THE EFFICACY OF GROUP COGNITIVE BEHAVIOUR THERAPY AMONG PATIENTS WITH DEPRESSION IN FEDERAL NEURO-PSYCHIATRIC HOSPITAL, YABA

BY

AJIROTUTU OMOTAYO FRANCIS

MATRIC NO: 162682

A Thesis in the Department of Psychology Submitted to the Faculty of the Social Sciences in partial fulfillment of the requirements for the degree of

MASTERS OF PHILOSOPHY

Of the

UNIVERSITY OF IBADAN

MARCH, 2015.

CERTIFICATION

I certify that, this work was carried out by **AJIROTUTU OMOTAYO FRANCIS** (162682) in the department of Psychology, University of Ibadan, Nigeria, under my supervision.

---------- ----------

Supervisor **Date**
Professor Helen O. Osinowo
Department of Psychology
University of Ibadan

---------- ----------

Head of Department **Date**
Professor B.O. Ehigie
Department of Psychology
University of Ibadan

DEDICATION

This work is dedicated to God Almighty who made it possible for me to achieve this pursuit through rigorous steps and stages.

Also to my wife, Titilayo and children; Erinayo, Ifeoluwa, Mojolaoluwa, Kehinde and Taiwo who showed understanding and support while working on this thesis.

ACKNOWLEDGEMENT

It is an opportunity to have you Professor. Helen. Osinowo as my supervisor. I sincerely express my appreciation and gratitude to my supervisor for her guidance, understanding, patience and encouragement during this period. Your motherly approach and constant motivation made my work came through. I am grateful to you and pray that the Lord would continue to guide and protect you.

I also express gratitude to Dr. B.O. Olley, Dr. P.O Olapegba, Dr. A.O. Adejumo and Dr. Grace A Adejuwon for their constructive contributions throughout the study. Your constructive critique added value and improved my work.

Furthermore, appreciation goes to Professor A.M. Sunmola, Dr. A.S Okakume, Dr.A.I Alarape, Dr. J.O Ekore and other lecturers in the department who had helped to viewed this project.

My appreciation to Dr. Rahman Lawal (Medical Director) Federal Neuro-psychiatric Hospital Yaba, Lagos for his permission and encouragement to embark on the programme, I am so grateful sir. My many thanks goes to my colleagues Mr M.O Gabriel, Miss C. C Okonkwo, Mrs A.O Akinola, Mr A.B Gboyega Bamisile and others.

Special thanks go to Mrs. O.A Akintoyese, Miss I.R Ogunboye and Miss T. Odebunmi for their assistance and contribution to this work. Also, my heartfelt gratitude to Professor Osinowo who guided me in this work from the beginning even to the very end, this work is beautiful because of your extraordinary effort.

Finally, I cannot fail to mention the effort of my family, and firstly, my wife and children, my parents who laid the pedigree for my foundation in education.

ABSTRACT

Depression is a major psychological disorder which is common among patients in psychiatric hospitals. Cognitive Behavioural Therapy (CBT) has been used as complimentary intervention for depression, its efficacy on group cognitive therapy in Nigeria is yet to be established. This study therefore, examined the efficacy of group CBT in the treatment of depressed patients at the Federal. Neuro psychiatric Hospital, Yaba (FNPHY) Lagos.

The cognitive and behavioural theories provided the theoretical basis. This study involved a survey, using cross sectional expost facto an experimental design. A total of 116 clinically diagnosed depressed patients were purposively recruited at the outpatient clinic of FNPHY. A Questionnaire which contained Becks Depressive Inventory-2 (BDI-II) and Centre for Epidemiological studies-Depression Scale (CES-D) were used for the experiment. Forty participants with highest scores on the depression rating scale were randomlyassigned into 4 groups of 10 each. Three of the groups: Individual and Group (I&G), Individual Therapy (IT), Group Therapy (GT) benefitted from psychological intervention while the last was the control (C). The three experimental groups had eight sessions on goals, identification of problems, activity of negative thoughts, correction of irrational beliefs/thoughts, review, evaluation and termination. The control group had no intervention. The four groups were re-assessed after intervention. Data were analysed with one-way analysis of variance (ANOVA), t-test and multiple regression ($p<0.05$).

The mean age was 36±4.38, nineteen of the participants were single (47.5%), twenty-two were male (55%), and twenty-one had secondary education (52.5%).Participants who received GT and IT significantly reported lower scores on depression than the control, (t=-9.489, df=18).Participants who received group CBT significantly reported lower scores on depression than those who did not: t (18)=-2.218, and t(18)=-2.256, for BDI and CES-D respectively. Participants who received IT significantly reported lower scores on depression than those who did not: t (18) =-6.774 and t (18) =-8.316, for BDI and CES-D. Participants who benefited from GT and IT significantly reported lower score on depression than those who received only GCBT on CES-D t (18) =-4.979. Age, sex, marital and educational status did not predict depression among the participants.

Combined Group and Individual Cognitive Behaviour Therapy produced higher efficacy in the treatment of depression. Group and Individual Cognitive Behaviour Therapy may be encouraged for the management for depression.

Keywords: Depression, Group cognitive behaviour therapy, Becks depressive inventory.

Word count: 410

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER ONE

1.0 INTRODUCTION

1.1 BACKGROUND

Depression has been referred to in psychiatry as the common cold illness. World Health Organization (WHO) rated it as being present in as much as 1 in 4 people with a prevalence rate of 15% and a lifetime disease development of 6%, by the year 2020 and it is estimated that it would become the second largest cause of disability occuring more in females than males (WHO, 2009). In Nigeria however, Gureje, Uwakwe, Oladeji, Makanjuola and Esan (2010) estimated a 3.1% rate of major depression during a lifetime and within a 12 month period. They also found the age of onset to be about 29.2 years.

As a psychiatric disorder which lowers the quality of life, increases disease progression and mortality, depression has been discovered to be a major pathology among people with complaints such as loss of appetite, fear of future, feelings of sadness, sleeplessness, and hopeless confusion. Recently, a few studies have begun to address the specific treatment of residual depression using cognitive behavioral therapy (CBT).

Depression could be said to be both a common experience and a common illness. As an experience, we have all sometimes felt 'low' and found it hard to enjoy things. As an illness, it is so widespread that Seligman (1975) called it the common cold of psychiatry. However, Gilbert (1992) suggests that this comparison is unfortunate, for it conveys the impression of a frequent but mild complaint. In fact, severe depression can lead to tearfulness, irritability,

feelings of guilt, emotional numbness, loss of enjoyment, lack of energy, poor concentration, disruption of sleep/appetite/sexual functioning, negative rumination, hopelessness and, in some cases, suicidal ideation/attempts (Fennell, 1989).

It is therefore a potentially fatal illness. Estimates of lifetime risk for severe depression vary from 5% to over 12 % (Paykel, 1989; Gilbert, 1992; Fennell, 1989). According to Fennell (2002) depression has been estimated to account for 75% of psychiatric hospitalizations. 'Gilbert (1992) also points out that depression is frequently a factor in other disorders e.g. anxiety, eating disorders, addictions, schizophrenia etc.

Many forms of treatment have been tried for depression, including a variety of drugs, ECT, and many psychotherapeutic approaches. This brought to the fore the Cognitive Behavioral approach developed by Aaron Beck and his colleagues.

