THEORY AND TREATMENT OF ALEXITHYMIA: AN AFFECT THEORY PERSPECTIVE

By

David Rose

A DISSERTATION

Submitted to Michigan State University in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

Department of Psychology

2002

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

UMI Number: 3100493



UMI Microform 3100493

Copyright 2003 by ProQuest Information and Learning Company. All rights reserved. This microform edition is protected against unauthorized copying under Title 17, United States Code.

> ProQuest Information and Learning Company 300 North Zeeb Road P.O. Box 1346 Ann Arbor, MI 48106-1346

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

ABSTRACT

THEORY AND TREATMENT OF ALEXITHYMIA: AN AFFECT THEORY PERSPECTIVE

By

David Rose

Alexithymia is a personality construct that refers to the inability to identify or describe feelings. Alexithymia is associated with a wide range of negative outcomes regarding both psychological and physical well being. Current understanding of effective treatments for alexithymia is limited. This study aims to remedy these deficits by studying alexithymia treatment from the perspective of <u>affect theory</u>. Affect and Self-Esteem is a psychoeducational curriculum taught to undergraduate students at Michigan State University that is based on the principles of affect theory. The present study investigated the influence of the ASE curriculum on two measures of alexithymia, the TAS-20 and the LEAS, and on a measure of psychopathology, the BSI.

Results indicated a significant increase in LEAS scores in the ASE curriculum compared with a control group, but no significant change in TAS-20 scores. Contrary to prediction, Obsessive-Compulsive increased significantly in the experimental group, and there were significant interactions for Global Psychopathology, Interpersonal Sensitivity, Anxiety, Hostility, Paranoia, and Psychoticism indicating a trend toward increasing scores in the experimental group and decreasing scores in the control group. Further, change in TAS-20 scores, but not in LEAS scores, was a significant predictor of changes

in BSI scores. Finally, it was found that as LEAS scores increased, the correlation between the LEAS and the TAS-20 became more strongly negatively correlated.

Dedicated to Lisa Martin for her constant, wacky, and everloving support. I doubt I would have accomplished this without you. Thanks Red!

ACKNOWLEDGMENTS

I would like to thank the members of my committee including Gershen Kaufman, Alexander "BMW-F1" von Eye, Bertram P. Karon, and the highly esteemed Andrew M. Barclay for their help, time, and assistance. Thanks guys! I would also like to thank Laura Symonds for her assistance both as research mentor and for helping out with the committee. A personal thanks goes out to Mike "Smell Ya' Later" Cutler, Tamineh "Queen of the Eyeroll" Gueremy, Brett "God, I hate those yuppies" Johnson, Dan "I'm Dan Martin" Martin, Marc "Evil Metalhead" Paschke, and all the other wackos in my life who helped to keep me sane during graduate school. You people rock! Finally, a super special heartfelt appreciation goes out to Greta McVay and Seka Remsing for their excellent support and warm humor.

TABLE OF CONTENTS

LIST OF TABLES	viii
LIST OF FIGURES	ix
CHAPTER 1	
STATEMENT OF THE PROBLEM AND LITERATURE REVIEW	1
Alexithymia Theory and Research	1
Definition and Description of Alexithymia	1
Comorbidity and Consequences	4
Etiology of Alexithymia	8
Primary Alexithymia: Neuropsychological Factors	8
Secondary Alexithymia: Retrograde Trauma	10
Secondary Alexithymia: Anterograde Trauma	11
Secondary Alexithymia: Social and Family Factors	11
Treatment of Alexithymia.	13
Affect Theory	
Script Theory	
An Affect Theory Approach to the Treatment of Alexithymia	23
Learning the Language of Affect	24
Dissolving Shame Binds	25
Overcoming Trauma	27
Coping with Chronic Powerlessness	33
Learning to Use Imagery	35
Conclusion	36
A Psychoeducational Curriculum Based on Affect Theory	36
Unit I: Powerlessness – Affect – Stress Cycles	37
Unit II: Shame and Self-Esteem	38
Unit III. Identity: The Self's Relationship With the Self	39
Unit IV: Affect Regulation and Release	40
Unit V: Interpersonal Relationships	41
Prior Research	42
CHAPTER 2	
DESIGN OF THE CURRENT STUDY	45
Hypotheses	46
CHAPTER 3	
METHOD	47
Subjects	47
Measures	47
Toronto Alexithymia Scale – 20	47
Levels of Emotional Awareness Scale	48

Brief Symptom Inventory	49
Procedure	50
Data Analysis	51
CHAPTER 4	
RESULTS	
Data Prenaration and Data Cleaning	52
Data Analyses	52
Hypothesis 1	52
Hypothesis 7	53
Hypothesis 3	53
CHAPTER 5	
DISCUSSION	
APPENDICES	60
Appendix A: Table 1	61
Appendix B: Table 2	63
Appendix C: Table 3	65
Appendix D: Figure 1	66
Appendix E: Figure 2	67
Appendix F: Informed Consent Form	68
Appendix G: Demographic Questionnaire	70
Appendix H: TAS-20.	71
Appendix I: LEAS	73
Appendix J: BSI	
••	
BIBLIOGRAPHY	

LIST OF TABLES

<u>Table</u>		Page
1	Means and Standard Deviations for Variables by Group across Time	61
2	Repeated Measures MANOVA of TAS-20, LEAS, and BSI Scores	63
3	Summary of Regression Analyses for Change in TAS-20 and LEAS Scores Predicting Changes in BSI Scores	65

LIST OF FIGURES

Figure		Page
1	Scatterplot of TAS-20 and LEAS Scores Where LEAS Scores are Less Than 65 ($\underline{r} = .134$)	66
2	Scatterplot of TAS-20 and LEAS Scores Where LEAS Scores are Equal To or Greater Than 65 ($\underline{r} =017$)	67

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

Chapter 1

STATEMENT OF THE PROBLEM AND LITERATURE REVIEW

Alexithymia is a personality construct that refers to the inability to identify or describe feelings. Alexithymia is associated with a wide range of negative outcomes regarding both psychological and physical well being. Current understanding of effective treatments for alexithymia is limited. The few empirical studies that have been conducted have several limitations. First, existing studies have used alexithymia measures with poor psychometric properties. Next, the majority of treatment studies have only used self-report measures of alexithymia. Finally, none of the interventions for the treatment of alexithymia have been grounded in a comprehensive theory of affect. This study aims to remedy these deficits by studying alexithymia treatment from the perspective of <u>affect theory</u>. This will also shed new light on the understanding of alexithymia itself.

ALEXITHYMIA THEORY AND RESEARCH

Definition and Description of Alexithymia

Alexithymia is a widespread, but little known condition that influences the way people experience and express emotion. More specifically, alexithymia, which literally means "no words for feelings" (Sifneos, 1972), is the inability to identify, describe, and distinguish between subjective feelings. The prevalence of alexithymia is estimated to be 39.8% in psychiatric outpatients (Taylor, Parker, Bagby, & Acklin, 1992), and 8 to 17% in student samples (Campos, Chiva, & Moreau, 2000). Currently, the 20-item Toronto Alexithymia Scale (TAS-20) is the most psychometrically solid and widely used measure

of alexithymia (Bagby, Parker, & Taylor, 1994). According to these researchers, individuals with alexithymia have difficulty in the three following areas: 1) identifying feelings; 2) describing and expressing feelings; and 3) an externally oriented cognitive style which reflects the inability to think about or reflect upon their own internal affective experiences (Bagby, Parker, et al., 1994). As a result, individuals with alexithymia often cannot tell whether they feel sad, angry, or anxious, despite being aware of unpleasant sensations of physiological arousal (Krystal, 1988).

The externally oriented thinking associated with alexithymia is believed to stem from a cognitive condition known as <u>pensee operatoire</u>, which means "thought operating" (Montreuil, Jouvent, Carton, Bungener, & Widlocher, 1991). Because alexithymics cannot symbolically represent and understand their inner environment, their perception of the external environment changes, consequently they tend to focus on concrete objects and the surface dimensions of objects. As a result these individuals usually present as dull, mundane, unimaginative, and utilitarian; their verbal descriptions resemble a sequential recitation of concrete facts. Not surprisingly, individuals with high levels of alexithymia demonstrate impaired creativity and fantasy (Clerici, Albonnetti, Pupa, Penata, & Invenizzi, 1992) and a lack of visual imagery (Campos et al., 2000; Friedlander, Lumley, Farchione, & Doyal, 1997).

Alexithymics also suffer from <u>aprosody</u>, or lack of affect in speech, and a deficit in the generation of spontaneous facial expressions. In addition, alexithymics have a pervasive deficit in their ability to recognize emotional stimuli: matching faces with words, sentences with words, sentences with faces, and faces with photographs of scenes (Lane, Sechrest, Riedel, Shapiro, & Kaszniak, 2000). According to Buck (1988),

emotion has the following three components: 1) subjective feelings, e.g., "I feel sad"; 2) facial and bodily elements of expression, e.g., frown, smile, crying, laughter, and voice tone; and 3) physiological arousal, e.g., increases in heart rate or skin conductance. Thus, although alexithymia is defined as a deficit in the ability to identify and communicate the first component of emotion, subjective feelings, it is also accompanied by deficits in the expression and perception of both verbal and nonverbal forms of emotion.

In recent years, there has been a growing consensus as to what alexithymia actually measures. Most theorists argue that the key characteristic of alexithymia is that there is a deficit in the symbolic representation of emotion (Clerici et al., 1992; Taylor, Bagby, & Parker, 1997). This implies that subjective physiological sensations are "normal" in terms of intensity. However, the patterns of autonomic arousal underlying affect are poorly differentiated because of a linguistic deficit in which subjective feelings are not semantically and verbally encoded. Therefore, it is believed alexithymics have the three basic components of emotion, but what is lacking are the connections or the linkages between the components.

Consistent with this line of thought, Lane and Schwartz (1987) have created a developmental theory for the cognitive differentiation of emotional states based on Piaget's theory of the stages of cognitive development. They argue that the cognitive identification and differentiation of emotions becomes increasingly complex with healthy development. Further, Lane and his colleagues (Lane, Quinlan, Schwartz, Walker, & Zeitlin, 1990) have developed a psychological measure, the Levels of Emotional Awareness Scale (LEAS), to assess the developmental level of cognitive complexity of emotion. The LEAS is comprised of five levels of increasing complexity. Participants

are asked to read a short vignette and to describe how both they and the other person in the vignette would feel. At the lowest level of scoring responses refer to thoughts and not emotions, whereas at the highest level responses refer to blends of discrete affects, such as I felt "glad" or "disappointed."

More recently, Bucci (1997) combined principles of contemporary cognitive science with psychoanalysis to form Multiple Code Theory. Essentially, Bucci argues there are three primary modes of thought: subsymbolic nonverbal (patterns of autonomic arousal and sensory information), symbolic nonverbal (images), and symbolic verbal (words for affect). In the absence of symbolic processing of nonverbal nonlinguistic information, physiological activation may go unregulated and "activation is likely to be prolonged and repetitive (Bucci, 1997, p. 165)." Bucci argues that in alexithymia all three modes of thought are disconnected, and that sub-symbolic nonverbal information may first have to be connected with symbolic nonverbal images before it can be linked to symbolic verbal words for feeling.

Comorbidity and Consequences

In some instances, it appears that alexithymia develops as a response to intense negative affect. Support for this comes from research literature on comorbid psychopathologies associated with alexithymia (Krystal, 1988). Alexithymia tends to be found in populations that are exposed to painful and distressing circumstances. Alexithymia has been found at high levels in brutally traumatized populations with posttraumatic stress disorder (PTSD) such as concentration camp survivors (Krystal, 1988), military combat veterans (Hyer, Woods, Summers, Boudewyns, & Harrison, 1990), and survivors of repeated sexual abuse (Zeitlin, McNally, & Cassiday, 1993).

Hyer et al. (1990) studied the relationship between PTSD and alexithymia in a sample of Vietnam Veterans ($\underline{N} = 227$). They found 85% of the PTSD patients to be alexithymic, using the MMPI alexithymia scale. In addition, these researchers observed clinically that PTSD patients and alexithymics share several similar characteristics. PTSD patients cannot read their own emotions and they tend to have many physical complaints, much like patients with alexithymia. Other research has shown that alexithymia and PTSD patients have similar patterns of neurotransmitters (Henry, Haviland, & Cummings, 1992). Both groups were found to have abnormally high norepinephrine levels, suggesting greater activation and effort of the left hemisphere compared to normals.

Alexithymia has also been found at high levels in populations that have been exposed to less acute traumatic situations, for example, such as those that lead to depression (Honkalampi, Hintikka, Tanskanen, Lehtonen, & Viinamaki, 2000; Taylor et al., 1992) and panic disorders (Parker, Taylor, Bagby, & Acklin, 1993; Zeitlin & McNally, 1993) as well as in dysfunctional families and abusive relationships (Mallinckrodt, King, & Coble, 1996), and also in stages of racial identity believed to involve poorer adjustment and greater distress (Dinsmore & Mallinckrodt, 1996).

Because individuals who have high alexithymia levels have difficulty identifying and differentiating internal states, it is difficult for them to use feelings as cues for selfregulation and the maintenance of homeostasis. Accordingly, alexithymics have difficulty regulating glucose levels, and high levels of alexithymia correspond positively with high rates of diabetes, bulimia nervosa, and anorexia nervosa (Abramson, McClelland, Brown, & Kelner, 1991; Taylor et al., 1992). The difficulty that

alexithymics have in identifying their feelings also leads to poor functioning in interpersonal relations, especially those that are intimate (Swiller, 1988).

As previously mentioned, because alexithymics cannot identify and symbolically represent their feelings, they often experience undifferentiated and unpleasant sensations of negative arousal. Research indicates that alexithymics have a delayed perception of internal stress states (Naataven, Ryynanen, & Keltikangas-Jarvinen, 1999). Taylor et al. (1997) suggest that individuals with alexithymia may attempt to modulate these negative sensations through food, the use of psychoactive substances, and compulsive behaviors. They argue that deficits in the ability to self-regulate affect may explain the high comorbidity of alexithymia with eating disorders, substance abuse, psychopathic deviance, impulsive and acting out behaviors, and pathological gambling. Further, alexithymia is associated with a <u>threefold</u> increase in the risk for death due to external causes such as accidents, injuries, and violence (Kauhanen, Kaplan, Cohen, Julkunen, & Salonen, 1996). Greenberg, Rice, and Elliott (1993) argue that psychopathology originates, in part, because of the inability to identify and express emotion. Thus, alexithymia can be seen as detrimental to the ability to process information regarding one's immediate environment.

