Further Investigation into the Rorschach and the Utility of a Modified DEPI in relation to Adolescent Depression.

By
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A Doctoral Project Submitted in Partial Fulfillment of the Requirements for the Degree of Doctor of Psychology in the Department of Psychology at Pace University

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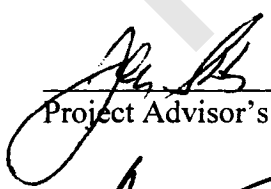

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ABSTRACT

This study investigated the Rorschach and its ability to identify adolescent depression through the current DEPI and CDI, and through a proposed adolescent depression index using Rorschach variables. Past research (Ball et al., 1991; Ilonen et al., 1999; Viglione, 1999) has shown that the Rorschach is a poor predictor of adolescent depression. A sample of 200 non-depressed inpatient adolescents and 77 severely depressed inpatient adolescents made up the extreme groups. The groups were formed by utilizing multiple criteria that was part of a comprehensive battery, but did not include the Rorschach. The current DEPI and CDI were not found to be effective predictors of adolescent depression. In general, the proposed adolescent depression index was not found to be significantly better than the original DEPI. However, variables such as morbid with shading, human content > 3, and AG + Fi + Bl > 3 were significant and distinguished between the extreme groups.

Chapter I

Introduction

There are multiple factors that interfere with making an accurate diagnosis of adolescent depression. One factor is the presence of co-morbid diagnoses with depression in adolescence (Brinkman, Overholster, & Klier, 1994; Brooke, & Kutcher, 2001; Parker & Roy, 1995; & Peterson, Compas, Brooks-Gunn, Stemmler, Ey, & Grant, 1993). Another factor that can interfere with making an accurate diagnosis is the variability of symptomology between adolescent and adult depression (Allgood-Merten, Lewinsohn, & Hops, 1990; Diego, Sanders, & Field, 2001; Parker et al., 1995). Further, the variability in diagnostic accuracy with different assessment measures also poses a problem (Carlson, Kula, & Laurent, 1997; Carter, & Dacey, 1996; Morgan-Gillard, 2002; Ornberg & Zalewski, 1994). Self-report measures, observer ratings, structured interviews and personality assessments using performance-based measures, such as the Rorschach are ways in which to help determine a diagnosis, however many times these different methods are not in agreement.

Depression in adolescence is associated with a higher likelihood of co-morbidity as compared to adults (Rohde, Lewinsohn, & Seeley, 1991). There are significantly higher co-morbid Axis I disorders for adolescents than adults (Peterson et al., 1993). Adolescent depression is often co-morbid with anxiety disorders, disruptive disorders, Attention Deficit Hyperactivity Disorder, and personality disorders (Parker et al., 1995). Co-morbid psychiatric disorders lead to difficulties in diagnosis.

There are some noted differences in adolescent depression as compared to adult depression that has been cited in the research (Allgood-Merten, Lewinsohn, & Hops, 1990; Diego, Sanders, & Field, 2001; Parker et al., 1995). Parker and Roy (1995) noted some significant features that distinguished adolescent depression from adult depression. One noted feature of adolescent depression was irritability and anger. Adolescent depression also has higher levels of anxiety, such as neuroticism, high trait anxiety, anxious worrying and shyness. Parker and Roy (1995) also noted that melancholic symptoms and psychomotor symptoms are common symptoms in adults but rare in adolescents. Increasing our understanding of the construct of adolescent depression will improve our abilities to assess this disorder.

The most challenging aspect of diagnosing adolescent depression for a professional is the lack of agreement between various methods of assessment. Much of the research has shown that there is little agreement between various assessment measures (Carlson, Kula, & Laurent, 1997; Carter, & Dacey, 1996; Morgan-Gillard, 2002; Ornberg & Zalewski, 1994). There have been notable problems in developing assessment methodology that is clinically useful and practical. Many of the assessments utilized have been self-reports or parent-report measures. Self-report measures are not always related to indirect measures such as the MMPI and the Rorschach Comprehensive System (CS) (Brinkman et al., 1994). There is also a higher prevalence of adolescent depression due to over-reporting (Allgood-Merten, Lewinsohn, & Hops, 1990). Finally self-report measures and parent-rating scales have a poor correlation with one another (Moretti et al., 1985). It is necessary to have a more reliable and valid assessment of depression that is based on the personality features of the individual. The Rorschach CS therefore has the potential to accurately identify depressed adolescents by utilizing a more indirect approach.