CBT aims at restructuringdysfunctional automatic thoughts which reflect a distorted perception of events and are associated with negative feelings such as sadness, anger and hopelessness. Examples of such thoughts are mind reading and labeling.CBT is a psychotherapeutic approach that aims to solve problems concerning dysfunctional emotions, behaviors and cognitions through a goal-oriented, systematic procedure. The title is used in diverse ways to designate behavior therapy, cognitive therapy, and to refer to therapy based upon a combination of basic behavioral and cognitive research.

There is empirical evidence that CBT is effective for the treatment of a variety of problems, including mood, anxiety, personality, substance abuse,

and psychotic disorders. Treatment is often manualized, with specific techniques which are brief, direct, and time-limited for specific psychological disorders. CBT is used in individual therapy as well as group settings, and the techniques are often adapted for self-help applications. Some clinicians and researchers are more cognitive oriented (e.g. cognitive restructuring), while others are more behaviorally oriented (in vivo exposure therapy).

Having combine both (e.g. imagine exposure therapy). CBT was primarily developed through a merging of behavior therapy with cognitive therapy. While rooted in rather different theories, these two traditions found common ground in focusing on the "here and now", and on alleviating symptoms. Many CBT treatment programs for specific disorders have been evaluated for efficacy and effectiveness.

The health-care trend of evidence-based treatment, where specific treatments for symptom-based diagnoses are recommended, has favored CBT over other approaches such as psychodynamic treatments. In the United Kingdom, the National Institute for Health and Clinical Excellence(NICE) recommends CBT as the treatment of choice for a number of mental health difficulties, including post-traumatic stress disorder, OCD, bulimia nervosa and clinical depression, and for the neurological condition chronic fatigue syndrome (NICE, 2005).

Cognitive–Behavioural Therapy (CBT) delivered in group format has attracted less research than CBT delivered on a one-to-one basis. Nevertheless, no doubt influenced by the challenge of limited resources, many centres do offer group therapy based on CBT principles as an alternative or in addition to more traditional CBT delivered to individuals. In England, the Improving Access to Psychological

Therapies (IAPT) programme (Department of Health 2008) aims to greatly increase the number of people treated using evidence-based approaches (as defined by the National Institute for Health and Clinical Excellence, NICE) for depression and anxiety disorders in primary care. The potential economies of scale afforded by the use of group work will be an attractive proposition for National Health Service providers working to implement IAPT.

Group psychotherapy has its origins in psycho-dynamic models of pathology and it developed before the practice of CBT was first outlined by Aaron Beck and colleagues (Beck 1979). Importantly, group psychotherapy views the interactions between the group members as the vehicle of change. In CBT groups, it has traditionally been assumed that the cognitive–behavioural model taught to the group is more of an 'active ingredient' than the relationships between the group members (Bieling 2006). Indeed, the educational ethos is inherent in the CBT model, and this lends itself very easily to the provision of groups or classes, as does the fact that CBT is structured, directive, collaborative and time-limited (Fennell, 1989).

However, group CBT cannot solely be about taught skills, and in common with individual CBT, non-specific interpersonal factors play a part. As with individual CBT, these non-specific factors have not attracted the same research attention as the underlying cognitive–behavioural theoretical models (such as which underlying thoughts and behaviours are paramount in different disorders) (Bieling, 2006; Tucker, 2007). This is in sharp contrast to group psychotherapy, which has focused on interpersonal relationships and related mechanisms of change.

Depression is not a straightforward issue. In practice, clients rarely seem to present with a single 'unadulterated' difficulty (depression or otherwise). On the other hand descriptions of behavioral, emotional,

physiological and cognitive factors contributing to distress in the patient can be used by the therapist to identify the general ground he/she is working on. (Wills & Sanders 1997).

The classification and descriptions of depression are found in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (American Psychiatric Association, 1994), otherwise known as the DSM-IV.

The largest such study to date is a controlled trial of 158 patients with residual symptoms after an episode of major depressive disorder (Paykel et al, 1999; Scott et al, 2000). All participants continued on antidepressant medication but some also received cognitive therapy. The latter group had a relapse rate of 29%, compared with 47% in the group that was not given cognitive therapy. Significant differences were also observed in the proportion in each group that achieved full remission, although the effect was not as dramatic as that seen in the reduction in the rate of relapse.

Paykel (2001) therefore concludedthat cognitive therapy appears to have a specific indication as a continuation or maintenance therapy for relapsing and recurring depression, particularly in the presence of residual symptoms and in conjunction with medication. He found that other psychological therapies have less evidence to support them in this role. In a study of 650 patients with chronic depressive disorder Keller et al(2000) also found a greater remission rate in the patients who received both an antidepressant medication and a modified form of CBT (42%) compared with either treatment on its own (22–24%).

It should be noted, however, that over 20% of these patients had not received treatment for their depression before inclusion in the study. By following up a cohort of patients seeing a psychiatrist over 2 years, Cornwall and Scott (1999) examined the factors that predict whether an individual with major depression will have a full or only a partial response to medication and hospitalisation. They noted that residual symptoms of depression remained in significant numbers of those who had received adequate doses of antidepressant medication for a sufficient period of time. Thus, some patients do not fully recover with medication alone. This group tended to have significantly lower levels of self-esteem. Over the 2-year period, they also tended to judge their levels of depression as worse than the ratings made by the professionals.

This divergence of self-ratings and professional ratings suggested to Cornwall and Scott (1999) that the persistence of depressive symptoms might be evidence of Teasdale's (1988) hypothesis that some individuals 'get depressed about being depressed'. This causes a feedback loop in which the depressive symptoms and the secondary beliefs maintain each other. Cornwall and Scott suggest, therefore, that CBT might have a special role in partial responders by both helping to alleviate the biological symptoms of depression through behavioural interventions such as increased activities and also addressing the maintaining beliefs.

CBT is therefore the most commonly prescribed psychological treatment for depression in Britain. Despite this, its use has been criticised because of the paucity of research supporting its effectiveness in this role. A recent illustration of this was the study in Derbyshire, UK, by Simpson et al.

(2000) which compared counselling and GP care for chronic depression with GP care alone. They found no benefit from adding the counselling to the usual GP interventions. This situation raises significant questions about how best to deliver services within the NHS. Traditionally, most services that deliver CBT do so within secondary or tertiary care situations that offer specialist CBT to a number of highly selected patients.

The qualifying criteria for receipt of this treatment are therefore high, resulting in long waiting lists and limited access to treatment in Nigeria. Thus, these services appear to offer a high-quality and specialist service – but only to a few. Consequently, there is a huge unmet need in primary and secondary care and a mismatch of demand and supply that results in frustrations for patients and their referring practitioners. Lovell & Richards argue that 'services characterised by 9–5 working, hourly appointments and face-to-face therapy disenfranchise the majority of people who would benefit from CBT'.

The concern of the study is that not only do such services fail to provide access to treatmentthey also fail to offer CBT in line with the available evidence. In short, although CBT is an evidence-based form of psychotherapy, we tend to deliver it in services that are themselves not evidence based.

1.2 Statement of Problem

Many people are often referred to psychiatric hospitals for the management of psychological distress such as depression. Often times, these patients are in and out of the hospital as it does not take long before they go back for re-admission because of relapse. They often complain that the

medications help them only for a very short time. Besides chemothgerapy has side effect while psychotherapy does not have side effect, it is readily availiable, easy to apply and its cheap and affordable. Therefore there is need for combination of therapy (CBT and pharmacological treatment) other than medications alone in the management of patient with depression.