Alexithymia is also related to a wide number of important personality constructs. Recently, there has been a growing interest in "emotional intelligence" mainly in response to Goleman's work (1995). According to Taylor (2000), "... emotional intelligence includes the ability to identify and label one's own and others' emotional states, the ability to express emotions accurately and make empathic responses to others, and the ability to reflect on emotions and use them in adaptive ways (p. 136)." A strong

negative correlation ($\underline{r} = -.72$) has been found between alexithymia and a recently developed measure of emotional intelligence, the BarOn Emotional Quotient Inventory (Taylor, 2000). Another interesting line of inquiry shows that while alexithymics and non-alexithymics have equivalent dependency needs according to projective measures, alexithymics fail to acknowledge they have dependency needs on self-report tests (O'Neill & Bornstein, 1996). These findings are related to another recent study which shows that high alexithymia levels are associated with primitive defense mechanisms such as passive aggression, projection, splitting, somatization, devaluation, and acting out (Kooiman, Spinhoven, Trijsburg, & Rooijmans, 1998).

Rates of psychosomatic disorders are also high among patients with alexithymia (Krystal, 1988). Headaches, numbness, constipation, pain in joints, dry mouth, heartburn, somatoform disorder, hypertension (high blood pressure), and inflammatory bowel disease (ulcerative colitis and Crohn's disease), all correlate positively with alexithymia (Cox, Kuch, Parker, Schulman, & Evans, 1994; Jula, Salminen, & Saarijarvi, 1999; Porcelli, Leoci, Guerra, Tayloy, & Bagby, 1996; Porcelli, Zaka, Leoci, Centonze, & Taylor, 1995; Todarello, Taylor, Parker, & Fanelli, 1995). Alexithymia is also related to lowered pain tolerance thresholds and is found in high frequency among chronic pain patients (Lumley, Asselin, & Norman, 1997; Nyklicek & Vingerhoets, 2000). Psychosomatic disorders are approximately twice as common among alexithymics as compared to the general population (Kauhanen, Kaplan, Julkunen, Wilson, & Salonen, 1993; Taylor et al., 1992).

Finally, alexithymia is associated with impairment of the immune system and carcinogenesis (Todarello et al., 1994; Todarello et al., 1997). Todarello et al. (1997)

suggest that the inability to symbolize and modulate emotions may lead to an increase in sympathetic arousal accompanied by elevation of blood norepinephrine levels, which leads to the physiological state we call "stress." This physiological stress state then leads to a malfunctioning of the immune system, and subsequently to an increase in the risk of cancer. These findings emphasize the utility of the alexithymia construct in studying the mind-body relationship between psychological and physiological processes, an area of investigation that is increasingly prominent in the research literature.

Etiology of Alexithymia

Alexithymia has two primary sources. Primary alexithymia has a biological basis which involves either or both of the following: 1) disconnection of interhemispheric areas of the neocortex via the corpus callosum (Parker, Keightley, Smith, Taylor, 1999; Zeitlin, Lane, and O'Leary, 1989), and 2) reduced activity in the anterior cingulate cortex (Lane, Reiman, et al., 1998). Secondary alexithymia is related to psychosocial factors, which are believed to result in a psychogenic "functional disconnection" of these neuroanatomical pathways (TenHouten, Hoppe, Bogen, & Walter, 1985). There are three possible sources of secondary alexithymia noted in the literature. First is exposure to traumatic situations in the past, or proactive trauma. Next is fear of psychological distress in the future, or retroactive trauma. Finally, there are various social factors and family environments the directly impact the incidence of alexithymia.

<u>Primary Alexithymia: Neuropsychological Factors.</u> Neuropsychological research suggests that alexithymia is related to a deficit in communication between the two cerebral hemispheres. Historically, alexithymia was noted to have occurred in the "splitbrain" patients of the 1960s. These patients had their corpus callosums severed in an

attempt to lessen the severity of intense epileptic seizures. Since the corpus callosum connects the right and left hemispheres of the brain at the level of the cortex, it was hypothesized that alexithymia was caused by a lack of communication of information between the two hemispheres (TenHouten, et al., 1985). Buck (1994) states that humans lack connections between the two hemispheres at the subcortical level and thus, the corpus callosum of the cortex is the primary route for interhemispheric communication. Two studies have used the tactile finger location task to determine the level of callosal transfer in non-neurologically impaired individuals (Parker, et al., 1999; Zeitlin, et al., 1989). The studies used the TAS-20 and the TAS-26, respectively, to measure alexithymia. Both studies found a positive correlation between high alexithymia scores and deficits in interhemispheric transference. Dewaraja and Sasaki (1990) found that alexithymics suffer from a deficit in transferring nonlinguistic information from the right to the left hemisphere, but not from a deficit in the callosal transfer of linguistic information.

Recently, the first study on alexithymia using contemporary brain imaging techniques was conducted (Lane, Reiman, et al., 1998). Participants were 12 nonclinical females who were shown emotion inducing pictures, and were also asked to recall emotional memories from their lives, while undergoing Positron Emission Tomography (PET) imaging. PET relies on the injection of radioactive oxygen into the bloodstream. Thus, PET is believed to be an indicator of changes in blood flow. Participants also completed the LEAS. Results show a positive relationship between LEAS scores and activation of the right anterior cingulate cortex. This suggests that individuals who are poor at cognitively processing and differentiating their emotional experiences may have

reduced activity in this region, especially in the presence of emotion inducing stimuli. These findings are consistent with our current understanding of the role of the anterior cingulate cortex in cognition and emotion. Lane, Ahern, Schwartz, & Kaszniak (1997) hypothesize that one of the many functions of the anterior cingulate cortex is that it is responsible for the conscious awareness of affect.

<u>Secondary Alexithymia: Retrograde Trauma.</u> There is much evidence to suggest that previously occurring psychological distress and trauma, or retrograde trauma, is a factor in the etiology of alexithymia. The intensity of an emotion that can be tolerated by anyone at anytime is finite. People who have experienced emotional distress beyond a certain threshold often experience automatization, robotization, freezing and momentary paralysis, or even psychogenic death in extreme cases (Krystal, 1988). This going beyond the threshold <u>is trauma</u>, and it is associated with the degree to which one feels completely helpless in a certain situation. Therefore, as one approaches this threshold, one is motivated to initiate activity to block or evade conscious recognition of emotion.

The blocking of intense affect that is associated with alexithymia may actually serve an adaptive function in some situations. In environments that are emotionally painful or where emotional expression is potentially harmful, alexithymia may act as a buffer between the individual and the environment, thereby enabling the individual to cope with an environment which the individual is unable to change (Dinsmore & Mallinckrodt, 1996). However, if the blocking of painful affect causes permanent and long-lasting change in that individual's own functioning, then a psychopathological state will ensue (Krystal, 1988). Once the affect barrier is in place, it is extremely difficult to

remove because the individual is highly motivated to avoid re-experiencing the intense and negative affect that was associated with the traumatic event.

Secondary Alexithymia: Anterograde Trauma. Anterograde trauma is the fear of overwhelming psychological distress in the future. The individual believes he or she is completely helpless to avoid the anticipated negative affect. Alexithymia is found in patients who are diagnosed with terminal illnesses and medical conditions, such as cancer. Fukinishi, Saito, and Ozaki (1992) found that alexithymia increased in patients after the initiation of hemodialysis therapy. The researchers speculate that the increase in alexithymia may be due to the activation of denial-defense mechanisms against psychological distress engendered by the hemodialysis treatment.

Secondary Alexithymia: Social and Family Factors. Research has also shown that alexithymia is related to sociodemographic factors. In a study of middle-aged men in Finland (N = 2,682), alexithymia correlated positively with lower levels of education, less annual income, poor social support, and lower occupational status (Kauhanen et al., 1993). A more recent study found positive associations between alexithymia and low levels of education, low SES, old age, and male gender (Lane, Sechrest, & Riedel, 1998). A possible explanation for these results is that all of these factors are associated with greater levels of psychological distress, from which alexithymia develops as a self-protective coping mechanism. Thus, alexithymia may develop as a way to fend off the chronic negative affects associated with lack of status, lack of power, and social isolation in one's social environment. Differences between the genders in affect socialization may also be a risk factor for higher alexithymia levels in men (Levant, 1998).

One variable which has not been found to be related to alexithymia is intelligence. A review of three studies by Taylor et al. (1997) found only a weak negative correlation or no correlation between alexithymia and intelligence. Thus, alexithymia does not appear to be related to a deficit in verbal intelligence.

A number of studies have examined the role of both parent-child relationships and family environments among alexithymics. Alexithymia is associated with low maternal care, low maternal-child bonding, and insecure attachment (Fukunishi, Sei, Morita, & Rahe, 1999; Solano, Toriello, Barnaba, Ava, & Taylor, 2000). Berenbaum and James (1994) found that certain childhood family environments correlated highly with alexithymia. The best predictor of alexithymia was having grown up in a home where little positive and supportive communication existed. Alexithymia rates were found to be high in people that grew up in family environments where family members were not permitted to express, or felt unsafe, expressing their feelings. Lumley, Mader, Gramzow, & Papineau (1996) found that maternal alexithymia and child alexithymia were indeed correlated. They also identified a number of family factors that were positively correlated with alexithymia levels, including under- and over-emotional involvement, lack of family values, and poor family problem solving abilities. These findings suggest that children who are not raised in a family environment where they can learn to regulate their own affects are at an increased risk to develop elevated levels of alexithymia (Taylor, et al., 1997; Taylor, 2000).

The last 15 years has seen a large acceleration in the rate of growth of the alexithymia literature base (Taylor, 2000). When aggregated, these research findings indicate the alexithymia construct is related to a variety of risk factors for deleterious

outcomes regarding both psychological and physical health. In addition, a number of etiological factors have been investigated, suggesting that there are likely multiple pathways involved in the development of alexithymia. Unfortunately, research on the treatment of alexithymia is lagging in both in terms of quantity and quality compared with these areas.

TREATMENT OF ALEXITHYMIA

Alexithymia has received much attention in the psychotherapy literature because it predicts poor treatment outcomes, especially when psychotherapeutic interventions requiring insight and expression of emotions are used (Taylor et al., 1997). According to Krystal (1982), alexithymia may be the single most important impediment in treatments involving psychoanalysis and psychodynamic psychotherapy. Other theorists have gone even further and cautioned that the use of psychoanalytic psychotherapy with alexithymic patients could be harmful to them because they create excessive arousal which the individual is unable to regulate (Taylor, et al., 1997). Subsequently, the first theoretical attempts in the treatment of alexithymia were made by psychoanalysts who had to use treatment approaches that were not founded on traditional psychoanalytic theory. What subsequently developed was a trial and error approach to treatment.

Apfel-Savitz, Silverman, and Bennett (1977) used intensive group therapy lasting two years. The therapy focussed on guiding clients to talk about feelings regarding life events, the use of videotape to give feedback of nonverbal forms of emotional communication such as facial expressions, and the use of meditation to relax clients so they can focus their attention inside themselves. More specific details are lacking in their report.

Swiller (1988) also used group psychotherapy to treat alexithymia. The first stage in treatment was to teach clients how to label their affects, and some of this was done individually before the client was introduced to the group. The group therapy environment had to be safe, supportive, and empathic in order as to protect clients' selfesteem and allow them to explore their difficulties labeling emotions. According to Swiller (1988), group therapy for alexithymia offers four advantages over individual therapy. First, emotional arousal is dampened by the presence of others in the group. Second, the alexithymic patient can see that other alexithymic patients are having the same problems and therefore do not feel as isolated or unusual. Third, the client is able to get feedback from the group about how others perceive the expression of their emotions. Finally, alexithymic patients can mimic others who are more advanced in their emotional skills. For these reasons, Swiller (1988) recommends that the group consists of no more than three alexithymic patients per group of seven, and that the alexithymic patients in the group be at different stages of treatment.

Krystal's (1988) theory of alexithymia is largely derived from his understanding of trauma and defense mechanisms from a psychoanalytic perspective. Krystal (1988) argues that alexithymia is the result of a self-protective, homeostatic mechanism against excessive psychological distress and pain. According to this theory, if the level of psychological pain that is consciously experienced becomes too great, death can occur, as in cases of psychogenic death. Therefore, as the level of psychological pain reaches a certain threshold, a mechanism is triggered that causes a freezing of experience, thereby dampening the amount of pain. This mechanism may in fact become permanent because the individual is motivated never again to experience those painful feelings. As a result

of this process, the individual feels less emotion. From Krystal's (1988) perspective, alexithymia is not caused by a deficit in the ability to liguistically encode feelings, but rather it is caused by emotions that are weak in intensity or absent altogether and therefore difficult to differentiate or even detect.

This leads Krystal (1988) to view alexithymia as resulting from a regression (in adults) or a state of arrest (in children) due to psychic trauma. Psychic trauma arises from any situation where an individual becomes overwhelmed by intense affect. Krystal (1982) argues that there are three primary lines of normal affective development. First, the differentiation of specific affects from a more global affect system occurs. Second, individuals learn how to recognize and verbalize their various affective states. Finally, as individuals learn how to verbalize their affect, the expression of affect becomes desomaticized in its expression. According to Krystal, alexithymia is the regression or arrest of affective development in these areas, and thus alexithymia is a state characterized by difficulty differentiating affect and verbalizing affect, which modulates an increase in psychosomatic distress. Regarding treatment, Krystal notes that traditional psychoanalytic methods, which rely on introspection of affective experiences, are rarely helpful in cases of alexithymia. However, he gives little specific information about interventions that do work well in the treatment of alexithymia (Krystal, 1988).

Levant (1998) understands alexithymia as primarily developing from the normal male gender role socialization process. More specifically, Levant argues that alexithymia develops in males primarily because of two reasons. First, emotions that make one feel vulnerable such as fear, sadness, and rejection are prohibited in males. Second, emotions related to interpersonal connection such as caring, warmth, affection, and neediness are

also prohibited in males. Levant's treatment for alexithymia in males takes a psychoeducational and cognitive-behavioral approach to help clients accurately identify and label their emotions, especially those emotions related to vulnerability and interpersonal connection.

There are very few empirical studies which have looked at treatments for alexithymia. de Groot, Rodin, and Olmsted (1995) employed intensive group psychotherapy for eating disorders with 50 bulemics. Psychotherapy focussed on symptom management, nutrition, body image, relationships, improving the identification of emotions, and validating the expression of these emotions. The treatment group had significantly reduced alexithymia levels compared to controls, although the majority of participants in the treatment group were still in the alexithymic range after treatment. The study also found that as alexithymia scores were reduced, so were binge/purging symptoms of bulimia. One major limitation of this study is that it used the 26-item Toronto Alexithymia Scale (TAS-26), which has been shown to have questionable psychometric properties (Taylor et al., 1997).

Beresnevaite (2000) recently conducted a study on post-myocardial infarction patients in Lithuania. Participants were assessed for alexithymia before treatment, next at the end of a 4-month treatment, and subsequently at follow-ups of 6 months, 1 year, and 2 years. The treatment consisted of 16 weekly 90-minute sessions of group therapy over a period of 4 months. The treatment consisted of four main components. First, subjects were taught progressive muscle relaxation. Second, verbal and nonverbal expressions of emotion were promoted by various techniques including role playing and nonverbal mimicry, describing the emotional states and fantasies of self and others, and writing

down thoughts and fantasies while listening to music. Third, subjects were encouraged to talk about inner experiences instead of external events by replacing utilitarian statements with statements that expressed wishes and desires. Fourth, every session concluded with a hypnotic type of relaxation to reduce any heightened physiological activity.