Other factors also interfere with the accurate diagnosis of adolescent depression. Stredny and Ball (2005) have stated that depressive features in adolescence may be harder to assess due to the fact that adolescents are less able to articulate their feelings. This in combination with their lack of patience, interest, or reading ability make them less likely to be able to complete self-report measures frequently used with adults.

Exner and Weiner (1982) studied the features of the Rorschach Comprehensive System that have been demonstrated to equate with depressive features in adults with children and adolescents. They found similarities between adult and adolescent depression however made warnings as to the limitations of their study. Other studies also revealed further the inadequacies of the DEPI, and its inability to distinguish adolescent depression (Archer, & Gordon 1988; Ball, Archer, Gordon, & French, 1991; Lipovsky, Finch, & Belter, 1989; Stredny, & Ball, 2005). The DEPI has been more effective in targeting the appropriate characteristics of adult depression, and has had more success in its diagnosis of depression for that particular population than for adolescent depression (Ilonen, Taiminen, Karlsson, Lauerma, Leinonen, Wallenius, Tuimala, & Salokangas, 1999, Viglione 1999, Ball et al., 1991).

The purpose of this study is to explore the variables within the Rorschach CS in order to more effectively assess adolescent depression. The present CDI and DEPI have an unacceptably high false positive rate (Ball, Archer, Gordon & French, 1991). Therefore, the present study will rely on an extreme group design utilizing multiple criteria given as part of the comprehensive battery, but not including the Rorschach. The two groups will be those adolescents that were severely depressed at the time of their hospitalization as compared to those that were not depressed. The Rorschach variables will be assessed and will include

existing DEPI and CDI variables as well as other Rorschach variables, and the adolescent depression index will be assessed. The established depressed and non-depressed groups of the adolescent inpatients will be compared to determine the discriminating power of these Rorschach variables. A proposed depression index will be assessed with the extreme groups. The ability of the proposed adolescent depression index will be compared to the DEPI and CDI to assess which index is better able to diagnosis adolescent depression.

Chapter II

Review of the Literature

History of Adolescent Depression:

According to Kaslow, Croft, and Hatcher (1999), prior to the 1970's the dominant psychoanalytic theory maintained that depression could not occur among children and adolescents due to their level of psychological development. Other theories held that depressive symptoms in children and adolescents were transitory phenomena. These types of views limited the examination of depression in youth. However, a transition occurred when childhood depression was acknowledged as a mental disorder at the Fourth Congress of the Union of European Pedopsychiatrists in 1970. Over the past 25 years, child and adolescent depression has received increased attention, and the understanding of child and adolescent mood disorders has increased. Internalizing disorders in adolescents include the anxiety and affective disorders. They consist of problems related to worry, fear, shyness, low self-esteem, sadness, and depression. Internalizing problems in childhood and adolescence have detrimental effects on a growing child. Depressive disorders affect a significant number of adolescents, and there are important developmental factors to consider when making a diagnosis and in planning effective treatment for youngsters with these disorders. A thorough understanding of risk factors, development, family factors, and individual cognitive variables should be taken into account. Even though the existence of depressive disorders in adolescents is no longer a question as it was in the past years, effective diagnosis and assessment of depression is problematic. (Kaslow et al., 1999)

DSM-IV-TR Criteria for Depressive Disorders:

According to the DSM-IV-TR (2000), unipolar depression, or Major Depressive Disorder (MDD) is characterized by a history of one or more major depressive episodes without manic, hypomanic, or mixed episodes of mood disturbance. For a DSM-IV-TR diagnosis the youth's symptoms must cause impairment, must reflect a change from baseline, and may not be secondary to uncomplicated bereavement; the diagnosis also requires at least five of the following symptoms during the same two-week period. The symptoms are as follows: depressed or irritable mood, anhedonia, decreased weight or appetite or failure to make expected weight gains, sleep disturbance, psychomotor agitation or retardation, fatigue or loss of energy, feelings of worthlessness or inappropriate guilt, concentration difficulties or indecisiveness and thoughts of death and/or suicide. One symptom must be depressed or irritable mood or anhedonia. While criteria are virtually identical for youth and adults, DSM-IV-TR acknowledges that psychomotor retardation, hypersomnia, and delusions are rare in prepubertal children, whereas somatic complaints, irritability and social withdrawal may be prominent. In the DSM-IV-TR descriptive specifiers for the most recent episode refer to severity of depression, presence of psychotic features, remission status, chronicity and course, and the presence of catatonic, melancholic, or atypical features.