This will improve the quality of life of the patient, and enable the health-care experts to apply appropriate psychological techniques in the management of these distresses. Having observed through some studies reviewed and Clinical observation, Cognitive behavioral therapy as a treatment of choice has been found to alleviate symptoms associated with Depression. Lots of interventions have been carried out with the use of individual cognitive behaviour therapy while there are few documentations and evaluations of group cognitive behaviour therapy within our locality compared with studies abroad (e,g)

Group cognitive behaviour therapy is time saving and could accommodate large number of patients with the appropriate intervention techniques which would also be spent while conducting individual cognitive behavioral therapy.

There are few clinical psychologists available in Nigeria compared to the large number of patients seen at treatment centers. If group cognitive behavioral therapy is found to be effective it would enable these professionals render their services efficiently to a large population.

1.3 Purpose of the Study

The study aimed at the following:

1) To evaluate the efficacy of combined (individual and group) Cognitive behaviour therapy compared to the control group among depressive patients.

2) To evaluate the efficacy of group cognitive behaviour therapy between the treatment group and the control group.
3) To compare the efficacy of only individual cognitive behaviour therapy between the treatment group and the control group.
4) To compare the efficacy of combined (Individual and group) CBT to group CBT alone among patients with depression.
5) To examine the efficacy of combined (individual and group) CBT compared to group CBT alone among patients with depression.
6) To investigate the influence of age, gender, marital status, employment status, and educational level on depression.

1.4 Relevance of Study

Depression has been shown to respond well to CBT as it is structured, effective and in most cases relatively brief. A lot of studies have been carried out in Western societies, evaluating the impact of CBT but not much documentation is available in this part of the world especially Nigeria in terms of process and efficiency.

In some cases, CBT represents a proven and sometimes superior alternative to medication. In other cases, CBT is a beneficial addition to medication, hastening improvement and helping to maintain improvements over time.

Group cognitive behaviour therapy is attracting increasing levels of interest from health care professionals in Nigeria. A variety of factors may contribute to this rise in popularity. First, recent decades have seen a growing recognition of the high prevalence rates of many psychological problems which negatively affect the quality

of life for the person as well as his or her family. This study will assist patients and their families in understanding and coping with depression.

Equally the expert use of group cognitive behaviour and the ability to show its effectiveness on depression in Nigeria will have a lot of benefits and also help improve the practice of Clinical Psychologist in our locality. With its effective implementation, depression will reduce significantly which will then lead to a possible reduction in suicide rates and a increase in the quality of life of Nigerians.

Awareness of this study is bound to improve not only the way group cognitive behaviour therapy is carried out in institutions, but to improve our nation and get us to where the developed nations are in terms of mental health. A lot of depressed people go undiagnosed and later, this leads to endless dangers. Publication of this study might go some ways into gradually eradicating this problem and ultimately showcasing the contribution of clinical psychologists in the management of people with depression. Most importantly, there are few clinical psychologists in clinical practice in Nigeria and this would ensure faster and better service to reach lots of patients and clients.

CHAPTER TWO

2.0 LITERATURE REVIEW

This chapter was discussed under two sections; theoretical and empirical reviews.

2.1 Theoretical framework

2.1.1 Biomedical Model of Depression

By the end of the nineteenth century different researchers proposed various taxonomies of mental disorders but it was the ideas of Emil Kraepelin which first gained widespread acceptance (Bentall, 1998). He proposed that there were only two types of mental disorder: dementia praecox and manic-depressive illness. Kraepelin systematically collected data on the course and outcome of disorders, with the intention of developing a diagnostic system. In 1911 Eugene Bleuler went on to revise some of his forerunner's findings and to suggest the term schizophrenia instead of dementia praecox

Biomedical approaches to treatment are based on the assumption that biological factors are involved in the psychological disorder. This does not mean that biological factors cause the disorder however that there is a relationship between the disorder and brain chemistry. Various drugs are used to treat various disorders based on the theories of the brain chemistry however these drugs may not be completely effective in curing the patient but are used because they can help to change a person's mood in a positive direction. The biomedical model embraces reductionism and assumes disease is caused by any deviation from the norm of measurable biological/somatic variables and believes the only effective treatment for pain is via

medical approaches. Here the reductionistic primary principle is physicalistic; that is, it assumes that the language of chemistry and physics will ultimately suffice to explain biological phenomena. The medical model is a prevailing and dominant view of pathology in the world (Nevello,1999; Kaplan and Sadock, 1998). The medical model relies exclusively on biological explanation of disease and illness and its interpreted in terms of malfunction of organs, cells and other biological system (e.g. heart disease, loss of oxygen to brain, liver disease or osteoporosis). Within the medical model, the health of the population is measured in terms of vital statistics, which are data on the degree of illness (morbidity) and the number of deaths (mortality) in a given population.

Biomedical approach to treatment of Depression is also based on the assumptions that if the problem is based on biological malfunctioning, drugs are able to restore and maintain the biological system. Since depression is known to involve the imbalance of neurotransmissions, drugs are used to restore an appropriate chemical balance in the brain. Drugs essentially work by affecting the transmission in the nervous system of neurotransmitters such as dopamine, serotonin, noradrenalin or GABA. Simply the outcome is to increase or decrease the levels of neurotransmitters in the synaptic gap, and as a result enhance or diminish the effectiveness of the chemical. The change in the transmission can have a calming or energizing effect on different kinds of behaviour. Antidepressant drugs are used to elevate the mood of people suffering from depression, the most common drug is selective serotonin re-uptake inhibitors (SSRIs), which increases the level of available serotonin by preventing its re-uptake in the synaptic gap. Common example: Prozac. Effective and relatively safe. Side effects include: vomiting, nausea, insomnia, sexual dysfunction or headaches.

The biomedical model posits that mental disorders are brain diseases and emphasizes pharmacological treatment to target presumed biological abnormalities. A biologically-focused approach to science, policy, and practice has dominated the American healthcare system for more than three decades. During this time, the use of psychiatric medications has sharply increased and mental disorders have become commonly regarded as brain diseases caused by chemical imbalances that are corrected with disease-specific drugs. However, despite widespread faith in the potential of neuroscience to revolutionize mental health practice, the biomedical model era has been characterized by a broad lack of clinical innovation and poor mental health outcomes. In addition, the biomedical paradigm has profoundly affected clinical psychology via the adoption of drug trial methodology in psychotherapy research. Although this approach has spurred the development of empirically supported psychological treatments for numerous mental disorders, it has neglected treatment process, inhibited treatment innovation and dissemination, and divided the field along scientist and practitioner lines. The neglected biopsychosocial model represents an appealing alternative to the biomedical approach, and an honest and public dialog about the validity and utility of the biomedical paradigm is urgently needed.

The biomedical model of illness and healing focuses on purely biological factors, and excludes psychological, environmental, and social influences. This is considered to be the dominant or the modern way for health care professionals to diagnose and treat a condition in most Western countries. Most health care professionals do not first ask for a psychological or social history of a patient; instead, they tend to analyze and look for biophysical or genetic malfunctions. The focus is on objective laboratory tests rather than the subjective feelings or history of the patient.

According to this model, good health is the freedom from pain, disease, or defect. It focuses on physical processes that affect health, such as the biochemistry, physiology, and pathology of a condition. It does not account for social or psychological factors that could have a role in the illness. In this model, each illness has one underlying cause, and once that cause is removed, the patient will be healthy again.