The control group received two 90-minute educational sessions about coronary heart disease, including risk factors and coping methods. Also, five of the control subjects received four sessions of progressive muscle relaxation. Results indicate alexithymia levels dropped in the treatment group but not in the control group, and that these changes were maintained until 2 years later. 55% of the subjects in the treatment group demonstrated a reduction in alexithymia levels. In addition, a reduction in alexithymia was associated with a reduction in cardiac events over a 2-year period. Within the treatment group, 67% of the subjects who <u>did not</u> experience a change in alexithymia levels had cardiac events over the 2-year period, whereas only 18% of the subjects who <u>did</u> experience a reduction in alexithymia levels had cardiac events over the same time period. The number of cardiac events did not significantly differ between the experimental group and the control group over the 2-year period. This study has limitations because it also used the TAS-26 to measure alexithymia, and there was a small sample size of 20 in the experimental group and 17 in the control group.

Thus, the majority of work regarding the treatment of alexithymia is theoretical in nature. Very few empirical studies exist. The empirical studies that do exist have significant limitations due to the use of outdated and psychometrically poor measures of alexithymia, and because the measures of alexithymia that were used rely on self-report. Self-report measures have come under question in general, because "defensive deniers"

may underreport psychological difficulties and distress (Shedler, Mayman, & Manis, 1993), and specifically in the case of alexithymia, because the very skill needed to accurately self-report is the skill being measured. Therefore, individuals who have difficulty identifying and labeling their feelings may have low interoceptive awareness and may not be able to accurately self-report their inner experiences (Lane, Sechrest, et al., 1998). Another major problem with currently existing treatments for alexithymia is that they are not based on any central organizing theory. What is needed is an intervention which is grounded in a comprehensive and well-supported theory of emotion: affect theory.

AFFECT THEORY

Beginning in the late 1950s and early 1960s, Silvan Tomkins (1962, 1963, 1991, 1992) introduced a new theory for understanding human behavior, to which he gave the name <u>affect theory</u>. Tomkins's formulation was revolutionary because he argued that affect is the primary innate biological motivating mechanism in human beings. Affect takes precedence over both the drives and cognition. The human being comprises a series of discrete yet interdependent motivating sub-systems including affective, cognitive, sensory, motor, pain, and drive mechanisms. Tomkins hypothesized that the nature of the relationship between affect and cognition is such that affect influences the affect-laden quality of cognition. For example, positive affect predisposes one to recalling pleasant memories, seeing oneself and others in a positive light, and providing positive attributions, explanations, or "reasons" for why an event occurred, whereas negative affect does just the opposite (Tomkins, 1962, 1963). A child who has experienced fear and shame due to physical abuse may tend to ascribe angry and hostile

intent to others in subsequent ambiguous social situations. This is consistent with research by Dodge (Crick & Dodge, 1996) which has found a "hostile attributional bias" in children who have been physically abused.

The purpose of affect is to impart a sense of urgency to anything associated with it. Affect works as an amplifier. When affect is combined or co-assembled with any of the other sub-systems, "the affect amplifies by increasing the urgency of anything with which it is co-assembled (Tomkins, 1981, p. 322)." In this way, affect is the primary source of motivation in human beings. Further, the other sub-systems have to be amplified by affect in order to function properly. For example, if the sex drive is fused with shame, every time the sex drive is activated, shame also will be activated, including feelings of inadequacy and wanting to hide, a facial display of blushing or looking down, and associated physiological changes, resulting in sexual dysfunction. In order for the sex drive to function optimally, it must be fused with and amplified by excitement and enjoyment, the positive affects. The excitement experienced during sex is the same excitement experienced when attending an automobile race, or travelling to a new and exotic location.

Tomkins's theory posits that affect is innate and not learned. Tomkins has theorized the existence of nine innate affects which are universal in all cultures. The innate affects are defined as "sets of muscle and glandular responses located in the face and also widely distributed through the body, which generates sensory feedback which is either inherently 'acceptable' or 'unacceptable'" (Tomkins 1962, p. 243). Thus, features of the affective response include facial muscle responses, vocalization, respiration, blood flow, skin responses, and various muscle responses.

Tomkins posits the face to be the primary site of the affective response (Tomkins, 1981). Each innate affect has its own corresponding facial expression. The affect programs that control these facial expressions are located in the subcortex (Tomkins & McCarter, 1964) and are triggered by different patterns of neural stimulation, in a manner similar to how the ear detects different tones (Tomkins, 1981).

The following is a list of the proposed nine innate affects, along with the accompanying distinct facial display (Tomkins & McCarter, 1964; Kaufman, 1992, 1996).

- 1. Interest Excitement: eyebrows down, eyes track, look, listen
- Enjoyment Joy: smile, lips widened up and out, smiling eyes (circular wrinkles)
- 3. Surprise Startle: eyebrows up, eyes blink
- Distress Anguish: cry, arched eyebrows, mouth down, tears, rhythmic sobbing
- Fear Terror: eyes frozen open, pale, cold, sweaty, facial trembling, with hair erect
- 6. Anger Rage: frown, clenched jaw, eyes narrowed, red face

Auxiliary

- 7. Shame -- Humiliation: eyes down, head down
- 8. Dissmell: upper lip raised
- 9. Disgust: lower lip lowered and protruded

The affects with a double - name indicate differences in intensity, ranging from mild to intense. Only interest -- excitement and enjoyment -- joy are considered to be positive affects because one is motivated to maximize or approach these affects. They feel rewarding. All other affects with the exception of surprise -- startle are considered negative affects because one is motivated to minimize or avoid these affects. They feel punishing. Surprise -- startle is a resetting affect, which quickly resets consciousness in order to attend to another stimulus. These affects are found to be innate among human beings across all cultures. In many cases, these affects are only seen exhibited in infancy, as the expression of affect is culturally patterned. In a given culture, certain affects are discouraged from full expression, while the expression of other affects is encouraged.

The nine innate affects are similar to the three primary colors, red, blue, and yellow, in that they serve as the foundation for all other affective experiences. Like the primary colors, all other affects are derived from these primary affects. The innate affects also can be blended together. For example, contempt is a learned affect blend of dissmell and anger, and depression is a blend of shame and distress which are conjointly brought to peak intensity and prolonged for an extended period of time. Tomkins also introduces the concept of <u>affect complexes</u> (Tomkins, 1981), where the same underlying affect may be activated in different circumstances and in different ways. For example, the innate affect shame, when activated before an audience, is termed embarrassment, while shame activated in the presence of strangers is labeled shyness. Nonetheless, the underlying affect of shame remains identical in these two situations. However, because the activator of shame is different, there is a different and unique experience and phenomenology for each shame complex. By extension, the same holds for each of the

affects. Thus, we can see the operation of the various innate affects in explaining such affective states as ecstasy and hatred.

Strong empirical evidence supports Tomkins' theory of innate affects. Tomkins and McCarter (1964) found a .86 correlation between pictures of faces posed to represent the primary innate affects and the judgements of affect. Further research by Ekman (1971) has extended these results cross-culturally.

Tomkins argues that much of the experience and expression of affect that we see among adults is not innate affect but rather <u>pseudo</u> or <u>backed-up affect</u>. Because the expression of affect is highly contagious and leads others to experience the identical affect, all societies control the expression of affect. Thus, the expression of affect is inhibited by constricting breathing and suppressing both the vocalization and facial display of affect. Backed-up affect also leads to changes in the endocrine system, which in turn leads to stress. Backed-up affect is thus a risk factor for psychosomatic diseases such as high blood pressure and asthma (Tomkins, 1979). Understanding the concept of backed-up affect provides important insights into the mind-body connection.

Script Theory

Script theory is Tomkins's attempt to apply his understanding of affect toward a comprehensive theory of human personality. Tomkins introduces three concepts in his script theory: <u>scene</u>, <u>scene magnification</u>, and <u>script</u>. A scene is the basic element in life as it is lived (Tomkins, 1979). Scenes are essentially emotional memories, except that they are not static "photographs" stored in memory. Scenes have the additional quality of being dynamic, in that scenes can interconnect and fuse directly together, and thus grow in magnification. Scene magnification occurs when scenes comprised of the same affect

fuse together, becoming interconnected, and the affect thereby grows or is magnified in intensity. Scenes become magnified when scenes of the same affect are repeated with a difference. When shame is activated in different circumstances, for example under different conditions or with different persons, the shame scenes fuse together and magnify each other, thereby resulting in shame becoming a more pervasive experience.

Scripts are the rules for interpreting, responding to, controlling, escaping, avoiding, and celebrating scenes (Tomkins, 1979, 1987). According to Tomkins, scripts develop from scenes when three things occur. First, discrete scenes are co-assembled into a family of scenes and thereby undergo psychological magnification. Second, the magnified scene is thought about or cognitively processed. Third, the individual then designs strategies and tactics, scripts, for responding to that family of scenes in the future (Tomkins, 1979, 1987). The function of scripts is to increase overall positive affect by increasing the frequency of positive affect scenes and decreasing the frequency of negative affect scenes. Scripts originally develop from a family of magnified scenes, but they also increasingly come to determine future scenes.

AN AFFECT THEORY APPROACH TO THE TREATMENT OF ALEXITHYMIA

Unlike previous attempts to understand and treat alexithymia, affect theory understands alexithymia as arising from any combination of five sources. Therefore these etiological factors must be assessed, with different psychotherapeutic intervention applying to each of the five factors. Thus, affect theory allows us to be more specific than any previously proposed theories, and we would expect better treatment outcomes because of this increased specificity. The five factors leading to the development of

alexithymia are: 1) a lack of language for one's affects; 2) affect-shame binds; 3) trauma;4) chronic exposure to situations of powerlessness; and 5) imagery.

Learning the Language of Affect

Alexithymia is more likely to develop in individuals if they have not learned to accurately identify and label their inner experiences. For example, the subjective experience of feeling painfully diminished and revealed as the piercing eyes of others or the self are able to see our innermost flaws is best labeled as a <u>shame</u> experience. Affect theory allows us to develop a "roadmap" of inner experience, which helps us to become clear and precise in the identification and labeling of our affects. Thus, when working with individuals whose alexithymia primarily develops because they never initially formed the language of affect and how to symbolically represent their inner experience through language, treatment should focus on teaching clients the vocabulary of the innate affects.

After the individual has gained a thorough understanding of the innate affects, the individual is then able to engage in the process of Differentiated Owning (Kaufman, 1991). Differentiated Owning consists of three primary actions or steps: 1) <u>experiencing</u> the affect; 2) <u>naming</u> and accurately labeling the affect; and 3) <u>owning</u> the affect as an inherent and valid part of the self.

In experiencing the affect, the individual focuses attention inward and allows for the experiencing of ambiguous physiological sensations to their fullest intensity. The individual becomes fully immersed in the experience of interoceptive information. Individuals may vary widely in their ability to recognize and consciously experience internal events, which in turn influences their ability to approach and experience negative

affect. In a therapeutic context, the therapist increases the likelihood of approaching unpleasant internal events by providing a relationship that maximizes the positive affects of excitement and enjoyment while also minimizing or neutralizing negative affects, especially shame, and also by enabling the client to cope more effectively with the sources of negative affect.

Second, clients are helped to accurately name and label their inner experiences. Language is the primary mechanism that brings inner experience into full conscious awareness. Tomkins's model of the affect system can be used as a theoretical guide: together, therapist and client examine the client's inner experience as it is lived, trying to match up internal experience with affect phenomenology as well as activators of specific affects. In this way, the group of innate affects and their activators help to serve as a map which clients can use to illuminate their own inner experience.

Finally, clients must fully connect with and integrate the affects into their concept of self by owning all of their affects. They must be able to say, and believe: "These feelings are an important and valuable part of me." This very important step allows for both the valuing of the affects, and an increased social intelligence because the client learns the evolutionary adaptive value of each affect. This in turn enables clients to have more information at their disposal with which to make salient social decisions.

Dissolving Shame Binds

A shame bind develops when the affect of shame follows the expression of any affect. The outward expression of any affect may be discouraged or prohibited by others, resulting in the activation of shame. Initially, shame inhibits the expression of the prohibited affect. Repeated exposure to further events during which expression of the

affect is prohibited and is thus paired with shame eventuates in inhibition of the internal experience of that affect. The experience of the affect may be partially or completely wiped away from awareness.

For example, an anger-shame bind might develop in a child if she is reprimanded for expressing anger, even when appropriate. The reprimand can be any combination of verbal and other non-verbal forms of communication, such as facial expressions or voice tones of anger, contempt, disgust, or mocking and derisive forms of humor. Shame is also elicited in this situation when the anger is met with indifference or an absence of a response. A single event of high shame affect intensity or several events of low shame affect intensity, which fuse together and magnify in intensity, may solidify the linkage between the original affect of anger and shame. Subsequently, when anger is triggered in this individual, she will have difficulty expressing her anger because shame will spontaneously inhibit it. In addition, she may not experience any anger, but only shame, or she may experience a mixture of both shame and anger.

It is at this point that shame binds can give rise to alexithymia. In this case, the shame bind may be so strong that it not only prohibits the expression of anger, but also erases the conscious awareness of anger. When asked to describe how she feels, she may not be able to identify that she feels angry, but may only be aware of the experience of shame, which may manifest itself through feeling wrong, bad, or even guilty.

Shame binds are commonly patterned by gender in the culture: females are differentially shamed for expressing the affects of excitement and anger, whereas males are commonly shamed for expressing the affects of fear, distress, and shame (Kaufman, 1996; Kaufman & Raphael, 1996). Thus, the development of shame binds first inhibit
and control the expression of affect, but then eventually lead to diminished awareness of the internal experience of affect.

To dissolve shame binds, they must be brought back into full conscious awareness. The person needs to be able to identify all of their affects and label them accurately. At this point, there are two routes available. First, the person can look inward and identify their shame binds. Then they can follow this back in history and become aware of early scenes in which the shame bind is rooted. This will be sufficient to dissolve shame binds in many individuals. However, in cases where the shame bind persists, scene transformation will be necessary. In scene transformation, the person fully visualizes and re-experiences the shame in the original shame-bound scene. Transforming the original shame scene involves imagining what was needed to in order to feel validated and affirmed in the expression of the original affect.

Scene transformation alters the way the original shame scene is stored in memory. The process of repeatedly replaying the original shame scene immediately followed by the transformed scene breaks the bind between the original affect and shame and replaces it with the fusion of the original scene to the new affects of enjoyment and excitement. As a result, the person gradually experiences a sense of confidence and well-being in expressing the previously prohibited affect.

Overcoming Trauma

In order to understand trauma from the viewpoint of affect theory, an understanding of its three primary components is necessary: <u>affect</u>, <u>scene</u>, and <u>script</u>. Affect is the set of innate affects which are the primary motivators of human behavior.