According to the DSM-IV-TR (2000), Dysthymic Disorder (DD) is characterized by chronically depressed or irritable mood and two of the following symptoms: appetite change, sleep change, decreased energy, low esteem, difficulty concentrating, and feelings of helplessness. In order for a youth to receive a DSM-IV-TR diagnosis of DD, symptom duration must be at least one-year (as opposed to two years for adults), without a symptom free period of two months. DD before the age of 21 is early onset. There are a number of

depressive diagnoses according to the DSM-IV-TR, however for the purpose of this study only these diagnoses will be utilized.

Kaslow et al. (1999), has critiqued the DSM-IV criteria for mood disorders as it lacks a developmental perspective failing to account for adolescent's cognitive, affective, and interpersonal competencies and biological maturation. These criteria are similar to the DSM-IV-TR and therefore these areas remain incomplete in terms of adolescent depression. The lack of information for adolescents makes diagnosis more difficult.

Course of Adolescent Depression:

Research has begun to examine the degree to which child or adolescent unipolar depression is chronic or relapsing. Costello, Pine, Hammen, March, Plotsky, Weissman, Bierdman et al. (2002) conducted a meta-analysis of the findings of the research done over the past twenty years on the development and natural history of major affective disorders. Evidence suggests that childhood depression entails a high risk for recurrence during adolescence. Similar rates of recurrence between 30% and 50% are reported in studies that observe adolescents through early adulthood (Angst et al., 1990; Lewinsohn et al., 1999; Rao, et al., 1999; Warner, et al., 1992). The few studies that have followed depressed children through adolescence into adulthood have all found an increased risk (Fombonne et al. 2001; Garber et al., 1988, Harrington et al., 1990; Lewinsohn et al., 1999, Rao et al., 1999, Weissman et al., 1999). The consequences of depression during childhood and adolescence are significant. Depressive disorders are neither normal developmental occurrences nor short-lived problems that dissipate with time. Even when episodes remit, they commonly recur and interfere with the child's ability to function competently. Cicchetti and Toth (1998) reported that when DD as compared to MDD is the first to emerge in children, there is a greater risk for

developing subsequent mood disorders. Kovacs, Akistal, Gatsonis, and Parrone (1994) found that 76% of children with earlier onset DD developed a subsequent MDD, and 69% with DD as the first emergent mood disorder developed a combined DD and MDD (double depression). They also concluded that the average length of an episode of MDD in children and adolescents was seven to nine months. Approximately 90% of MDD episodes remit within two years post onset, whereas the remaining episodes last for a more protracted period of time. MDD frequently recurs in children and adolescents.

Parker and Roy (1995) stated that, "depressive episodes tend to last 7-9 months on average, and while 90% remit within one to two years, the remainder experience distinctively longer episodes or develop chronic depression. Thus aggregated longitudinal studies indicate a 40% cumulative probability of recurrent depression by two years and 70% by five years". Depression in adolescents is often recurrent depending on the severity and number of episodes. Research has shown that MDD can frequently recur in adolescents.

Parker and Roy (1995) found that the rates of comorbidity in association with depression have been estimated from 40% to 95% with the most frequent being anxiety disorders, such as over-anxious disorder, agoraphobia, social phobia, separation anxiety, enuresis, obsessive-compulsive disorder and dysthymic disorder. The next most frequent disorders are the disruptive disorders, such as conduct, antisocial disorder, and ADHD. Personality disorders are also comorbid with depressive disorders, such as antisocial and borderline personality. Comorbid psychiatric disorders lead to difficulties in diagnosis and increased risks for recurrent and longer periods of depression as well as poorer treatment response and treatment recommendations. (Parker, & Roy, 2001).