The biomedical model is often contrasted with the biopsychosocial model. George L. Engel(1977) questioned the dominance of the biomedical model, proposing the biopsychosocial model to holistically assess a patient's biological, social, psychological, and behavioral backgrounds to determine his or her illness and path of treatment. Although the biomedical model has remained the dominant theory in most places, many fields of medicine including nursing, sociology, and psychology make use of the biopsychosocial model at times. In recent years, some medical professionals have also begun to adopt a biopsychosocial-spiritual model, insisting that spiritual factors must be considered as well.

Many scholars in disability studies describe a medical model of disability that is part of the general biomedical approach. In this model, disability is an entirely physical occurrence, and being disabled is a negative that can only be made better if the disability is cured and the person is made "normal." Many disability rights advocates reject this, and promote a social model in which disability is a difference — neither a good nor bad trait. Proponents of the social model see disability as a cultural construct. They point out that how a person experiences his or her disability can vary based on environmental and societal changes, and that someone who is considered disabled can often be healthy and prosperous without the intervention of a professional or the disability being cured. The attempt to make psychiatric diagnosis more reliable, combined with a return to a biomedical model of mental illness, has been called the "neo-Kraepelinian" approach (Klerman, 1978). This perspective promotesmany of the ideas associated with the views of Emil Kraepelin, often considered to

be the founder of modern psychiatry. It regards psychiatry as a scientific, medical specialty that clearly demarcates mentally ill patients, who require treatment, from normal people. The focus is on biological aspects of mental illness and an intentional concern with diagnosis and classification. The biomedical hypothesis seems to function as an apology for psychiatric practice. It needs to be defended and promoted because it appears to provide the foundation for psychiatric intervention and treatment. To deny that mental illness is a physical disease may therefore be seen as dangerous as it seems to undermine orthodox practice. The biomedical perspective tends to dominate other points of view in psychiatry. In a power struggle, the authority of the biomedical viewpoint needs to be ascendant.

The main point of this model of disease is that it attempts to uncover underlying pathological processes and their particular effects. The problem with earlier symptom-oriented approaches to health was that no such sequence of events could be established, and treatment could only be symptomatic. In the case of depression, the symptom could also be found confused withmedical illness and this problem of linking symptoms to specific underlying mechanism frustrated medical development. Although observation and the treatment of symptoms were established practices in early morden medicines and have remained important to physicians ever since, it was often difficult to distinguish such approaches from a wide variety of unorthodox practices. The model is quite relevant to the study in that its principles can be applied by health givers to have a better knowledge on causation of disease and how it can be reduced.

2.1.1.1 Application of the Biomedical Model

Depression is a serious mental disorder that requires the up most care and approach when being treated. Various techniques can be used to fight the symptoms of depression; however no method can eradicate the illness by itself. Many different

treatments, which can be biomedical, individual, and group approaches, at times must all be used to effectively and safely eradicate depression and its dangerous symptoms. All of these approaches have their ups and downs, which only shows how complicated and difficult it can be to treat mental illnesses such as depression and the amount of effort and care needed in approach to treating it.

In the biomedical category of treating depression, antidepressants are among the most popular of medicine used today. Antidepressants target the endocrine system as well as some parts of the nervous system to stimulate the production of either more, less, or balanced amount of a hormone or neurotransmitter. For example, the antidepressant Prozac acts as a Serotonin reuptake inhibitor, meaning that they stimulate a decreased production of Serotonin as well as decreasing the bodies sensitivity to Serotonin to create a more balanced amount of hormones and neurotransmitters in the body in hopes of eradicating the symptoms of depression (Saisan, 2006).

Another method that falls within the biomedical category is psychosurgery. This method is considered the most extreme of the treatments used on patients with depression. This method is only used on the most serious of conditions and when all methods have failed. The reason psychosurgery is consider so extreme is because it requires a part of the brain, specifically a part of the frontal lobe, to be cut off. This surgery involves separating the neural pathways between the frontal lobe and the basal ganglia regions of the brain. Although this method is effective, it may cause the patient to loose other mental attributes such as the ability to comprehend certain situations and the ability to recall certain memories (“Measuring the Unmeasurable”) Today, the most common group of drugs used are selective serotonin re-uptake inhibitors. These drugs simply prevent the re-uptake in the synaptic gap. A study,

conducted by psychologist Elkin's in 1989, explored the effect of biomedical trials. In this study, “individuals were randomly assigned to treatment using either an antidepressant drug (imipramine), interpersonal therapy (IPT), or cognitive-behavioral therapy (CBT) or another form of therapy. The results showed that just over 50% per cent of patients recovered in each of the CBT and IPT groups, as well as the drug group." This is significant as only 29% of the patients recovered in the placebo group (Crane). This study illustrates, through the improvements shown, the effectiveness of antidepressants and how they treat depression as a bio-medical approach.

Many studies have shown that individuals with depression can take initiative and improve their condition by themselves. Research has proven that life style changes have resulted in astonishing improvement to the mental health of depressive patients. Regular exercise has been proven to boost serotonin and endorphin levels, which would lead to positive results for depressive patients with low levels of serotonin. A balanced meal, if consistently consumed, is said to aid in minimizing inconsistencies in behavior, which could help patients that are near recovery from depression maintain their mental health and not worsen their condition. Reducing the amount of stress is said to aid in eradicating the symptoms of depression as well as stress can cause the imbalanced production of hormones and neurotransmitters (Saisan, 2006). These are all factors that could be controlled by an individual and through effort and care an individual could treat his/her depression and its symptoms.

In addition, the biomedical paradigm has profoundly affected clinical psychology via the adoption of drug trial methodology in psychotherapy research. Although this approach has spurred the development of empirically supported psychological treatments for numerous mental disorders, it has neglected treatment

process, inhibited treatment innovation and dissemination, and divided the field along scientist and practitioner lines.

Antidepressant drugs are an effective short- term treatment for depression, helping 60-80%. Modern drug treatments provide effective long -term results and control for mood disorders and may help in preventing suicide in depressive patients. Reduction of hospital cases of depression has been ensured.

The above model is relevant for many disease-based illnesses and is supported by a wealth of biological findings. The model is effective in acute illnesses that have predictable outcomes (e.g. treatment for bacterial infections using antibiotics) and is therefore suitable to healthcare practitioners (HCP's) who have to focus on one part of an individual's health. However, despite success in the treatment of many disease processes, some difficult and important medical problems have proven resistant to the biomedical model. For example, the biomedical model does not explain why pain can continue when tissue damage is no longer present (chronic pain) or clinical phenomena such as phantom pain. It leaves no room within its framework for the social, psychological, and behavioral dimensions of illness (Engel, 1977). The biomedical model looks at the underlying pathophysiology in isolation and often cannot explain why prescribed treatments fail, e.g. treatment for chronic low back pain (LBP).

The biomedical model has helped greatly irrespective of any criticism to understand the role of neurotransmitters in the development of depression. It has made us to understand that chemical imbalance is respoinsible for this problem. It has also given insight into how chemotherapy can be effective in helping people with depression.

2.1.2 Bio-Psychosocial Model of Depression

The biopsychosocial model proposes that biological, psychological (emotional or cognitive), and social (or environmental) factors all play a role in the presentation of a disease or disorder. Indeed health is best understood in terms of a combination of biological, psychological and social factors rather than purely in biological terms. This is in contrast to the biomedical model of medicine that suggest every diseases process can be explained in terms of an underlying deviation from normal function such as pathogen, genetic or developmental abnormality, neurotransmitter or injury.