Affect imparts a sense of urgency on our world and makes us care about whatever it is that triggers the affect.

According to Kaufman (1992, 1996), scenes are comprised of three primary components: affect, imagery, and language. Imagery includes not only the visual representation of the scene, but may include records from other sensory modalities as well, such as auditory, kinesthetic, and tactile images or sensory imprints. The language component of the scene may come from a variety of sources: it could be the vocalization of the affect associated with the scene; it could be something said by the individual experiencing the scene; it could be something said by another person who is in the scene; or it could manifest as inner dialogue or self-talk. For example, it is not unusual for someone to say or hear "You stupid idiot!" at a subvocal level of communication to oneself following a shame scene like making a mistake or blunder. Finally, affect combines with imagery and language to complete the processes that generate the scene.

Affect-laden scenes stored in memory will fuse with other scenes of the same affect in a process Tomkins refers to as <u>psychological magnification</u> (Kaufman, 1996). If sufficient magnification of scenes occurs, a family of scenes -- also referred to by Kaufman (1992, 1996) as a <u>governing scene</u> -- emerges which then captures and dominates the self. For example, in cases of trauma, the emotional memory of the traumatic event lays the foundation for the governing scene. An important aspect of scene dynamics is that scenes can be re-triggered in the present by any new situation which is sufficiently similar to an old scene but also different. Old scenes can be re-triggered by activators in the present, for example, the smell of urine or the voice tone of a stranger.

There are three ways in which scene dynamics play out. First, scenes can be <u>reactivated</u>: here the affect, imagery, and language components associated with the original governing scene or family of scenes are reexperienced in the present. Scenes are reactivated by stimuli and events in the present which are similar to stimuli and events associated with the governing scene. The scene reactivated may involve any combination of affect, imagery, and language. For example, an individual may experience affects in the present which are seemingly unrelated to any causal event. In this case, it is likely that only the affective component of a scene has been reactivated and consciously experienced, while the language and imagery associated with the scene are not in awareness.

Second, scenes can be directly <u>reenacted</u>. Scene reenactment occurs when a scene is both reactivated and when a role from the original scene is played out in the reactivated scene. For example, a boy who was physically abused by his father may have this scene reactivated when he becomes a parent himself. In the case of scene reenactment, he could find himself playing either the identical or else a different role from the abused child which he played in the original scene. When that reenactment involves role reversal, the scene has been <u>recast</u>. Thus he may find himself playing the role of his father as an adult, and actually physically abusing his own son. He, in effect, has recast the scene.

A third aspect of scene dynamics is that of <u>scene intrusion</u>. This occurs in the aftermath of trauma when scenes of enormous affect invade or intrude directly into consciousness, seemingly spontaneously. It is common for traumatized populations, such as war veterans, to have "flashbacks" of traumatic scenes in which the individual

suddenly feels as though he or she is reliving the traumatic event. Consciousness is completely overtaken and invaded by the intruding scene. Individuals may report experiencing a blackout of current conscious functioning while being swept away by the intruding scene. When scene intrusion occurs during sleep, it is called night terrors. In the case of trauma, governing scenes of intense negative affect are created and are subject to all three forms of scene dynamics.

Scripts are the rules for predicting, interpreting, responding to, and controlling scenes. For example, a person who has been traumatized may develop a phobic script because these encounters reactivate the unpleasant affect associated with the original scene. Scripts originally develop in response to scenes, but increasingly they come to determine future scenes. Thus, a female who is raped while walking alone at night may seek out scenes which involve daylight and companions.

Trauma leads to the development of alexithymia in two primary ways. First, the affect embedded in the traumatic scene may not be in conscious awareness. Scene dynamics are such that any combination of the three elements of the traumatic scene -- affect, imagery, and language -- may or may not be available to conscious awareness (Kaufman, 1992, 1996). For example, an individual who only has access to the imagery component of the traumatic scene will be able to recall traumatic events in great detail without experiencing any of the affect associated with the traumatic event. In other individuals, an inner voice associated with either words actually spoken during the traumatic event or new words spoken silently to the self may be the only component of the traumatic event which the individual is aware of. In both of these cases, the individual has lost conscious awareness of the affect associated with the traumatic event.

This makes it difficult to accurately identify and label affects associated with trauma, thereby contributing to the development of alexithymia.

The second way that trauma leads to the development of alexithymia is that the intense negative affects associated with trauma, such as terror, humiliation, and anguish, in turn activate the pain drive system. At this point, the pain system goes into an emergency response which is activated by the perceived threat to the physical integrity and existence of the organism and a massive analgesic response is elicited, which dampens the subjective experience of the intensity of all nonsymbolic information. Alexithymia results from this reduction in the subjective experience of the signal intensity of the affects. Thus, the ability to experience all of the affects is interfered with, and is replaced by a state of emotional numbness and flatness, accounting for symptoms such as "psychic numbness" and "anhedonia" which are associated with severe trauma.

In the treatment of trauma from an affect theory perspective, the original governing scene must be transformed. Scene transformation is comprised of three primary steps. First, the scene must be fully recovered in conscious awareness. This means that the three components of the scene—affect, imagery, language—must be recovered and integrated into one coherent reconstituted whole. Second, one must fully re-experience the affect embedded in the traumatic governing scene. It is not enough to simply label and describe the affect. There must be a direct experiencing of the affect in combination with the imagery and language from the scene. Full vocalization of the original affect coupled with more relaxed, full breathing may be important components in reexperiencing traumatic affect. A stronger working alliance and <u>interpersonal bridge</u> (Kaufman, 1992) between therapist and client will make it easier for the client to

approach the affect embedded in the governing scene as opposed to continuing to avoid it. The key component of a strong working alliance is the interpersonal bridge between therapist and client, where mutual enjoyment is the primary affect in the relationship. Enjoyment allows the client to feel cared for and respected, which results in the subjective experience of feeling safe and secure.

A third step in the transformation of scenes involves the creation of a new scene, in which the action and affect associated with the original scene are changed. <u>Reparenting imagery</u> is a technique that is used to transform scenes; the adult, stronger version of the self is re-introduced into the original scene involving the younger self (Kaufman, 1996; Kaufman & Raphael, 1991). When asked, "What is it you needed most at this time to feel safe and secure?" The client begins constructing a new scene with new action that is combined with the affect of enjoyment, the source of warmth and safety. In a traumatic scene of victimization, for example, the victimization is stopped through the introduction of the adult self who acts to stop or prevent the victimization in the original scene. If the client believes that the adult self would have been unable to have met their emotional needs during the traumatic event, the image of a trusted older adult may be recruited and inserted into the scene, even the therapist if the client so desires.

When the traumatic scene has been transformed, it may still be unexpectedly reactivated, but the intensity and duration of the affect embedded in the governing scene will be greatly attenuated. This allows the individual to cope in a more effective way with the traumatic event as the individual becomes better able to successfully manage scene reactivation.

Transformation of the traumatic governing scene will also reduce levels of alexithymia. As the affect in the governing scene is released and transformed, the analgesic barrier that protects the individual from reexperiencing affect slowly begins to lift because it is no longer needed. Also, the affects associated with the governing scene are now brought fully and willfully into awareness, where they can be appropriately modulated and regulated by the individual.

Coping with Chronic Powerlessness

Power is the felt sense of inner control over events in one's environment. Powerlessness, the experience of little or no control over events in one's life, lies on a continuum with power. Power and powerlessness are extremely potent activators of affect in a fairly predictable manner: power elicits positive affect, while powerlessness elicits negative affect. A powerful governing scene is created in infancy and childhood when human beings experience the inevitable period of helplessness, which is longer than for any other species. Current situations of experienced powerlessness will tend to reactivate the affects associated with helplessness in infancy. The affects that are triggered by powerlessness include all of the innate negative affects, the affect blend of contempt, which is a combination of anger and dissmell, and depression, which is a blend of shame and distress that are conjointly brought to peak intensity and prolonged over time.

Chronic powerlessness leads to the development of alexithymia because the negative affects that are triggered are rapidly magnified in their intensity and are subsequently suppressed. The suppression of the rapidly magnified negative affects makes them difficult to readily identify and label.

In order to treat alexithymia, which arises from chronic powerlessness, two basic steps must be taken. First, the specific negative affects that are triggered during powerlessness must be accurately identified, experienced, and released. If the negative affects are not fully experienced, this will lead to backed up affect, which has deleterious psychological consequences. Backed up affect leads to an exaggerated and prolonged emotional response when that affect is triggered. Also, backed up affect inpacts the endocrine system, leading to hormonal changes, and also adversely affects the functioning of the immune system.

After the negative affects that are triggered by chronic powerlessness are identified and experienced, at least two choices for dealing with the situation of powerlessness differently must be identified. Choice creates power, reduces powerlessness and helplessness, and thus reduces the chronic activation of negative affect.

Power differences between groups are a very real and important part of our world. It is crucial that the therapist validate differences in power experienced by individuals so that identifying the affects associated with scenes of powerlessness can be explored and new choices for dealing with powerless situations created. Examples of groups that are more powerful in contemporary American culture include: adults, men, wealthy, whites, boss, heterosexual, able bodied, Christian, formally educated, and born in the United States. Less powerful groups include: youths, women, poor, people of color, worker, lesbian, gay, bisexual, transgender, people with disabilities, Jewish, Buddhist, Atheist, other non-Christians, non-formally educated, and recent immigrants. The affect dynamics of powerlessness speak directly to group membership.

Learning to Use Imagery

Affect theory puts a significant emphasis on the use of imagery. Tomkins argued that information or "messages" become conscious only when they first take the form of an image: "What is consciously experienced is <u>imagery</u> which is created by the organism itself" (Tomkins, 1962, p. 13). According to Kaufman (1996), imagery is the link between inner and outer experience. Thus, imagery is required for the internalization of affect and scenes, which come to form the identity of the self. As already noted, imagery encompasses auditory, kinesthetic and visual components. In this regard, empirical research has found that alexithymia is associated with poor visual imagery skills (Campos et al., 2000; Friedlander, et al., 1997). Further, Bucci (1997) argues that it may be necessary for alexithymics to first symbolize physiological and sensory information with imagery before connecting it with language. Deficits in imaging ability may develop in alexithymia for a number of reasons, including shaming for daydreaming, shaming for discussing dreams, and trauma.

Imagery is used in almost all of the psychological interventions discussed above. Imagery is employed in enabling individuals to remember early scenes of powerlessness, shame, and shame binding. And, in the reparenting imagery process, individuals are taught how to use active imagery to transform traumatic scenes. Imagery is used identify unmet developmental needs, to identify how those needs are to be met, and to provide directly for those unmet developmental needs. It is hypothesized that the frequent use of imaging techniques in affect theory approaches will help the alexithymic to increase their imaging ability, thereby increasing the ability of the alexithymic to make affects

conscious and also symbolize them. This, in turn, allows for affects to be identified and described more readily.

Conclusion

Affect theory views alexithymia as a multidetermined construct: any combination of the five following factors may lead to the development of alexithymia. First, individuals may have difficulty labeling affect because they never learned the language of affect. Second, shame binds not only inhibit the expression of affect, but they also erase the conscious awareness of affect. Third, trauma promotes alexithymia through the disconnection of affect from imagery and language in the traumatic scene, and also by an analgesic response from a hyperactivated pain drive system. Fourth, chronic powerlessness leads to alexithymia because negative affect is activated and subsequently suppressed, making it difficult to identify affect. Fifth, imagery may be necessary in order to promote the symbolic representation and, in turn, the conscious awareness of affect. Because of the greater specificity and precision of affect theory in understanding both the etiology and treatment of alexithymia, treatment outcomes for alexithymia ought to be significantly improved.

A PSYCHOEDUCATIONAL CURRICULUM BASED ON AFFECT THEORY

Kaufman (1991, 1992, 1996) has taken Tomkins's affect and script theories and developed them into a psycho-educational curriculum designed to improve overall psychological health while also teaching the principles of affect. This program is an analog of psychotherapy. The program is offered as an undergraduate psychology course at Michigan State University, titled "Affect and Self-Esteem." The course meets twice a week for 80 minutes per class meeting over a 15-week semester. The course consists of a conceptual mode in which students are taught how to apply principles of affect theory and an experiential mode in which students directly engage their own experience through various "tools" or psychological self experiments. These affect tools are translations of the theory into action strategies. The course consists of five units, each two to four weeks in length.

Unit I: Powerlessness – Affect – Stress Cycles

Power and powerlessness lie at opposite ends of a spectrum. Power is the felt sense of inner control, and powerlessness is the experience of little or no control. Power and powerlessness are activators of affect: power activates positive affect, while powerlessness activates negative affect. Because human beings experience a lengthy period of helplessness, which is longer than that for any other species, a powerful governing scene is created. Current situations of experienced powerlessness will tend to reactivate the affects associated with helplessness in infancy. The affects triggered by powerlessness include any or all of the six innate negative affects, plus the learned affect blend of contempt, a combination of anger and dissmell, and depression, which is a blend of shame and distress that together are brought to peak intensity and prolonged over time. Whenever adults encounter powerlessness, this will activate any combination of negative affects. It will also re-activate the governing scene of powerlessness from infancy and childhood.

Two sets of tools are used in this unit. The first set of tools consists of collecting and storing positive affect by keeping two lists: a Happiness List and a Pride List. In the first, participants are asked to recall five events each day that leave a smile on their face; in the second, they have to recall five events every day that leave them feeling proud.

The purpose of these tools is to promote the storage of positive affect scenes, and to transform pride from always being performance-contingent to being generated from within. The second set of tools directly addresses the problem of powerlessness. Participants are asked to identify and describe a current situation in which they feel powerless, the affects experienced, and also to identify two choices for dealing with the powerlessness scene. Becoming aware of powerlessness and creating choices for dealing with it directly enhance power.

Unit II: Shame and Self-Esteem

Kaufman conceptualizes low self-esteem as shame-based. Low self-esteem is shame that has been internalized, a state in which shame is the primary affect of the inner relationship of the self. On the other hand, positive self-esteem arises from the affects of enjoyment and excitement. Participants are taught how to identify the phenomenology of shame and its associated features such as activators, facial displays, shame complexes, and defending scripts. The role shame plays in low self-esteem and in various forms of psychopathology such as eating disorders, addictions, depression, and anxiety disorders is elucidated.

In the first of the tools in the shame unit, participants have to identify an early shame scene from childhood, its associated affects, and how that shame scene influences them now. The second tool involves identifying shame binds and describing the experience of having these shame binds activated. The tools are designed to increase the awareness of the impact of shame on the self, and to understand the role shame plays in one's life.

Unit III. Identity: The Self's Relationship With the Self

Identity is defined as the self's relationship with the self. Within the self, there is an inner relationship between an observer part of the self or subject, and the observed part of the self or object. Just as interpersonal relationships with others have an affective component, so does the <u>intrapsychic</u> relationship with oneself. The affective quality of this inner relationship is based on internalized affect embedded in scenes that derived from early interpersonal relationships. According to how the self was treated by others, now the self in turn treats the self. Thus, identity develops through a combination of magnified scenes and identity scripts, which develop in response to those scenes. These scripts reproduce shame. Identity scripts take the form of inner voices and may be activated in situations such as looking in the mirror, making a mistake or blunder, or meeting strangers. A total of twelve situations likely to elicit identity scripts are explored. Three self-shaming identity scripts which re-produce shame are highlighted: comparison making, self-blame, and self-contempt.