Peterson, Compas, Brooks-Gunn, Stemmler, Ey, and Grant (1993), also reported significantly higher co-morbid of Axis I disorders for adolescents than adults. In a large community sample, 42% of the adolescents who had experienced a depressive disorder had an Axis I co-morbid disorder, which was significantly higher than expected from the base rates and higher than the rates for adults with depression. Co-morbidity is similar for males and females, however males are more likely to have both disruptive behavior disorders and depression and females were more likely to have eating disorders and depression. Depression in adolescence is associated with a higher likelihood of co-morbidity with other disorders than is true for adults (Rohde et al., 1991).

Etiology:

Genetic Factors:

Risk for depression in adolescence is associated with parental depression, for biological, environmental, and interpersonal reasons. Weissman et al. (1987) found that having a depressed parent increases the risk for their children to suffer from depression. The risk for transmission of depression from parent to child considers heritability, depressive maternal affect and symptoms, stress within the household with a depressed parent, and neuroregulatory consequences from that environmental circumstance. The timing, severity, and duration of depressive symptoms interact with the developmental tasks and challenges for adolescents which also contributes to their psychological well-being. The effect of early relationship quality on later depression in adolescence is unclear, as is the contribution made by youngsters with a predisposition for depression to the negative quality of their interpersonal interactions. However, depressed youth do appear to have poorer social competence, perceive less support from peers, and spend less time with peers than do non-depressed youth.

Interpersonal deficits or struggles, including peer and parental relationships are a part of child and adolescent depression.

Genetically, informed studies of high-risk families have shown that there is a considerable familial component to unipolar depression. Costello et al. (2002) stated that these observations have been demonstrated in follow-up and follow-back studies. In top-down studies, children of depressed parents have been found to be approximately three times more likely to have a lifetime episode of depression than offspring of controls. In bottom-up studies, the first-degree relatives of depressed children have been found to have significantly elevated rates of major depression.

Merikangas and Low (2004), concur with the Costello et al. (2002) findings. Merikangas and Low (2004) reviewed the epidemiology of mood disorders. The role of genetic factors in the etiology of mood disorders has been suspected for more than a century. The numerous articles reviewed by Merikangas and Low (2004) have shown that genetic factors are involved in the susceptibility to mood disorders, particularly bipolar disorder. The average risk ratio for MDD among relatives of probands with MDD compared with those of control participants was 3.6, indicating a moderate familial aggregation on unipolar mood disorders. Twin studies have also revealed that genetic factors have a greater etiologic role in bipolar disorder than MDD. Twin studies have shown that mood disorders are strongly heritable, with bipolar disorder having a greater degree of genetic involvement. Adoption studies are extremely important in assessing genetic factors and environmental factors in the etiology of mood disorders. There are few adoptions studies for mood disorders, and those that were done involved small samples. According to Merikangas and Low (2004), with respect to bipolar disorder there is little evidence for differential risk among biologic compared to

adoptive relatives of adoptees with bipolar disorder. There are also few controlled family studies that have focused on the manifestation of mood disorders among adolescents. Controlled studies of offspring of parents with bipolar disorder exhibit wide variation in the frequency of mood disorders among their offspring, it ranges from 23% to 92%, collectively it represents a familial component. Children of bipolar parents show greater specificity of transmission of mood disorders than do children of parents with unipolar depression.

Wals and Verhulst (2005), describe the continuity of mood disorders from childhood to adulthood, in relation to genetic factors and environmental adversities. Their findings concur with Costello et al. (2002), in that environmental adversity in childhood can have prolonged effects that it may influence the mental health of the individual. Traumatic experiences interfere with normal development of psychosocial functions, and the likelihood of onset of depression is greatest if environmental stress occurs in genetically vulnerable individuals. According to their results, Wals and Verhulst (2005) indicated that child and adolescent adversity increases the risk for onset of mood disorders in adulthood. However, a problem with studies on the effects of life events is that it is often unclear whether life events are contributors to depression onset or consequences of depression through person-environment correlation.

According to Kaslow et al. (1999), adolescents are at increased risk for depression if they have a family history of depression, particularly if a parent evidences depression. Monozygotic twins, even when reared apart, have a higher rate of mood disorder than dizygotic twins. Adopted children of biological parents with a positive history for depression have more depression than do adopted controls.