Health is traditionally equated to the absence of disease. A lack of a fundamental pathology was thought to define one's good health as good, whereas biologically driven pathogens and conditions would render an individual with poor health and labeled "diseased". However, such a narrow escape on health limited our understanding of wellbeing, thwarted our treatment efforts and perhaps more importantly, suppressed prevention measures.

The biopsychosocial model of health is based in part on social cognitive theory and it shows an interaction between the environment and the mind. This bio-psychosocial model is of the view that our mental health encompasses not just biological factors but also psychopathological factors (thoughts,emotions, and goal directed behaviour) and social factors (family,traditions, culture). The biopsychosocial model is both a philosophy of clinical care and a practical clinical guide. Philosophically, it is a way of understanding how suffering, disease and illness are affected by multiple levels of organization from the societal to the molecular. At the practical level, it is a way of understanding the patient's subjective experiences as

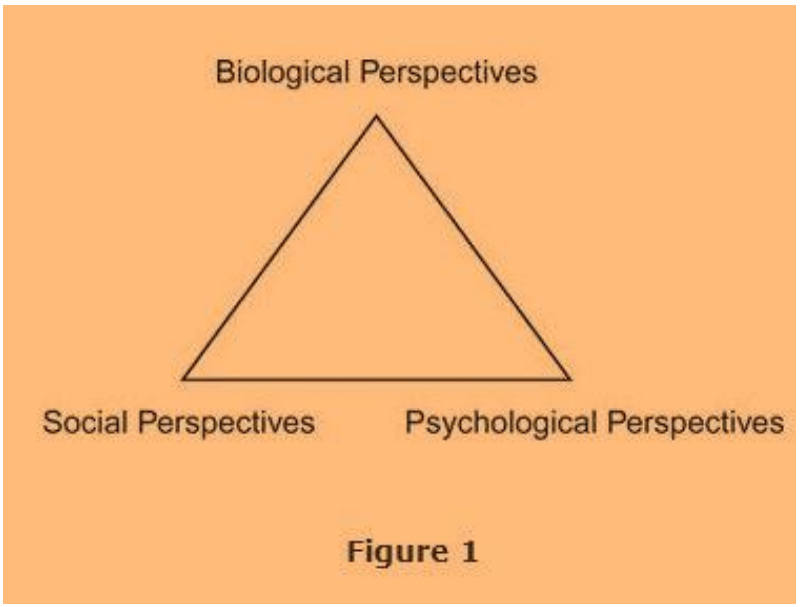
an essential contributor to accurate diagnosis, health outcomes and humane care (Borell-Carrio, Suchman & Espstein, 2004).

The biopsychosocial model implies that treatment of disease processes, for example type 2 diabetes and cancer, requires that the health care team address biological, psychological and social influences upon a patient's functioning. In a philosophical sense, the biopsychosocial model states that the workings of the body can affect the mind, and the workings of the mind can affect the body. This means both a direct interaction between mind and body as well as indirect effects through intermediate factors.

The biopsychosocial model presumes that it is important to handle the three together as a growing body of empirical literature suggests that patient perceptions of health and threat of disease, as well as barriers in a patient's social or cultural environment, appear to influence the likelihood that a patient will engage in health-promoting or treatment behaviors, such as medication taking, proper diet or nutrition, and engaging in physical activity.

Theinteraction of biological, psychological, and social aspects of developmental psychology form the essence of the holistic biopsychosocial perspective. The biopsychosocial perspective attributes complex phenomena or events to multiple causes.

Figure 1 shows the interrelationship of the fields of study that constitute the biopsychosocial perspective. In contrast to the biopsychosocial perspective is the reductionistic perspective, which reduces complex phenomenon or events to a single cause.



George Engel (1980)

This approach to diagnosis and treatment originated in the medical field with George Engel (1980) asserting:

> "The crippling flaw of the [biomedical] model is that it does not include the patient and his attributes as a person, a human being. Yet in the everyday work of the physician the prime object of study is a person, and many of the data necessary for hypothesis development and testing are gathered within the framework of an ongoing human relationship and appear in behavioral and psychological forms, namely, how the patient behaves and what he reports about himself and his life. The biomedical model can make provision neither for the person as a whole nor for data of a psychological or social nature."

The same can be said of the psychosocial model, in which biological risk factors of disorders have historically been less emphasized and sometimes ignored. In contrast to these limited models, Engel encouraged physicians and psychiatrists to adopt a biopsychosocial model in order to understand human suffering, wherein both biomedical and psychosocial factors are considered in the evaluation process.

Several studies, recognizing the need for an understanding of depression that goes beyond assessment of symptoms, have also attempted to integrate biological, psychological, and social approaches to diagnosing depression (Clark, 2005; Gold& Chrousos, 2002; Heim, 2005; Kendler, Gardner, & Prescott, 2002; Luyten, 2006). All of these studies found that interactions among etiological factors, including biological processes, life stress, personality traits, and thinking style, affect the development of depression. However, as stated previously, though many studies have incorporated biopsychosocial aspects into interaction models aimed at better understanding depression, the details and validity of a specific biopsychosocial model have yet to be established by research. It has been frequently proposed as a basis for diagnosis and treatment (Luyten, 2006, Schotte,2006), but found no empirical support for its successful implementation in assessing and treating depression. Additionally, it has been criticized for its somewhat ambiguous definition (McLaren, 1998). There is however those that believe the biopsychosocial model is flawed and their views must be considered when assessing what contribution the biopsychosocial model makes to physiotherapy practice. Reviews of the literature do however generally support the effectiveness of a biopsychosocial approach (Ostelo et al.,2005,George,2008, Scascighini,2008) and frequently find multi-disciplinary, multi-modal treatments give good outcomes in the treatment of chronic pain. Others argue that multidisciplinary biopsychosocial rehabilitation requires substantial staff and financial resources. Similarly Karjalainen (2003) found there was not enough good evidence to support multidisciplinary rehabilitation in adults with neck/shoulder pain. However this study was a methodologically low quality randomized controlled trial (RCT) which failed to randomize patients, use a power calculation or intention-to-treat analysis. In contrast, high quality studies (Mosely et al.,2002,Smeets, 2006) looked at biopsychosocial approaches to individual physiotherapy care (which requires less financial resources) and provided strong evidence of their effectiveness. They had a population match to the target population (primary care) and were

sufficiently well described to be applied by a sole physiotherapy practitioner. It is interesting to note that such studies are in the form of bio medically orientated RCT's whereas it is qualitative research that is concerned with exploring people's perceptions, beliefs, attitudes and experiences.

This model has given explicit insight into the development and maintainance of depression. There is an interaction among etiological factors, including biological processes; life stress, personality traits, and thinking style as it affect the development of depression. Taking this into cognizant then the treatment approach will follow the model so as to adequately help individuals with depression.