Participants are also taught how to access earlier scenes through imagery. Development is conceived of as a series of emotional phases, much as rings of a growth form the trunk of a tree, which are each an important part of the self. Reparenting imagery is used to identify and access these scenes from the past, so they can be brought to full awareness.

The first set of identity tools aims to identify and change inner self-shaming identity scripts. This involves attending to inner voices and becoming conscious of what they are saying, and then identifying what type of self-shaming script is operating. Participants are urged identify whose voice is speaking the words, and also identify the

scenes in which the voice originated. Inner self-shaming scripts are then re-scripted by introducing a trusted older figure in the original scene to provide what was needed. Self-shaming identity scripts are changed to reflect enjoyment and excitement of the self. The second tool, reparenting imagery, is used to transform shame or other traumatic scenes from the past. The imagery involves the adult self comforting the hurt inner child, and doing whatever the child needs to feel warm, safe, and secure. By changing the scene is this way, the scene is fused with new enjoyment affect and thereby transformed, and then stored back in memory as a changed scene.

Unit IV: Affect Regulation and Release

Participants are first taught the processes of differentiated owning, which involves a focusing of attention inwards in order to expand conscious awareness. Next they are taught the process of detachment which involves learning how to let go of negative affect. After participants experience, name, and own their affects, they must also find ways to release or detach from their negative affect. It is generally more beneficial to have several different methods for detaching from negative affect because flexibility and adaptability are thereby increased. Detaching from negative affect prevents negative affect from spiraling and backing up, leading to stress.

The consulting with self tool allows individuals to focus inward and to determine what affects and other inner experiences (drives, interpersonal needs) are most prevalent in their life and to distinguish among them. Participants are asked to do this for 10 minutes every day. The second set of tools are detachment tools, which are used to release negative affect. These include writing, humor, meditation, refocusing attention, imagery, and self-observation.

Unit V: Interpersonal Relationships

The final unit takes these concepts and applies them to developing interpersonal skills. There are three major sections to this unit. First, participants apply the concepts of affect and scene for the purpose of identifying what they need in relationships. Kaufman's theory of interpersonal needs (1992, 1996) forms the basis for these needs. Second, participants are urged to adopt a detached stance or an objective point of view in order to consciously observe the other person. Again, the focus is on the <u>specific</u> behaviors of the other, including facial expressions. Third, participants are asked to match their expectations and needs to what the other person can actually provide. Special focus is given to the interplay among power, shame, and intimacy.

There is one relationship tool for this unit which consists of three steps. Participants are asked to apply these three concepts to one important relationship in their life. The relationship can be with a family member, friend, or romantic partner. The first step is to identify their own specific needs and expectations in the relationship selected. Second, the other person must be objectively observed and the quality of the match between expectations and reality determined. Third, the relationship is examined for power, shame, and intimacy.

The common element running through all units of the program is a focus on <u>increasing conscious awareness</u> of affects and related inner experiences so that the participants will have more inner control, more choices, and more power in their everyday lives. Conscious awareness of affect is the foundation for achieving this.

Prior Research

There have been four empirical studies conducted on Kaufman's psychoeducational curriculum, Affect and Self-Esteem (ASE). Readett (2001) evaluated the ASE curriculum in terms of four constructs: polarity theory, depression, the ability to differentiate shame, and openness to feelings. Results showed that subjects in the Affect and Self Esteem curriculum scored significantly higher on the humanistic ideological position of the Polarity Scale compared with a control group. Results also indicated there were significant reductions in depression and internalized shame, and a significant increase in openness to feelings.

Mayne (1992) investigated the influence of both the ASE curriculum and a cognitive-behavioral intervention for parents on pathogenesis, compared with a control group of undergraduate psychology students. Pathogenesis, defined as the degree to which one uses others to satisfy one's own needs was measured by scoring responses to the TAT as either pathogenic, neutral, or benign. Participants in all conditions were assessed pre and post for pathogenesis. Responses where a dominant person fails to take into account the needs of a dependent person, or responses that deal with themes of loneliness, dependency, and helplessness, all are scored as pathogenic. Stories with themes of potency, helpfulness, and success are scored as benign. Stories that are neither pathogenic nor benign are considered to be neutral. Results indicate that pathogenesis decreased in both experimental groups, but for different reasons. Participants in the ASE curriculum showed a decrease in pathogenic responses, while participants in the

Mayne concluded that there was support for the ASE curriculum decreasing pathogenesis.

Meola (1988) assessed the ASE curriculum both pre and post for changes in four variables compared with a control group: self-esteem, depression, state anxiety, trait anxiety, and identification of shame. Results indicated there was a significant reduction in trait anxiety and a significant increase in the awareness of shame, while no changes were found for self-esteem, depression, and trait anxiety. Meola concluded that no effects for self-esteem and depression were found due to a lack of sensitivity of the measures used. In addition a qualitative questionnaire was administered to determine the frequency of the use of each of the tools and their perceived effectiveness. Results indicated participants used the tools fairly regularly and had a solid perception that the tools were effective.

Finally, Rosenberg (1984) conducted a study of the ASE curriculum to determine if change occurred in both locus of control and the ability to differentiate internal experience. Locus of control is an important variable related to beliefs about the source of power and change in one's environment. Individuals with an external locus of control believe they are largely at the mercy of external events, whereas persons with an internal locus of control tend to believe that their own actions are largely responsible for controlling events. Rosenberg hypothesized that locus of control would become significantly more internal in the experimental condition. This hypothesis was not supported.

This study also tested whether individuals would be able to differentiate their internal experience better as a result of participating in the ASE curriculum. Results

indicate that subjects were significantly better able to differentiate affect and there was a significant increase in the frequency of positive inner dialogue patterns. However, no effects were found regarding a predicted decrease in the frequency of negative dialogue patterns nor with replacing negative inner dialogues with positive dialogues, and negative effects were found for the ability to differentiate inner dialogue patterns. In addition, the tools were again assessed for perceived effectiveness and frequency of use. Participants rated overall effectiveness of the tools as very effective, with a moderate frequency of their use.

On the basis of these studies, there is evidence to support the hypothesis that participating in the ASE curriculum will lead to reduced levels of alexithymia. Previous research on the program indicates support for an increase in the ability to differentiate affect, which is an important component of alexithymia. In addition, previous research regarding the impact of the curriculum on psychopathology is mixed, having demonstrated a reduction in anxiety, but no change in depression. Thus, measuring several dimensions of psychopathology is warranted in order to assess the impact of the curriculum on a wide range of psychopathology.

Chapter 2

DESIGN OF THE CURRENT STUDY

This is a longitudinal study investigating whether a 15-week psycho-educational program designed to improve self-esteem and overall psychological functioning will lead to a reduction in alexithymia levels upon completion of the program. A course on Humanistic Psychology was used as a control group. This study employed an experimental design with a control group and pre- and post- measures. Two measures of alexithymia were used, the 20-item Toronto Alexithymia Scale (TAS-20) and the Levels of Emotional Awareness Scale (LEAS). Recently, the TAS-20 has been criticized because it is a self-report measure that is used on a population which, by definition, has difficulty identifying and labeling inner experiences (Lane, Sechrest, et al., 1998). The skills that are necessary for accurate self-appraisal may be missing in individuals who do indeed have high alexithymia levels. Lane, Sechrest, et al. (1998) have suggested that a performance measure of alexithymia, which requires subjects to label their feelings, may be preferable to a self-report measure. LEAS is not a self-report measure, but rather is a performance-based measure of the cognitive complexity of the awareness of emotion. Therefore, as LEAS scores increase, the strength of the negative correlation between the LEAS and the TAS-20 should also increase. Perhaps this helps to explain why research thus far indicates only a weak negative correlation ($\underline{r} = -.19$) between the TAS-20 and the LEAS (Lane, 2000).

The current study was the first to use both the TAS-20 or the LEAS to measure alexithymia in a treatment context. In addition, global psychopathology was measured

using the Brief Symptom Inventory (BSI), and a comparison was made between the TAS-20 and the LEAS to determine which predicts changes in global psychopathology more accurately.

HYPOTHESES

This study had three main hypotheses.

First, it was predicted that students who participate in a psycho-educational program designed to improve self-esteem will show decreases in alexithymia and psychopathology at the end of the course as measured by the TAS-20, the LEAS, and the BSI in comparison to a control group.

Second, it was predicted that the change in LEAS scores between the beginning and the end of the course will be a better predictor of changes in BSI scores than changes in the TAS-20.

Third, it was predicted that the TAS-20 and the LEAS will be more strongly negatively correlated as LEAS scores increase.

Chapter 3

METHOD

Subjects

The subjects included 101 participants enrolled in two undergraduate psychology courses at Michigan State University. The experimental group consisted of students enrolled in PSY 325, Affect and Self-Esteem. There were 37 students in the experimental group that were sampled in the first week of class, 27 of whom were also tested at the end of the 15-week class. The control group consisted of students enrolled in PSY 488: Humanistic Psychology. There were 64 students in the control group that were sampled in the first week of class, 37 of whom were also tested at the end of the 15-week class, 37 of whom were also tested at the end of the 15-week class.

Measures

<u>Toronto Alexithymia Scale (TAS-20)</u>. The TAS-20 is a 20-item self-report questionnaire designed to assess alexithymia (Bagby, Parker, et al., 1994). The 20 items are rated on a 5-point scale, where 1 = strongly disagree and 5 = strongly agree. The TAS-20 consists of the following three subscales: 1) difficulty identifying feelings, i.e., "I am often confused about what emotion I am feeling", 2) difficulty describing feelings, i.e., "It is difficult for me to reveal my innermost feelings, even to close friends", and 3) externally oriented thinking, i.e., "I prefer talking to people about their daily activities rather than their feelings." The internal consistency reliability for the three factors were found to be .78, .75, and .66 respectively in a large sample ($\underline{N} =$ 965) of Canadian undergraduates. A test-retest reliability of .77 for the total scale score, with a 3-week

interval between testing, was found using a separate and smaller sample ($\underline{N} = 72$) of Canadian college undergraduates. The TAS-20 also demonstrates good convergent validity. In a sample of ($\underline{N} = 85$) Canadian undergraduates, the TAS-20, as predicted, was negatively correlated with measures of psychological mindedness ($\underline{r} = -.68$, $\underline{p} < .01$), need for cognition ($\underline{r} = -.55$, $\underline{p} < .01$), openness for experience ($\underline{r} = -.49$, $\underline{p} < .01$), and positive emotions ($\underline{r} = -.36$, $\underline{p} < .01$) and positively correlated with a measure of neuroticism ($\underline{r} = .27$, $\underline{p} < .01$) (Bagby, Taylor, & Parker, 1994). Using this same sample, the TAS-20 was found to have adequate discriminant validity as it was not significantly related to either measures of agreeableness ($\underline{r} = .09$) or conscientiousness ($\underline{r} = .21$) (Bagby, Taylor, et al., 1994).

Levels of Emotional Awareness Scale (LEAS). The LEAS is designed to measure awareness and differentiation of emotional states in both self and other. Participants are presented with 20 scenes of vignettes, which are described in 2 to 4 sentences, and are asked to describe the anticipated feelings of themselves and another person depicted in the vignette. More specifically, participants are presented with one vignette per page, which is followed by two questions: "How would you feel?" and "How would the other person feel?" Participants are instructed to write their answers on the remainder of the page, using as much or as little space on the page as needed. Responses for each vignette are scored on a scale from 0 to 5, where 0 indicates the lowest level of emotional awareness and 5 indicates the highest, resulting in a maximum total score of 100.

For each vignette, there is a separate score for self and other. The lowest level (Level 0) is scored when the subject replies with a thought and not a feeling. Level 1

reflects an awareness of physiological sensations such as, "I feel tired." Level 2 consists of action tendencies, "I'd feel like punching the wall" or vague words typically used in other contexts, "I'd feel bad." Level 3 responses contain one specific word for affect, "I'd feel happy." Level 4 responses are comprised of two or more Level 3 responses. To get the highest score of 5, there must be a Level 4 response for <u>both</u> self and other. Thus, for each of the 20 items, there is a <u>self</u> score, an <u>other</u> score, and a <u>total</u> score. The total score is calculated by taking the higher of either the self or other score. For example, if a participant scored a 3 on the self score and a 1 on the other score, their total score would be a 3. The only exception occurs in the case where both self and other scores are Level 4. In this case, a total score of 5 is given. A glossary of emotional words is provided to guide scoring (Lane, et al., 1990).

The interrater reliability of the LEAS is .84. and the internal consistency reliability of the LEAS is .81 (Lane, et al., 1990). The test-retest reliability of the LEAS is not available (Lane, Reiman, et al., 1998). Convergent validity was demonstrated by correlations with two measures of cognitive maturity, a measure of openness to experience, and to a full range of emotions. Discriminant validity was demonstrated by a lack of correlation with any specific emotion and with a measure of repression. In addition, the LEAS does not correlate with the number of words used in LEAS responses, indicating that the LEAS is not merely a measure of verbal productivity (Lane, et al., 1990).

<u>Brief Symptom Inventory (BSI).</u> The BSI is a self-report symptom inventory with 53 items designed to assess psychopathology. Each item is rated on a five point Likert scale from 0 to 4, where 0 indicates no distress and 4 suggests extreme distress.

The BSI is currently widely used for both research and clinical assessment with such diverse topics as cancer patients, psychoneuroimmunology, pain assessment and management, therapeutic intervention, HIV research, hypertension, and student mental health (Derogatis, 1993). The BSI has a total of nine symptom dimensions including: Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Phobic Anxiety, Hostility, Paranoid Ideation, and Psychoticism. The internal consistency reliability for the nine factors were found to be very good, ranging from .71 on the Psychoticism dimension to .85 on the Depression dimension in a sample (N = 719) of psychiatric outpatients (Derogatis, 1993). The BSI also demonstrates excellent test-retest reliability. The stability coefficient for the Global Severity Index was found to be .90, and coefficients for the nine BSI dimensions ranged from .69 for the Somatization dimension to .91 for the Phobia dimension, in a sample of nonpatients (N = 60) tested across a two-week interval. The dimensions of the BSI also show impressive convergent validity with major measures of psychopathology, including the MMPI and the SCL-90-R (Derogatis, 1993). In a sample of symptomatic volunteers (N = 209), the Hostility dimension of the BSI was positively correlated (r = .42) with the Depression scale of the MMPI.

