that boys and girls who had not yet reached puberty showed about the same prevalence of measured depression, but by age 16 girls showed twice the symptom rate of the boys. Since then, a number of studies have shown that among pre-pubertal children, the likelihood of showing so-called depressive symptoms was in fact higher among boys, with positions after puberty being reversed – girls clearly showing the higher rate (Cyanowski, Frank, Young, & Shear, 2000; McGee et al., 1992). While this suggests explanations that involve hormonal changes at puberty and/or other issues surrounding the transition from childhood to adolescence, Duggal et al. (2001) found that the ability of early childhood attachment to predict adolescent depression varied according to gender, thus adding another potential explanation to the mix.

The points raised above do not necessarily rule out the prospect that childhood depression is an existing condition. However, the evidence seems clear enough to warrant a second look at current thinking on its appearance. To paraphrase American writers James Whitcomb Riley and Douglas Adams, if it doesn’t look like a duck and it doesn’t sound like a duck, we have to at least consider the possibility that it is not a duck.

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References