2.1.2.1 Application of the Biopsychosocial Model

The biopsychosocial model can be used to obtain a better understanding of the disease process. Some thinkers see the biopsychosocial model in terms of causation. On this understanding the biological component of the biopsychosocial model seeks to understand how the cause of the illness stems from the functioning of the individual's body. The psychological component of the biopsychosocial model looks for potential psychological causes for a health problem such as lack of self-control, emotional turmoil, and negative thinking. The social part of the biopsychosocial model investigates how different social factors such as socioeconomic status, culture, poverty, technology, and religion can influence health. However a closer reading of Engel's seminal paper in the American Journal of Psychiatry (1980) embeds the biopsychosocial model far more closely into patient care. it is not just about causation but about how any clinical condition (medical, surgical or psychiatric) can either be seen narrowly as just biological or more widely as a condition with psychological and social components, which will impinge on a patient's understanding of her condition and will affect the clinical course of that condition.

While operating from a BPS framework requires that more information be gathered during a consultation, a growing trend in US healthcare (and already well-established in Europe such as in the U.K. & Germany) includes the integration of professional services through integrated disciplinary teams, to provide better care and address the patient's needs at all three levels. As seen, for example in integrated primary care clinics, such as used in the U.K., Germany, U.S. Veteran's Administration, U.S. military, Kaiser Permanente, integrated teams may comprise physicians, nurses, health psychologists, social workers, and other specialties to address all three aspects of the BPS framework, allowing the physician to focus on predominantly biological mechanisms of the patient's complaints.

Psychosocial factors can cause a biological effect by predisposing the patient to risk factors. An example is that depression by itself may not cause liver problems, but a depressed person may be more likely to have alcohol problems, and therefore liver damage. Perhaps it is this increased risk-taking that leads to an increased likelihood of disease. Most diseases in BPS discussion are such behaviourally-moderated illnesses, with known high risk factors, or so-called "biopsychosocial illnesses/disorders". An example of this is type 2 diabetes, which with the growing prevalence of obesity and physical inactivity, is on course to become a worldwide pandemic. For example, approximately 20 million Americans are estimated to have diabetes, with 90% to 95% considered type 2.

It is important to note that the biopsychosocial model does not provide a straightforward, testable model to explain the interactions or causal influences (that is, amount of variance accounted for) by each of the components (biological, psychological, or social). Rather, the model has been a general framework to guide theoretical and empirical exploration, which has amassed a great deal of research

since Engel's 1977 article. One of the areas that has been greatly influenced is the formulation and testing of social-cognitive models of health behavior over the past 30 years. While no single model has taken precedence, a large body of empirical literature has identified social-cognitive (the psycho-social aspect of Engel's model) variables that appear to influence engagement in healthy behaviors and adhere to prescribed medical regimens, such as self-efficacy, in chronic diseases such as type 2 diabetes, cardiovascular disease, etc. These models include the Health Belief Model, Theory of Reasoned Action and Theory of Planned Behavior, Transtheoretical Model, the Relapse Prevention Model, Gollwitzer's implementation-intentions, the Precaution–Adoption Model, the Health Action Process Approach, etc

This biopsychosocial model of developmental psychology may be applied to the case of John, a depressed adolescent male, who finds it difficult to socialize with his peers. John's problem may be the result of any one of a number of causes. For example, injunctions**,** or messages received during childhood, may be considered one possible cause of John's depression. Injunctions may include messages regarding worthlessness and shame, distorted perceptions, fears of rejection, and inadequate communication and social skills. John's overly critical parents raised him to believe that he would never amount to anything or have any friends. As John experiences distress over his negative injunctions about relationships (psychological), he "tries too hard" to make others like him, which causes his peers to distance themselves from him (social). In time, John may experience rejection and become more depressed (psychological). Berating himself (psychological), John may become less concerned with his outward appearance and hygiene (biological), which in turn may cause his peers to avoid further contact with him (social). The biopsychosocial model uses a holistic approach as it aims to treat both the patient and the disease. For example,

using the biomedical model(which focuses solely on the disease/impairment) treatment for a sprained ankle is independent of the patient; treatment includes rest, compression, and elevation. Using the biopsychosocial model, treatment would be based on the individual. For example, if the patient was a busy mother, treatment would be adjusted accordingly by understanding that rest may not be achievable for this individual due to social factors. The biopsychosocial model aims to encourage patients to contribute to their treatment (e.g. through shared decision-making) and empower them in self-managing their pain (Edwards et al., 2004). However, if patients believe their pain is solely physiological they are less likely to accept self-management (Underwood, 2006) or biopsychosocial approaches (Stone, 2002). It is therefore vital to establish patient pain beliefs in order to provide an effective intervention.

Urquhart et al. (2008) support the need to alter maladaptive beliefs/behaviour. The study suggests negative beliefs (e.g. fear-avoidance) are predictive of chronic, disabling pain and that changing these beliefs is more important than biomedical factors in pain intervention success, supporting the need for a biopsychosocial model.

For obvious reasons, developmental psychologists are cognizant of these types of interacting biological, psychological, and social components when considering life-span events and issues. In a case such as John's, a developmentalist may choose to conceptualize and treat his problem from all three perspectives, rather than focusing on one. Because of the developmentalist's method of exploring all three perspectives, John benefits from a holistic and comprehensive approach to his difficulties.

The biopsychosocial model however extends beyond medical-care and looks at the patient's unique biologic, psychological, social, co-morbidities, illness beliefs, coping strategies, fear, depression, employment, and financial concerns and may give further insight into what has hindered recovery and sustained patient-hood. The biopsychosocial model gives the clinician biologic and psychosocial factors with which to explain why people persist with pain and therefore a set of alternative tools to treat patients.

2.1.3 Cognitive Theory of Depression

Cognitive theories of depression hypothesize that particular negative ways of thinking increase individuals' likelihood of developing and maintaining depression when they experience stressful life events. According to these theories, individuals who possess specific maladaptive cognitive patterns are vulnerable to depression because they tend to engage in negative information processing about themselves and their experiences. Two of the most influential pioneers of cognitive theory were Ellis (1958) and Beck (1967).

2.1.3.1 Aaron Beck's Cognitive Theory of Depression

Becoming disillusioned with long-term psychodynamic approaches based on gaining insight into unconscious emotions and drives, Beck came to the conclusion that the way in which his clients perceived, interpreted and attributed meaning in their daily lives is a process scientifically known as cognition which was a key to therapy.

Beck's (1967) cognitive theory of depression was based on the rational that an individual's affect and behavior are largely determined by the way in which he structures the world (Beck, Rush, Shaw & Emery, 1979) and the therapeutic techniques were designed to identify, reality test, and correct distorted

conceptualizations and the dysfunctional beliefs underlying these cognitions. Beck's (1967) cognitive therapy for depression was based on the assumption that the affected people engage in faulty processing and reasoning, and subscribe to schemas that are self-defeating. He argued that depressed people look at the world through a negative cognitive triad. This implies that they have negative views of themselves, of the world, and of the future. Beck outlined his approach in *Depression: Causes and Treatment* in 1967. He later expanded his focus to include anxiety disorders, in *Cognitive Therapy and the Emotional Disorders* in 1976, and other disorders and problems. He also introduced a focus on the underlying "schema"—the fundamental underlying ways in which people process information about themselves, the world or the future. Beck hypothesized that depression-prone individuals possess negative self schemata (beliefs), which he labeled the "cognitive triad." Specifically, depressed patients have a negative view of themselves (seeing themselves as worthless, inadequate, unlovable, deficient), their environment (seeing it as overwhelming, filled with obstacles and failure), and their future (seeing it as hopeless, no effort will change the course of their lives). This negative way of thinking guides one's perception, interpretation, and memory of personally relevant experiences, thereby resulting in a negatively biased construal of one's personal world, and ultimately, the development of depressive symptoms. For example, the depression-prone individuals are more likely to notice and remember situations in which they have failed or did not live up to some personal standard and discount or ignore successful situations. As a result, they maintain their negative sense of self, leading to depression. Cognitive theories rose to prominence in response to the early behaviorists' failure to take thoughts and feelings seriously. The cognitive movement did not reject behavioral principles, however. Rather, the idea behind the cognitive movement was to integrate mental events into the behavioral framework. Cognitive Behavioral theories

(sometimes called "cognitive theories") are considered to be "cognitive" because they address mental events such as thinking and feeling. They are called "cognitive behavioral" because they address those mental events in the context of the learning theory that was the basis for the pure behavioral theory described above. The rise in popularity of cognitive behaviorism continues today; it forms the basis of the most dominant and well-research formed of psychotherapy available today: Cognitive-Behavioral Therapy, or CBT.