Procedure

Students were measured during the first week and last week of the semester for both the experimental and the control conditions. Participants were given a packet including: an informed consent form, a socio-demographic questionnaire, the TAS-20, the LEAS, and the BSI. Sociodemographic information included age, gender, and SES so as to rule out possible confounding variables between the groups. The order of

presentation of the TAS-20, LEAS, and BSI was counterbalanced so as to rule out any response bias. Subjects wrote their name on the informed consent form where it was matched with a subject ID number. Informed consent forms were separated from the packet, and stored separately in a locked cabinet, so as to ensure participant confidentiality. The subject ID number was be used to identify all other forms in the packet. Administration time took approximately one hour. Participants were entered into a raffle for \$50 dollars for students who completed both packets of questionnaires pre and post in each class, in order to maximize participation and increase subject retention rates. Data Analysis

First, a repeated measures MANOVA was used to determine if there was a significant change in TAS-20, LEAS, and BSI scores in the experimental group compared with the control group.

Second, regressions were conducted to determine if the two measures of alexithymia, the TAS-20 and the LEAS, independently predicted significant changes in BSI scores compared with one another.

Third, the hypothesis that the correlation between the TAS-20 and the LEAS will become increasingly more strongly negative as LEAS scores increase was tested.

Chapter 4

RESULTS

Data Preparation and Data Cleaning

The scales for the TAS-20 and the LEAS were calculated by summing the items for each scale score. Missing values were hand scored and were prorated by multiplying the summed score of items by the total number of items in each scale and then dividing by number of items for each scale that were completed. The scales for the BSI were calculated by summing the items for each scale score and then by dividing by the number of items for each scale score that were completed. Missing values were hand scored and were also calculated by summing the items for each scale score and then by dividing by the number of items for each scale score that were completed.

Data Analyses

<u>Hypothesis 1.</u> A repeated measures MANOVA was used to determine if there were significant changes in TAS-20, LEAS, and BSI scores from pre to post testing in the experimental group compared with the control group (see Table 1). As predicted, LEAS scores increased significantly (p < .002) in the experimental group compared with the control group, indicating that the subjects in the ASE curriculum increased in their cognitive awareness and differentiation of their emotional states (see Table 2). Significant changes were not found in either TAS-20 scores or global BSI scores. However, contrary to what was predicted, there was a significant increase (p < .001) in the Obsessive-Compulsive dimension of the BSI in the experimental group compared with the control group. Also contrary to prediction, there was a significant effect for the global BSI scores X group interaction (p < .020). There were also significant interactions for a number of dimensions of the BSI including: Interpersonal Sensitivity X group (p < .032), Anxiety X group (p < .007), Hostility X group (p < .002), Paranoid X group (p < .033), and Psychoticism X group (p < .015). In each of these interactions, the trend was toward increasing scores in the experimental group and decreasing scores in the control group, as indicated by differences in means (see Table 1).

<u>Hypothesis 2.</u> Regressions were conducted to determine if the TAS-20 and the LEAS independently predicted significant changes in the BSI (see Table 3). Results indicated that the change in TAS-20 scores significantly predicted changes in global BSI scores (p < .012), suggesting that changes in a self-report measure of alexithymia predicted changes in psychopathology. Change in LEAS scores was not found be a significant predictor of changes of global BSI scores.

<u>Hypothesis 3.</u> The hypothesis that the correlation between the LEAS and the TAS-20 will become more strongly negative as LEAS scores increase was tested. This hypothesis was supported. When LEAS scores were below 65, the correlation between LEAS and TAS-20 scores was mildly positive ($\mathbf{r} = .134$), indicating that when LEAS scores are low, they are positively correlated with TAS-20 scores (see Figure 1). When LEAS scores were equal to or above 65, the correlation between LEAS and TAS-20 scores are high, they are negatively correlated with TAS-20 scores are high, they are negatively correlated with TAS-20 scores are high, they

Chapter 5

DISCUSSION

The first hypothesis predicted that alexithymia and psychopathology levels would significantly decrease in subjects who participated in the ASE curriculum compared with a control group. Results for changes in alexithymia were mixed. LEAS scores increased significantly in the experimental/ASE group, but the TAS-20 did not change significantly. The LEAS is a performance-based measure of the cognitive complexity and differentiation of emotions in self and other. The TAS-20 is a self-report measure of alexithymia that presumably measures one's self-perception of one's ability to identify and describe feelings. Results indicate that subjects' self-perception of their ability to identify change. Thus, subjects' actual ability to label and describe feelings as measured by the LEAS improved, but their self-perception of their ability to label and describe their feelings as measured by the TAS-20 did not significantly change as measured by the TAS-20 did not improve.

It was also predicted that psychopathology would decrease in the experimental/ASE group compared with the control group. This hypothesis was not supported. Results for psychopathology did not show significant main effects for changes in global psychopathology, indicating that overall psychopathology levels did not significantly decrease in subjects who participated in the ASE curriculum. However, significant main effects for changes in the Obsessive-Compulsive dimension of the BSI were found, but in the opposite direction than predicted. Obsessive-Compulsive scores increased significantly in the experimental group compared with the control group. The BSI Obsessive-Compulsive dimension is comprised of six items which indicate

difficulties with cognitive functioning including: "trouble remembering things," "feeling blocked in getting things done," "having to check and double-check what you do," "difficulty making decisions," "your mind going blank," and "trouble concentrating." The BSI also showed significant interactions for the Interpersonal Sensitivity, Anxiety, Hostility, Paranoid Ideation, and Psychoticism dimensions, as well as for global psychopathology. The direction of all significant interactions showed an increase in group means for the experimental group and a decrease in group means for the control group.

Contrary to hypothesis, these results demonstrate a significant trend in which psychopathology decreased in the control group compared with the experimental group. One explanation for these results is that the BSI is a measure of state psychopathology, which may not detect changes in trait psychopathology. The BSI asks participants to determine their symptomatology based on the past seven days, and thus is a state measure. When Meola (1988) used measures of state and trait anxiety in her research on the ASE curriculum, she found a significant reduction in trait anxiety only. Another possible explanation is that the control class may have had some influence on a reduction of state psychopathology scores, owning to the fact that subjects in this class spent a significant amount of time engaged in laughter and play. Perhaps play behavior and laughter serves to reduce state measures of psychopathology such as the BSI.

The second hypothesis predicted that the changes in LEAS scores would be a better predictor of changes in BSI scores than the TAS-20. This hypothesis was not supported. Changes in TAS-20 scores did significantly predict changes in the BSI, but there was no significant effect of changes in LEAS scores on the BSI. It was

hypothesized that the LEAS would be a better predictor of psychopathology than the TAS-20 because the TAS-20 is a self-report measure, and individuals who have difficulty identifying and describing inner events may also have difficulty accurately self-reporting their perception of their ability to identify and describe their feelings. Both the TAS-20 and the BSI are self-report measures. Perhaps these results can be accounted for by a self-report bias, with individuals tending to self-report high on all self-report measures.

The third hypothesis predicted that the TAS-20 and the LEAS would be more strongly negatively correlated as LEAS scores increased. There was support for this hypothesis because the correlation between LEAS and TAS-20 scores was moderately positive when LEAS scores were below 65 whereas the correlation was slightly negative for LEAS scores above 65. Lane (2000) has argued that the TAS-20 may be problematic because it is a self-report measure of alexithymia. According to Lane (2000), in order to accurately self-report one's awareness of one's feelings on the TAS-20, one must possess the same ability that is being tested. If one is lacking in this ability, one's self-report is likely to be inaccurate.

On the basis of Lane's (2000) argument, it was predicted that as LEAS scores increased, the correlation between the LEAS and TAS-20 would become more strongly negatively correlated. Results indicated that subjects' self-perception of their ability to identify and describe their feelings does not remain constant. Instead subjects evaluate their ability to identify and describe their feelings more positively than their actual ability to cognitively describe their feelings indicates, as measured by the LEAS. Thus, as the ability to cognitively label and differentiate emotion increases, the correlation between the LEAS and the TAS-20 becomes more negative. This lends support to the hypothesis

that it may become more difficult for individuals who already have difficulty identifying and describing their feelings to accurately self-report their feelings of alexithymia on the TAS-20.

The results from this study must be interpreted with caution because of the limitations of this study. First, the number of subjects who participated in the study was low. Greater numbers of subjects would have increased the power to detect significant differences, and may have shed more light on some of the trends found in the current sample. Second, approximately one third of the subjects at pre-testing dropped out and were not post-tested. Because there is no way to measure the characteristics of these subjects, there is no way to determine if there was a systematic pattern related to drop outs, nor what influence the drop outs had on the current sample. Third, the BSI is a clinical measure used to assess clinical populations. In this study the BSI was used on a university undergraduate population, where rates of psychopathology are relatively low compared to clinical samples, and thus the BSI may not be sensitive enough to be able to detect psychopathology in a normal sample. Fourth, both the TAS-20 and the BSI rely on the self-report of participants. Research has shown that self-report measures fail to distinguish between defensive deniers, who underreport their distress or difficulties, and psychologically healthy individuals (Shedler, Mayman, & Manis, 1993). Finally, the events of the terrorist attacks of Sept. 11, 2001, while presumably affecting both groups equally, may have differentially influenced one group in significant but unknowable ways.

Future research should address these limitations. To increase sample size, pretesting should not occur until the second week of classes, when participant enrollment

stabilizes. In addition, the prize for winning the raffle should increase from \$50 to \$100 because many participants indicated they came from households with an income in excess of \$70,000, and \$50 may not be sufficient to sustain interest in the study.

There are also major concerns about the nature of the alexithymia construct itself. According to Bucci (1997), alexithymia occurs when there is disconnection between the nonlinguistic nonsymbolic, nonlinguistic symbolic, and linguistic symbolic dimensions of consciousness. Kaufman (1996) also argues that for maximal psychotherapeutic change to occur, language, affect, and imagery must be reconnected to one another in conscious awareness. This is also consistent with clinical work. Clients may be extremely facile in their ability to cognitively label their feelings, but if this is not linked together with actually experiencing feelings and also actively creating imagery to reflect internal states, then usually there is little improvement in these clients. Neither the LEAS nor the TAS-20 measures all three domains of consciousness. Therefore, from an affect theory perspective, neither one of these instruments is likely to be a completely valid measure of the alexithymia construct.

In order to more thoroughly understand and assess alexithymia, researchers will need to first be able to assess each of the three domains of consciousness (affect, imagery, language), and then will need to be able assess the degree to which those three domains of consciousness are related to each other. The difficulty of this task becomes apparent when we consider the current ability to assess affect in general, which is still limited. Yet, until researchers are able to measure these dimensions of affect with reliable precision, they will not be able to fully grasp its relationship to alexithymia.

In summary, subjects' actual ability to cognitively label feelings as measured by the LEAS significantly improved as predicted in the ASE curriculum compared with a control group. However, subjects' self-perception of their ability to identify and describe their feelings as measured by the TAS-20 did not change significantly. In addition, psychopathology scores did not decrease as predicted in the ASE group, but instead there was a significant trend for a decrease in psychopathology in the control group compared with the experimental group. Finally, as LEAS scores increased, the correlation between the LEAS and the TAS-20 became increasingly negative as predicted. Thus, it seems likely that the LEAS and the TAS-20 are not measuring the same construct, and this study raises serious concerns about their validity as measures of the alexithymia construct. Future research conducted on alexithymia should incorporate both the LEAS and the TAS-20 in an effort to further elucidate the relationship between these two measures. APPENDICES

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

APPENDIX A

Table 1

Variable	Group	Time 1		Time 2	
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
TAS-20	1	45.162	11.258	45.519	11.362
	2	42.531	10.474	43.105	10.363
LEAS	1	65.297	7.652	71.963	9.757
	2	63.016	7.915	61.631	7.947
BSI	1	.834	.664	.985	.713
	2	.893	.609	.767	.615
Somatiz	1	.510	.786	.626	.694
	2	.589	.627	.491	.597
Obsess	1	1.117	.823	1.506	1.030
	2	1.122	.787	1.282	.822
Sensitiv	1	1.155	1.180	1.269	1.102
	2	1.348	1.042	.929	.987
Depress	1	1.014	1.031	1.090	.987
	2	1.083	.926	.964	1.027
Anxiety	1	.865	.765	1.186	.933
	2	.935	.744	.756	.613
Hostilit	1	.741	.671	.862	.787
	2	.930	.821	.733	.830
Phobic	12	.432 .391	.635 .574	.427 .303	.613 .485
Paranoid	1	.930	.822	1.015	.898
	2	.892	.779	.700	.867

Means and Standard Deviations for Variables by Group across Time

Psychotic	1	.800	.739	.862	.889
	2	.858	.728	.595	.710

Group 1 = Experimental Group (ASE Curriculum)

Group 2 = Control Group
APPENDIX B

Table 2

Variable	SS	df	MS	F	Р
TAS-20	9.263	1	9.263	.254	.616
TAS-20 X Group	8.001	1	8.001	.220	.641
LEAS	283.205	1	283.205	10.928	.002**
LEAS X Group	338.713	1	338.713	13.070	.001***
BSI	.127	1	.127	1.260	.266
BSI X Group	.579	1	.579	5.736	.020*
Somatiz	.114	1	.114	.575	.451
Somatiz X Group	.212	1	.212	1.070	.305
Obsess	3.647	1	3.647	13.341	.001***
Obsess X Group	.276	1	.276	1.009	.319
Sensitiv	.148	1	.148	.502	.481
Sensitiv X Group	1.417	1	1.417	4.802	.032*
Depress	.05585	1	.05585	.161	.689
Depress X Group	.483	1	.483	1.394	.242
Anxietv	.615	1	.615	2.457	.122
Anxiety X Group	1.960	1	1.960	7.831	.007**
Hostilit	.02197	1	.02197	.156	.694
Hostilit X Group	1.505	1	1.505	10.669	.002**
Phobic	.05417	1	.05417	.396	.531
Phobic X Group	.03078	1	.0.078	.225	.637
Paranoid	.02437	1	.02437	.112	.739
Paranoid X Group	1.031	1	1.031	4.747	.033*

Repeated Measures MANOVA of TAS-20, LEAS, and BSI Scores

Psychotic	.105	1	.105	.552	.460
Psychotic X Group	1.205	1	1.205	6.316	.015*

*<u>p</u><.05 **<u>p</u><.01 ***<u>p</u><.001

APPENDIX C

Table 3

Summary of Regression Analyses for Change in TAS-20 and LEAS Scores Predicting

Changes	in	BSI	Scores
	_		

			· · · · · · · · · · · · · · · · · · ·		
Variable	b	se	bs	t	р
ChangeLEAS	.010	.007	.176	1.42	.159
ChangeTAS	.018	.007	.320	2.60	.012*

*<u>p</u><.05 **<u>p</u><.01 ***<u>p</u><.001

Figure 1

Scatterplot of TAS-20 and LEAS Scores Where LEAS Scores are Less Than 65 (r = .134)



Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

APPENDIX E

Figure 2

Scatterplot of TAS-20 and LEAS Scores Where LEAS Scores are Equal To or Greater Than 65 (r = -.017)



APPENDIX F

Informed Consent Form

MICHIGAN STATE UNIVERITY

CONSENT TO ACT AS A RESEARCH SUBJECT

You are invited to participate in a research study investigating emotion and psychological functioning. The study is comprised of three questionnaires that take approximately 1 hour to complete. The questionnaires will be administered during the first week and the last week of class. Participants who complete both sets of questionnaires will be eligible for \$50 to be given away during a raffle during the final administration session.