In a prospective longitudinal study by Duggal, Carlson, Sroufe, and Egeland (2001), significant differences emerged for factors associated with child onset and adolescent onset depression. Abuse at an earlier age, higher maternal stress, and less supportive early care differentiated the two. In general adverse family relationships were associated with childhood onset depression but not adolescent onset depression. In the adolescent onset group, adverse family relationships were less significant, however greater levels of maternal depression were noted. Therefore, maternal depression influences adolescent depression.

In addition to inherited factors, specific neurotransmitters have been implicated in the onset and course of the depression in children and adolescents. One prominent theory according to Ollendick, Shortt, and Sandler (2005), suggests that select neurotransmitters such as monoamines, norepinephrine, serotonin, and dopamine are not available at receptor sites in sufficient supply. Kaslow et al. (1999) also reported that abnormalities in neurotransmitter systems, such as acetylcholine, norepinephrine, serotonin, and neuropeptides have been observed and postulated to be of etiological significance in adult depression. Sleep and neuroendocrine abnormalities in depressed adults may indicate a dysfunction of the limbic system and the hindbrain areas of the brain that may be involved with affective illness. Although early studies failed to demonstrate sleep abnormalities in depressed children, pre-pubertal children studied after recovery from depression have improved sleep continuity compared to when they were depressed. They had decreased rapid eye movement (REM), latency, and increased REM density compared to normal and non-depressed controls. Neuroendocrine markers in the depressed youth yield a variety of abnormalities, including growth hormone abnormalities and elevated serum thyrotropin. Taken together these

preliminary data make it premature to postulate a neurobiological etiological model of childhood depression.

Environmental Factors:

Psychosocial issues have been linked with affective disorders, such as high neuroticism in parents. The goal of the study by Ellenbogen and Hodgins (2004) was to evaluate the idea that high neuroticism in parents represents an environmental risk factor for behavioral and emotional problems among offspring who are at elevated risk for affective disorders. High neuroticism in parents was associated with indices of genetic risk for affective disorders. The participants included parents with MDD, BD and with no mental illness. The parents had to have at least one child between the ages of 4-14. A number of assessments were performed both on the children and the parents. The parents with high neuroticism were associated with a host of psychosocial deficits that included poor occupational, social, and familial functioning, difficulty in intimate relationships and low levels of social support. These parents then experienced more negative life events and provided low levels of emotional support to the offspring. This then suggests that high levels of neuroticism lead offspring to have negative life events. It was also found that major affective disorders, such as MDD and BD and neuroticism share a common genetic vulnerability. Ellenbogen and Hodgins (2004) displayed that inappropriate parenting and presence of an affective disorder puts the offspring at greater risk for an affective disorder and poor coping skills and supports.

Population based studies (Brown et al. 1999, Johnson et al. 2001, and Mulder et al. 1998) described significant associations between depression, abuse, maltreatment, and related forms of environmental adversity. Abuse is a pathway that may lead to depression, and there seems to be multiple pathways to depression in children and adolescents. Recent studies

document the potential long-lasting effects of stress exposure on human physiology, brain structure, and function. Adversity during critical periods of prenatal or postnatal brain development seems to induce plastic changes, leading to permanent alterations in perceptions of, and responsiveness to environmental events.

Gender Differences and Prevalence Rates:

The prevalence of depression varies depending on setting, age, sex, sociocultural background, informant, and measurement devices. Prevalence and incidence rates of depressive disorders vary across studies, and large-scale research is sparse in the area of child and adolescent depression. However, on average narrowly defined DSM major depression is thought to affect 1% to 2% of children and 2% to 4% of adolescents at any given time. Prevalence rates of depressive disorders are different for boys and girls according to Ollendick, Shortt, and Sander (2005). "The World Health Organization/World Bank's estimates of the global burden of the disease, calculated in terms of disability-adjusted life years (DALYs), found that unipolar depression was the leading cause of disability world-wide in the 15-44 year old age group, whereas bipolar disorder is the sixth most disabling disorder" (World Health Organization, 1996). Even though the effect on children has not been calculated there is growing evidence that early mood disorders are widespread, recurrent, and often chronic diseases, which increase the risk of lifelong disability. The differences change systematically with development, adding an additional factor to consider.

Before adolescence there are approximately equal proportions of depressive disorder among boys and girls. However, beginning in adolescence and continuing into adulthood, depressive disorders occur more frequently among females than males. Although the reason for these differences is unclear, they are found to be consistent across cultures. Cicchetti and