Cognitive behavioral theorists suggest that depression results from maladaptive, faulty, or irrational cognitions taking the form of distorted thoughts and judgments. Depressive cognitions can be learned socially (observationally) as is the case when children in a dysfunctional family watch their parents fail to successfully cope with stressful experiences or traumatic events. Or, depressive cognitions can result from a lack of experiences that would facilitate the development of adaptive coping skills.

According to cognitive behavioral theory, depressed people think differently than non-depressed people, and it is this difference in thinking that causes them to become depressed. For example, depressed people tend to view themselves, their environment, and the future in a negative, pessimistic light. As a result, depressed people tend to misinterpret facts in negative ways and blame themselves for any misfortune that occurs. This negative thinking and judgment style functions as a negative bias; it makes it easy for depressed people to see situations as being much worse than they really are, and increases the risk that such people will develop depressive symptoms inresponse to stressful situations.

Different cognitive behavioral theorists have developed their own unique twist on the Cognitive way of thinking. According to Dr. Aaron Beck, negative thoughts, generated by dysfunctional beliefs are typically the primary cause of depressive

symptoms. A direct relationship occurs between the amount and severity of someone's negative thoughts and the severity of their depressive symptoms. In other words, the more negative thoughts you experience, the more depressed you will become.Beck also asserts that there are three main dysfunctional belief themes (or"schemas") that dominate depressed people's thinking: 1) I am defective or inadequate, 2) All of my experiences result in defeats or failures, and 3) The future is hopeless. Together, these three themes are described as the Negative Cognitive Triad. When these beliefs are present in someone's cognition, depression is very likely to occur (if it has not already occurred).

An example of the negative cognitive triad themes will help illustrate how the process of becoming depressed works. Imagine that you have just been laid off from your work. If you are not in the grip of the negative cognitive triad, you might think that this event, while unfortunate, has more to do with the economic position of your employer than your own work performance. It might not occur to you at all to doubt yourself, or to think that this event means that you are washed up and might as well throw yourself down a well. If your thinking process was dominated by the negative cognitive triad, however, you would very likely conclude that your layoff was due to a personal failure; that you will always lose any job you might manage to get; and that your situation is hopeless. On the basis of these judgments, you will begin to feel depressed. In contrast, if you were not influenced by negative triad beliefs, you would not question your self-worth too much, and might respond to the lay off by dusting off your resume and initiating a job search.

Beyond the negative content of dysfunctional thoughts, these beliefs can also warp and shape what someone pays attention to. Beck asserted that depressed people pay selective attention to aspects of their environments that confirm what they already

know and do so even when evidence to the contrary is right in front of their noses. This failure to pay attention properly is known as faulty information processing. Particular failures of information processing are very characteristic of the depressed mind. For example, depressed people will tend to demonstrate selective attention to information, which matches their negative expectations, and selective inattention to information that contradicts those expectations. Faced with a mostly positive performance review, depressed people will manage to find and focus in on the one negative comment that keeps the review from being perfect. They tend to magnify the importance and meaning placed on negative events, and minimizes the importance and meaning of positive events. All of these maneuvers, which happen quite unconsciously, function to help maintain a depressed person's core negative schemas in the face of contradictory evidence, and allow them to remain feeling hopeless about the future even when the evidence suggests that things will get better.

2.1.3.2 Rational Emotive Therapy (RET) of depression.

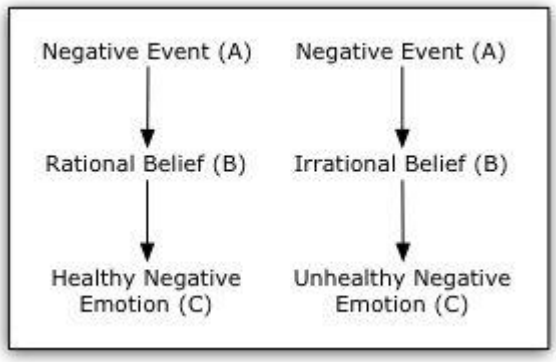
Rational emotive behaviour therapy focuses on uncovering irrational beliefs which may lead to unhealthy negative emotions and replacing them with more productive rational alternatives.

Ellis (1958) was an early and assertive advocate of direct form of cognitive theory which he originally described as rational psychotherapy, a term which he later expanded into rational-emotive psychotherapy. He argued that emotional or psychological disturbances are largely a result of illogical or irrational thinking and that if one can learn to maximize his rational and minimize his irrational thinking, he can rid himself of most of his emotional or mental unhappiness (Ellis, 1962).

Rational emotive behaviour therapy ('REBT') views human beings as 'responsibly hedonistic' in the sense that they strive to remain alive and to achieve some degree of happiness. However, it also holds that humans are prone to adopting irrational beliefs and behaviours which stand in the way of their achieving their goals and purposes. Often, these irrational attitudes or philosophies take the form of extreme or dogmatic 'musts', 'shoulds', or 'oughts'; they contrast with rational and flexible desires, wishes, preferences and wants. The presence of extreme philosophies can make all the difference between *healthy* negative emotions (such as sadness or regret or concern) and *unhealthy* negative emotions (such as depression or guilt or anxiety). For example, one person's philosophy after experiencing a loss might take the form: "It is unfortunate that this loss has occurred, although there is no actual reason why it should not have occurred. It is sad that it has happened, but it is not awful, and I can continue to function." Another's might take the form: "This absolutely should not have happened, and it is horrific that it did. These circumstances are now intolerable, and I cannot continue to function." The first person's response is apt to lead to sadness, while the second person may be well on their way to depression. Most importantly of all, REBT maintains that individuals have it within their power to change their beliefs and philosophies profoundly, and thereby to change radically their state of psychological health.

REBT employs the 'ABC framework' — depicted in the figure below — to clarify the relationship between activating events (A); our beliefs about them (B); and the cognitive, emotional or behavioural consequences of our beliefs (C). The ABC model is also used in some renditions of cognitive therapy or cognitive behavioural therapy, where it is also applied to clarify the role of mental activities or predispositions in mediating between experiences and emotional responses.

The figure below shows how the framework distinguishes between the effects of rational beliefs about negative events, which give rise to healthy negative emotions, and the effects of irrational beliefs about negative events, which lead to unhealthy negative emotions.