Your decision to participate in this study will have no effect on your grade for this class.

You may experience some minor discomfort as a result of examining your feelings and your psychological functioning.

You have certain rights as a participant in this experiment:

- 1. Even if you agree to participate, you have the right to change your mind and to *not* participate before the experiment begins, and to do so without penalty. Even if you agree to participate and have begun the experiment, you have the right to discontinue your participation at any time and for any reason, and to do so without explanation or penalty. If you decide to discontinue participation, all questionnaires and other information about you will be destroyed, and your name will be deleted from all records.
- 2. You have the right to confidentiality. Your privacy will be protected to the maximum extent allowable by law. To ensure this, you will be assigned a subject number, which will be the only way to identify you in any reports about the study. Only the experimenters will have access to files and lists that can be used to link your name with a subject number. Any publication resulting from this work will not identify you by name or circumstances that would allow your identity to be discovered.
- 3. It is unlikely that your participation in the study will elicit significant emotional distress or discomfort. However, if you do have any emotional concerns, you may contact Olin Health Center at (517) 353-4660 or the Counseling Center at (517) 355-8270.
- 4. If you wish further information regarding your rights as a research subject, you may contact the Office of the University Committee on Research Involving Human Subjects (UCRIHS), 246 Administration Building, Michigan State University, East Lansing, MI, 48824-1046, by telephoning or emailing the chairperson, David E. Wright at (517) 355-2180, and at UCRIHS@msu.edu.
- 5. After the entire study is completed, if you have any questions or if you want a written summary of the general results, you may contact the primary investigator, David Rose, M. A., at the Department of Psychology, 149 Snyder Hall, Michigan State University, East Lansing, MI, 48824, by leaving a message at either (517) 355-4599, or <u>rosedavi@msu.edu</u>, or Gershen Kaufman, Ph.D., at the MSU Counseling Center, 207 Student Services Building, Michigan State University, 48824, by leaving a message at either (517) 355-2310, or gershk@post.couns.msu.edu.

Please print and sign below to indicate that you have read and received a copy of the consent form and that you voluntarily agree to participate in this study.

Print Name Clearly

Signature of Participant

Date

Signature of Witness

Date

APPENDIX G

Demographic Questionnaire

	Subject #&					
	Subject Number					
	1. How many siblings do you have?					
2) Wh	nat are the last four digits of your MSU student ID?					
(# sibli (Do no	ings) & (last 4 digits ID) = Subject Number& of use addition with these numbers.)					
	Demographic Questionnaire					
	2. Age					
2.	Gender Male or Female (circle)					
3.	What is your estimated annual household income for family of origin? If you come from 2 or more households, average the incomes. (please check one)					
	Under \$19,999 per year					
	\$20,000 to \$34,999 per year					
	\$35,000 to \$49,999 per year					
	\$50,000 to \$69,999 per year					
	\$70,000 to \$100,000 per year					
	Above \$100,000 per year					

APPENDIX H

<u>TAS-20</u>

TAS-20

Instructions: We are studying emotions and are interested in your experiences. Please complete this questionnaire by circling the number which indicates your level of agreement or disagreement with each statement. Give only one answer for each statement.

G. 1		Neither		Otraca 1			
Strongly	Moderately Disagree	Disagree	Moderately A gree	Strongly			
l	2	3	4	5			
1. I am often confused about what emotion I am feeling.							
1	2	3	4	5			
2. It is difficult f	or me to find the r	ight words for my	feelings.				
1	2	3	4	5			
3. I have physica	al sensations that e	ven doctors don't	understand.				
1	2	3	4	5			
4. I am able to de	escribe my feeling	s easily.					
1	2	3	4	5			
5. I prefer to ana	5. I prefer to analyze problems rather than just describe them.						
1	2	3	4	5			
6. When I am up	set, I don't know	if I am sad, frighte	ened, or angry.				
1	2	3	4	5			
7. I am often puzzled by sensations in my body.							
1	2	3	4	5			
8. I prefer to just let things happen rather than to understand why they turned out that way.							
1	2	3	4	5			
9. I have feelings	s that I can't quite	identify.					
1	2	3	4	5			

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

10. Being in touch with emotions is essential.

1	2	3	4	5		
11. I find it hard to describe how I feel about people.						
1	2	3	4	5		
12. People tell n	ne to describe my f	feelings more.				
1	2	3	4	5		
13. I don't know	what's going on i	nside me.				
1	2	3	4	5		
14. I often don't	know why I am fe	eling angry.				
1	2	3	4	5		
15. I prefer talki	ng to people about	their daily activit	ies rather than the	ir feelings.		
1	2	3	4	5		
16. I prefer to w	16. I prefer to watch "light" entertainment shows rather than psychological dramas.					
1	2	3	4	5		
17. It is difficult	for me to reveal m	ıy innermost feeli	ngs, even to close	friends.		
1	2	3	4	5		
18. I can feel clo	ose to someone, ev	en in moments of	silence.			
1	2	3	4	5		
19. I find examinations of my feelings useful in solving personal problems.						
1	2	3	4	5		
20. Looking for	hidden meanings i	n movies or plays	distracts from the	ir enjoyment.		
1	2	3	4	5		

72

APPENDIX I

LEAS

INSTRUCTIONS

Please describe what you would feel in the following situations. The only requirement is that you use the word "feel" in your answers. You may make your answers as brief or as long as necessary to express how you would feel. In each situation there is another person mentioned. Please indicate how you think that other person would feel as well.

1. A neighbor asks you to repair a piece of furniture. As the neighbor looks on, you begin hammering the nail but then miss the nail and hit your finger. How would you feel? How would the neighbor feel?

2. You are walking through the desert with a guide. You ran out of water hours ago. The nearest well is two miles away according to the guide's map. How would you feel? How would the guide feel? 3. A loved one gives you a back rub after you return from a hard day's work. How would you feel? How would your partner feel?

4. You are running a race with a friend with whom you have trained for some time. As you near the finish line, you twist your ankle, fall to the ground, and are unable to continue. How would you feel? How would your friend feel? 5. You are traveling in a foreign country. An acquaintance makes derogatory remarks about your native country. How would you feel? How would your acquaintance feel?

6. As you drive over a suspension bridge you see a person standing on the other side of the guardrail, looking down at the water. How would you feel? How would the person feel?

7. Your sweetheart has been gone for several weeks but finally comes home. As your sweetheart opens the door... how would you feel? How would your sweetheart feel?

8. Your boss tells you that your work has been unacceptable and needs to be improved. How would you feel? How would your boss feel?

9. You are standing in line at the bank. The person in front of you steps up to the window and begins a very complicated transaction. How would you feel? How would the person in front of you feel?

10. You and your spouse are driving home from an evening out with friends. As you turn into your block you see fire-trucks parked near your home. How would you feel? How would your spouse feel?

11. You have been working hard on a project for several months. Several days after submitting it, your boss stops by to tell you that your work was excellent. How would you feel? How would your boss feel?

12. You receive an unexpected long-distance phone call from a doctor informing you that your mother has died. How would you feel? How would the doctor feel?

13. You tell a friend who is feeling lonely that she/he can call you whenever she/he needs to talk. One night she/he calls at 4:00 a.m. How would you feel? How would your friend feel?

14. Your dentist has told you that you have several cavities and schedules you for a return visit. How would you feel? How would the dentist feel?

15. Someone who has been critical of you in the past pays you a compliment. How would you feel? How would the other person feel?

16. Your doctor told you to avoid fatty foods. A new colleague at work calls to say that she/he is going out for pizza and invites you to go along. How would you feel? How would your colleague feel?

17. You and a friend agree to invest money together to begin a new business venture. Several days later you call the friend back only to learn that she/he changed her/his mind. How would you feel? How would the friend feel? 18. You sell a favorite possession of your own in order to buy an expensive gift for your spouse. When you gave him/her the gift, he/she asks whether you sold the possession. How would you feel? How would the spouse feel?

19. You fall in love with someone who is both attractive and intelligent. Although this person is not well off financially, this doesn't matter to you – your income is adequate. When you begin to discuss marriage, you learn that she/he is actually from an extremely wealthy family. She/he did not want that known for fear that people would only be interested in her/him for her/his money. What would you feel? How would she/he feel?

20. You and your best friend are in the same line of work. There is a prize given annually to the best performance of the year. The two of you work hard to win the prize. One night the winner is announced: your friend. How would you feel? How would your friend feel?

94

APPENDIX J

BSI

<u>BSI</u>

Instructions: Here is a list of problems that people sometimes have. Please read each one carefully, and circle the number that best describes HOW MUCH THAT PROBLEM HAS DISTRESSED OR BOTHERED YOU DURING THE PAST 7 DAYS INCLUDING TODAY. Circle only one number for each problem and do not skip any items. If you change your mind, erase your first mark carefully.

0 = Not At All

1 = A Little Bit

2 = Moderately

3 =Quite A Bit

4 = Extremely

HOW MUCH WERE YOU DISTRESSED BY:

1.	0	1	2	3	4	Nervousness or shakiness inside
2.	0	1	2	3	4	Faintness or dizziness
3.	0	1	2	3	4	The idea that someone else can control your thoughts
4.	0	1	2	3	4	Feeling other people are to blame for most of your troubles
5.	0	1	2	3	4	Trouble remembering things
6.	0	1	2	3	4	Feeling easily annoyed or irritated
7.	0	1	2	3	4	Pains in heart or chest
8.	0	1	2	3	4	Feeling afraid in open spaces or on the streets
9.	0	1	2	3	4	Thoughts of ending your life
10.	0	1	2	3	4	Feeling that most people cannot be trusted
11.	0	1	2	3	4	Poor appetite
12.	0	1	2	3	4	Suddenly frightened for no reason
13.	0	1	2	3	4	Temper outbursts that you could not control
14.	0	1	2	3	4	Feeling lonely even when you are with people
15.	0	1	2	3	4	Feeling blocked in getting things done
16.	0	1	2	3	4	Feeling lonely
17.	0	1	2	3	4	Feeling sad
18.	0	1	2	3	4	Feeling no interest in things
1 9 .	0	1	2	3	4	Feeling fearful
20.	0	1	2	3	4	Your feelings easily being hurt
21.	0	1	2	3	4	Feeling that people are unfriendly or dislike you
22.	0	1	2	3	4	Feeling inferior to other people
23.	0	1	2	3	4	Nausea or upset stomach
24.	0	1	2	3	4	Feeling that you are watched or talked about by others
25.	0	1	2	3	4	Trouble falling asleep
26.	0	1	2	3	4	Having to check and double-check what you do
27.	0	1	2	3	4	Difficulty making decisions
28.	0	1	2	3	4	Feeling afraid to travel on buses or other public transportation
29.	0	1	2	3	4	Trouble getting your breath
30.	0	1	2	3	4	Hot or cold spells
31.	0	1	2	3	4	Having to avoid certain things, places, or activities because they frighten you
32.	0	1	2	3	4	Your mind going blank
33.	0	1	2	3	4	Numbness or tingling (cramps) in parts of your body
34.	0	1	2	3	4	The idea that you have committed sins you should be punished for
35.	0	1	2	3	4	Feeling hopeless about the future
36.	0	1	2	3	4	Trouble concentrating
37.	0	1	2	3	4	Feeling weak in parts of your body

38.0	1	2	3	4	Feeling tense or keyed up
39. 0	1	2	3	4	Thoughts of death or dying
40. 0	1	2	3	4	Having urges to beat, injure, or harm someone
41.0	1	2	3	4	Having urges to break or smash things
42. 0	1	2	3	4	Feeling very self-conscious with other people
43.0	1	2	3	4	Feeling uneasy in crowds, such as shopping or at a movie
44. 0	1	2	3	4	Never feeling close to another person
45.0	1	2	3	4	Spells of terror or panic
46. 0	1	2	3	4	Getting into frequent arguments
47.0	1	2	3	4	Feeling nervous when you are left alone
48.0	1	2	3	4	Others not giving you proper credit for your work and achievements
49. 0	1	2	3	4	Feeling so restless you couldn't sit still
50.0	1	2	3	4	Feelings of worthlessness
51.0	1	2	3	4	Feeling that people will take advantage of you if you let them
52. 0	1	2	3	4	Feelings of guilt
53. 0	1	2	3	4	The idea that something is wrong with your mind

BIBLIOGRAPHY

Abramson, L., McClelland, D. C., Brown, D., & Kelner, S. J. (1991). Alexithymic characteristics and metabolic control in diabetic and healthy adults. <u>Journal of Nervous</u> and <u>Mental Diseases</u>, 179, 490-494.

Apfel-Savitz, R., Silverman, D., & Bennett, M. I. (1977). Group psychotherapy of patients with somatic illnesses and alexithymia. <u>Psychotherapy and Psychosomatics</u>, 28, 323-329.

Bagby, R. M., Parker, J. D. A., & Taylor, G. J. (1994). The twenty-item Toronto Alexithymia Scale - I. Item selection and cross-validation of the factor structure. <u>Journal of Psychosomatic Research</u>, 38, 23-32.

Bagby, R. M., Taylor, G. J., & Parker, J. D. A. (1994). The twenty-item Toronto Alexithymia Scale - II. Convergent, discriminant, and concurrent validity. <u>Journal of Psychosomatic Research</u>, 38, 33-40.

Berenbaum, H., & James, T. (1994). Correlates and retrospectively reported antecedents of alexithymia. <u>Psychosomatic Medicine</u>, 56, 353-359.

Berenbaum, H., & Prince, J. P. (1994). Alexithymia and the interpretation of emotion-relevant information. <u>Cognition and Emotion</u>, 8, 231-244.

Beresnevaite, M. (2000). Exploring the benefits of group psychotherapy in reducing alexithymia in coronary heart disease patients: A preliminary study. Psychotherapy and Psychosomatics, 69, 117-122.

Bucci, W. (1997). Symptoms and symbols: a multiple code theory of somatization. <u>Psychoanalytic Inquiry, 17, 151-172</u>.

Buck, R. (1988). Human motivation and emotion. New York: John Wiley & Sons.

Buck, R. (1994). The neuropsychology of communication: Spontaneous and symbolic aspects. Journal of Pragmatics, 22, 265-278.

Campos, A., Chiva, M., & Moreau, M. (2000). Alexithymia and mental imagery. <u>Personality and Individual Differences</u>, 29, 787-791.

Clerici, M., Albonetti, S., Pupa, R., Penata, G., & Invenizzi, G. (1992). Alexithymia and obesity. <u>Psychotherapy and Psychosomatics</u>, 57, 88-93.

Cox, B. J., Kuch, K., Parker, J. D. A., Shulman, I.D., & Evans, R. J. (1994). Alexithymia in somatoform disorder patients with chronic pain. <u>Journal of</u> <u>Psychosomatic Research</u>, 38, 523-527. Crick, N. R., & Dodge, K. A. (1996). Social information-processing mechanisms in reactive and proactive aggression. <u>Child Development</u>, 67, 993-1002.

de Groot, J. M., Rodin, G., & Olmsted, M. P. (1995). Alexithymia, depression, treatment outcome in bulimia nervosa. <u>Comprehensive Psychiatry</u>, 36, 53-60.

Demos, V. E. (1995). <u>Exploring affect: The selected writings of Silvan S.</u> <u>Tomkins.</u> Cambridge: Cambridge University Press.

Derogatis, L. R. (1993). <u>BSI: Brief symptom inventory, administration scoring</u> and procedures manual. Baltimore, MD: Clinical Psychometric Research.

Dewaraja, R., & Sasaki, Y. (1990). A left to right hemisphere callosal transfer deficit of nonlinguistic information in alexithymia. <u>Psychotherapy and Psychosomatics</u>, 54, 201-207.

Dinsmore, B. D., & Mallinckrodt, B. (1996). Emotional self-awareness, eating disorders, and racial identity attitudes in African-American women. Journal of Multicultural Counseling and Development, 24, 267-277.

Ekman, P. (1971). Universals and cultural differences in facial expressions of emotion. In J. K. Cole (Ed.), <u>Nebraska symposium on motivation</u>, Vol. 19, 207-283.

Friedlander, L., Lumley, M.A., Farchone, T., & Doyal, G. (1997). Testing the alexithymia hypothesis: Physiological and subjective responses during relaxation and stress. <u>The Journal of Nervous and Mental Disease</u>, 185, 233-239.

Fukunishi, I., Saito, S., & Ozaki, S. (1992). The influence of defense mechanisms on secondary alexithymia in hemodialysis patients. <u>Psychotherapy and Psychosomatics</u>, <u>57</u>, 50-56.

Fukunishi, I., Sei, H., Morita, Y., & Rahe, R. H. (1999). Sympathetic activity in alexithymics with mother's low care. Journal of Psychosomatic Research, 46, 579-589.

Goleman, D. (1995). Emotional intelligence. New York: Bantam Books.

Greenberg, L. S., Rice, L. N., & Elliott, R. (1993). <u>Facilitating emotional change:</u> <u>The moment-by-moment process.</u> New York: Guilford.

Henry, J. P., Haviland, M. G., & Cummings, M. A. (1992). Shared neuroendocrine patterns of posttraumatic stress disorder and alexithymia. <u>Psychosomatic Medicine</u>, 54, 407-415.

Honkalampi, K., Saarinen, P., Hintikka, J., Virtanen, V., & Viinamaki, H. (1999). Factors associated with alexithymia in patients suffering from depression. <u>Psychotherapy</u> and <u>Psychosomatics</u>, 68, 270-275. Hyer, L., Woods, M. G., Summers, M. N., Boudewyns, P., & Harrision, W. R. (1990). Alexithymia among Vietnam veterans with posttraumatic stress disorder. Juournal of Clinical Psychiatry, 51, 243-247.

Jula, A., Salminen, J., K., & Saarijarvi, S. (1999). Alexithymia: A facet of essential hypertension. <u>Hypertension</u>, 33, 1057-1061.

Kaufman, G. (1992). <u>Shame: The power of caring</u> (3rd Ed.). Rochester, VT: Schenkman Books.

Kaufman, G. (1996). <u>The psychology of shame: Theory and treatment of shame-based syndromes</u> (2nd Ed.). New York: Springer.

Kaufman, G., & Raphael, L. (1991). <u>Dynamics of power: Fighting shame and</u> <u>building self-esteem (2nd Ed.)</u>. Rochester, VT: Schenkman Books.

Kaufman, G., & Raphael, L. (1996). <u>Coming out of shame: Transforming gay and lesbian lives</u>. New York: Doubleday.

Kauhanen, J., Kaplan, G. A., Cohen, R. D., Julkunen, J., & Salonen, J. T. (1996). Alexithymia and risk of death in middle-aged men. Journal of Psychosomatic Research, 46, 541-549.

Kauhanen, J., Kaplan, G. A., Julkunen, J., Wilson, T. W., & Salonen, J. T. (1993). Social factors in alexithymia. <u>Comprehensive Psychiatry</u>, 34, 330-335.

Kooiman, C. G., Spinhoven, P., Trijsburg, R. W., & Rooijmans, H. G. M. (1998). Perceived parental attitude, alexithymia and defense style in psychiatric outpatients. <u>Psychotherapy and Psychosomatics</u>, 67, 81-87.

Krystal, H. (1982). Alexithymia and the effectiveness of psychoanalytic treatment. International Journal of Psychoanalytic Psychotherapy, 9, 353-378.

Krystal, H. (1988). <u>Integration and self-healing: Affect, trauma, alexithymia.</u> Ann Arbor: The Analytic Press.

Lane, R. D., Ahern, G. L., Schwartz, G. E., & Kaszniak, A. W. (1997). Is alexithymia the emotional equivalent of blindsight? <u>Biological Psychiatry</u>, 42, 834-844.

Lane, R. D., Quinlan, D. M., Schwartz, G. E., Walker, P. A., & Zeitlin, S. B. (1990). The levels of emotional awareness scale: A cognitive-developmental measure of emotion. Journal of Personality Assessment, 55, 124-134.
Lane, R. D., Reiman, E. M., Axelrod, B., Lang-Sheng, Y., Holmes, A., & Schwartz, G. E. (1998). Neural correlates of levels of emotional awareness: Evidence of an interaction between emotion and attention in the anterior cingulate cortex. Journal of Cognitive Neuroscience, 10, 525-535.

Lane, R. D., & Schwartz, G. E. (1987). Levels of emotional awareness: A cognitive-developmental theory and its application to psychopathology. <u>American</u> Journal of Psychiatry, 144, 133-143.

Lane, R. D., Sechrest, L., & Riedel, R. (1998). Sociodemographic correlates of alexithymia. <u>Comprehensive Psychiatry</u>, 39, 377-385.

Lane, R. D., Sechrest, L., Riedel, R., Shapiro, D. E., & Kaszniak, A. W. (2000). Pervasive emotion recognition deficit common to alexithymia and the repressive coping style. <u>Psychosomatic Medicine</u>, 62, 492-501.

Levant, R. F. (1998). Desperately seeking language: understanding, assessing, and treating normative male alexithymia. In W. S. Pollack & R. F. Levant (Eds.), <u>New</u> psychotherapy for men (pp. 35-56). New York: John Wiley & Sons.

Lumley, M. A., Asselin, L. A., & Norman, S. (1997). Alexithymia in chronic pain patients. <u>Comprehensive Psychiatry</u>, 38, 160-165.

Lumley, M. A., Mader, C., Gramzow, J. & Papineau, K. (1996). Family factors related to alexithymia characteristics. <u>Psychosomatic Medicine</u>, 58, 211-216.

Mallinckrodt, B., King, J. L., & Coble, H. M. (1998). Family dysfunction, alexithymia, and client attachment to therapist. <u>Journal of Counseling Psychology</u>, 45, 497-504.

Mayne, B. (1992). <u>Lessening pathogenesis: Preventative interventions.</u> Unpublished master's thesis, Michigan State University, East Lansing.

Meola, M. S. (1988). <u>Evaluation of a psychoeducational curriculum for promoting</u> <u>psychological health and self-esteem.</u> Unpublished doctoral dissertation, Western Michigan University, Kalamazoo.

Montreuil, M., Jouvent, R., Carton, S., Bungener, C., & Widlocher, D. (1991). Parallel information processing test: An experimental assessment of alexithymia. <u>Psychotherapy and Psychosomatics</u>, 56, 212-219.

Naatanen, P., Ryynanen, A., Keltikangas-Jarvinen, L. (1999). The influence of alexithymic characteristics on the self-perception and facial expression of a physiological stress state. <u>Psychotherapy and Psychosomatics</u>, 68, 252-262.

Nyklicek, I., & Vingerhoets, J. J. M. (2000). Alexithymia is associated with low tolerance to experimental painful stimulation. <u>Pain, 85</u>, 471-475.

O'Neill, R. M. & Bornstein, R. F. (1996). Dependency and alexithymia in psychiatric inpatients. <u>The Journal of Nervous and Mental Disease</u>, 184, 302-306.

Parker, J. D. A., Keightley, M. L., Smith, C. T., & Taylor, G. J. (1999). Interhemispheric transfer deficit in alexithymia: An experimental study. <u>Psychosomatic</u> <u>Medicine, 61,</u> 464-468.

Parker, J. D. A., Taylor, G. J., Bagby, R. M., & Acklin, M. W. (1993). Alexithymia in panic disorder and simple phobia: A comparative study. <u>American</u> <u>Journal of Psychiatry, 150, 1105-1107</u>.

Porcelli, P., Leoci, C., Guerra, V., Taylor, G. J., & Bagby, R. M. (1996). A longitudinal study of alexithymia and psychological distress in inflammatory bowel disease. Journal of Psychosomatic Research, 41, 569-573.

Porcelli, P., Zaka, S., Leoci, C., Centonze, S., & Taylor, G. J. (1995). Alexithymia in inflammatory bowel disease. <u>Psychotherapy and Psychosomatics</u>, 64, 49-53.

Readett, B. T. (2002). <u>An evaluation of a psychoeducational self-esteem</u> <u>curriculum</u>. Unpublished doctoral dissertation, Michigan State University, East Lansing.

Rosenberg, J. K. (1984). <u>Evaluation of a program in preventative mental health:</u> <u>"Building a competent self"</u>. Unpublished doctoral dissertation, Michigan State University, East Lansing.

Shedler, J., Mayman, M., & Manis, M. (1993). The illusion of mental health. American Psychologist, 48, 1117-1131.

Sifneos, P. E. (1972). <u>Short-term psychotherapy and emotional crisis</u>. Cambridge: Harvard Press University.

Solano, L., Toriello, A., Barnaba, L., Ara, R., & Taylor, G. J. (2000). Rorschach interaction patterns, alexithymia, and closeness to parents in psychotic and psychosomatic patients. Journal of the American Academy of Psychoanalysis, 28, 101-116.

Swiller, H. I. (1988). Alexithymia: Treatment utilizing combined individual and group psychotherapy. International Journal of Group Psychotherapy, 47-61.

Taylor, G. J. (2000). Recent developments in alexithymia theory and research. <u>Canadian Journal of Psychiatry, 45, 134-142</u>.

Taylor, G. J., Bagby, R. M., & Parker, J. D. A. (1997). <u>Disorders of affect</u> regulation: Alexithymia in medical and psychiatric illness. Cambridge: Cambridge University Press.

Taylor, G. J., Parker, J. D. A., & Bagby, R. M. (1999). Emotional intelligence and the emotional brain: Points of convergence and implications for psychoanalysis. <u>Journal</u> of the American Academy of Psychoanalysis, 27, 339-354.

Taylor, G. J., Parker, J. D. A., Bagby, R. M., & Acklin, M. W. (1992). Alexithymia and somatic complaints in psychiatric out-patients. <u>Journal of</u> <u>Psychosomatic Research</u>, 36, 417-424.

TenHouten, W. D., Hoppe, K. D., Bogen, J. E., & Walter, D. O. (1985). Alexithymia and the split brain, I. <u>Psychotherapy and Psychosomatics</u>, 43, 202-208.

Todarello, O., Casamassima, A., Daniele, S., Marinaccio, M., Fanciullo, F., Valentino, L., Tedesco, N., Wiesel, S., Simone, G., & Marinaccio, L. (1997). Alexithymia, immunity, and cervical intraepithelial neoplasia: Replication. <u>Psychotherapy and Psychosomatics, 66,</u> 208-213.

Todarello, O., Casamassima, A., Marinaccio, M., La Pesa, M. W., Caradonna, L., Valentino, L., & Marinaccio, L. (1994). Alexithymia, immunity, and cervical intraepithelial neoplasia: A pilot study. <u>Psychotherapy and Psychosomatics</u>, 61, 199-204.

Todarello, O., Taylor, G. J., Parker, J. D. A., & Fanelli, M. (1995). Alexithymia in essential hypertensive and psychiatric outpatients: A comparative study. <u>Journal of</u> <u>Psychosomatic Research</u>, 39, 987-994.

Tomkins, S. S. (1962). <u>Affect, imagery, and consciousness: The positive affects</u>, (Vol. 1). New York: Springer.

Tomkins, S. S. (1963). <u>Affect, imagery, and consciousness: The negative affects</u> (Vol. 2). New York: Springer.

Tomkins, S. S. (1979). Script theory: Differential magnification of affects. In M. E. Howe and R. A. Dienstbier (Eds.), <u>Nebraska Symposium on Motivation</u> (Vol. 26., pp. 201-236). Lincoln: University of Nebraska Press.

Tomkins, S. S. (1981). The quest for the primary motives: Biography and autobiography of an idea. Journal of Personality and Social Psychology, 41, 306-329.

Tomkins, S. S. (1987). Script theory. In J, Aronoff, A. I. Rabin, & R. A. Zucker (Eds.), <u>The emergence of personality</u> (pp. 147-216). New York: Springer).

Tomkins, S. S. (1991). <u>Affect, imagery, and consciousness: The negative affects – anger and fear</u>, (Vol. 3). New York: Springer.

Tomkins, S. S. (1992). <u>Affect, imagery, and consciousness: Cognition</u> – <u>duplication and transformation of information</u>, (Vol. 4). New York: Springer.

Tomkins, S. S. (1995). Inverse archeology: Facial affect and the interfaces of scripts within and between persons. In V. Demos (Ed.), <u>Exploring affect: The selected</u> writings of Silvan S. Tomkins (pp. 284–290). Paper presented at the annual meeting of the International Society for Research on Emotions at Rutgers University on July 15, 1990.

Tomkins, S. S., & McCarter, R. (1964). What and where are the primary affects? Some evidence for a theory. <u>Perceptual and Motor Skills, 18</u>, 119-158. Monograph supplement 1-V18.

Tsytsarev, S. V., & Grodnitzky, G. R. (1995). Anger and criminality. In H. Kassinove (Ed.), <u>Anger disorders: Definition, diagnosis, and treatment.</u> Washington, DC: Taylor & Francis, pp. 91-108.

Zeitlin, S. B., Lane, R. D., & O'Leary, D. S. (1989). Interhemispheric transfer deficit and alexithymia. <u>American Journal of Psychiatry</u>, 146, 1434-1439.

Zeitlin, S. B., & McNally, R. J. (1993). Alexithymia and anxiety sensitivity in panic disorder and obsessive-compulsive disorder. <u>American Journal of Psychiatry, 150,</u> 658-660.

Zeitlin, S. B., McNally, R. J., & Cassiday, K. L. (1993). Alexithymia in victims of sexual assault: An effect of repeated traumatization? <u>American Journal of Psychiatry</u>, 150, 661-663.