In addition to the ABC framework, REBT also employs three primary insights:

1. While external events are of undoubted influence, psychological disturbance is largely a matter of personal *choice* in the sense that individuals consciously or unconsciously *select* both rational beliefs and irrational beliefs at (B) when negative events occur at (A)
2. Past history and present life conditions strongly *affect* the person, but they do not, in and of themselves, *disturb* the person; rather, it is the individual's responses which disturb them, and it is again a matter of individual choice whether to maintain the philosophies at (B) which cause disturbance.
3. Modifying the philosophies at (B) requires persistence and hard work, but it can be done.

The main purpose of REBT is to help clients to replace absolutist philosophies, full of 'musts' and 'shoulds', with more flexible ones; part of this includes learning to accept that all human beings (including themselves) are fallible and learning to increase their tolerance for frustration while aiming to achieve their goals. Although emphasizing the same 'core conditions' as person-centred counselling — namely, empathy, unconditional positive regard, and counsellor genuineness — in the counselling relationship, REBT views these conditions as neither necessary nor sufficient for therapeutic change to occur.

The basic process of change which REBT attempts to foster begins with the client acknowledging the existence of a problem and identifying any 'meta-disturbances' about that problem (i.e., problems about the problem, such as feeling guilty about being depressed). The client then identifies the underlying irrational belief which caused the original problem and comes to understand both why it is irrational and why a rational alternative would be preferable. The client challenges their irrational belief and employs a variety of cognitive, behavioural, emotive and imagery techniques to strengthen their conviction in a rational alternative. (For example, rational emotive imagery, or REI, helps clients practice changing unhealthy negative emotions into healthy ones at (C) while imagining the negative event at (A), as a way of changing their underlying philosophy at (B); this is designed to help clients move from an intellectual insight about which of their beliefs are rational and which irrational to a stronger 'gut' instinct about the same.) They identify impediments to progress and overcome them, and they work continuously to consolidate their gains and to prevent relapse.

To further this process, REBT advocates 'selective eclecticism', which means that REBT counsellors are encouraged to make use of techniques from other

approaches, while still working specifically within the theoretical framework of REBT. In other words, REBT maintains theoretical coherence while pragmatically employing techniques that work.

Throughout, the counsellor may take a very directive role, actively disputing the client's irrational beliefs, agreeing homework assignments which help the client to overcome their irrational beliefs, and in general 'pushing' the client to challenge themselves and to accept the discomfort which may accompany the change process.

As one leading proponent of REBT has indicated, REBT is easy to practise poorly, and it is from this that one immediate criticism suggests itself from the perspective of someone who takes a philosophical approach to life anyway: inelegant REBT could be profoundly irritating! The kind of conceptual disputing favoured by REBT could easily meander off track into minutiae relatively far removed from the client's central concern, and the mental gymnastics required to keep client and therapist on the same track could easily eat up time better spent on more productive activities. The counsellor's and client's estimations of relative importance could diverge rather profoundly, particularly if the client's outlook really does embody significant irrationalities. Having said all that, each of the preceding sentences includes the qualifier '*could*', and with a great deal of skill, each pitfall undoubtedly could be avoided.

Perhaps more importantly, it would appear that the need to match therapeutic approach with client preference is even more pressing with REBT than with many others. In other words, it seems very important to adopt the REBT approach only with clients who truly are suitable, as it otherwise risks being strongly counter-productive. On this point, however, it is crucial to realize that some clients specifically do

appreciate *exactly* this kind of approach, and counsellors who are unable or unwilling to provide the disputation required are probably not right for those clients

Ellis and Beck appear to share similar views on cognitive theory. However, Kendall, Maaga, Ellis, Bernard, Guiseppe, and Kassinove (1995), maintained the fact that Ellis was prescience in identifying the interaction between cognition and emotion as central question in cognitive approaches to therapy and his early recognition of the power of behavioral change did not attract much research and Clinical attention as did that of Beck. This was traced to the form of their writings. They claimed that the writings of Ellis were more anecdotal and loosely formulated and that the thrust of Beck's early work was on understanding and treating depression which was a Clinical problem that remained essentially unsolved by behavior therapists.

2.1.3.3. The Hopelessness Theory of Depression,

It was proposed by Abramson, Metalsky, & Alloy and is based on Seligman's work on learned helplessness and attribution styles. The hopelessness theory of depression posits that when confronted with a negative event, people who exhibit a depressogenic inferential (thinking) style, defined as the tendency to attribute negative life events to stable (enduring) and global (widespread) causes, are vulnerable to developing depression because they will infer that: a) negative consequences will follow from the current negative event, and b) that the occurrence of a negative event in their lives means that they are fundamentally flawed or worthless. For example, consider a woman whose fiancé breaks off their engagement. If she attributes the cause of the break-up to her personality flaws, a stable-global cause that will lead to many other bad outcomes for her, or if she infers that a consequence of the break-up is that she will never marry or have children, or if she infers that without a lover, she is worthless, she is likely to

become hopeless and develop the symptoms of depression. Thus, according to hopelessness theory, a specific cognitive vulnerability operates to increase the risk for depression through its effects on processing or appraisals of personally relevant life experiences.

Learned helplessness also fits with certain neuroscience findings; for example, depressed patients who see themselves as helpless tend to show high level of MHPG, a product of norepinephine metabolism (Samson, Mirin, Hauser, Fenton, & Schidkrant, 1992). Norepinephine abnormalities are often found in depressed people. In addition, Positron Emission Tomography (PET) scans of people doing unsolvable problems which tend to produce learned helplessness show that learned helplessness is associated with increased brain activity in the limbic system. The limbic system is implicated in the processing of negative emotions such as depression (Schneider, Gur, Alavi, Seligman, Mozley, Smith, Mozley & Gur, 1996).

When the theory of learned helplessness was developed, it had certain weaknesses. As Seligman and his colleagues pointed out, the model explained the passivity characteristic of depression but did not explain equally the characteristic sadness, guilt, and suicidal thoughts, neither did it account for the fact that different cases of depression varies considerably in intensity and duration. To fill these gaps, Abramson and colleagues adapted the model from helplessness theory. According to this view, depression depends not only on the belief that there is a lack of control over reinforcement but also on the belief that negative events will persist or reoccur. In HIV/AIDS situation, people living with the disease ruminate over a lot of things like regret and blame for being careless (involving self in risky behaviors such as indulging in unprotected sex, having multiple sexual partners

which could have led contacting the disease, whether to start and adhere to the antiretroviral treatment (ART), whether one will live to actualize one's dreams or not and others).

Experiments with human beings have yielded results similar to those experiments done with animals. People who are subjected to inescapable noise or inescapable shock, or who are confronted with unsolvable problems, fail later to escape noise and shock, or solve simple problems (Hiroro & Seligman, 1975).

Seligman noted similarities between the manifestations of helplessness observed in laboratory animal studies and at least some of the symptoms of depression. Many depressed people, like the animals appear passive in the face of stress, failing to initiate action that might allow them to cope. They develop anorexia, having difficulty eating or retaining what they ate and lose weight.

The cognitive theory is applicable to the Nigerian context, because following the difficulties many Nigerians do encounter today, such as loss of properties during fire disaster in the market, destruction of peoples properties during political, religious crisis or boundary or ethnic clashes, or even terminal illness like cancer most times some of the victims are affected psychologically such as depression and anxiety. Some think that the problem is the end of the road; negative thoughts fill their minds and they may find it very difficult to cope. Because of this, some persons do commit suicide or lack of motivation to continue struggling or resort to the use of psychoactive substances as a means of coping (Clinical observation by the researcher).

This study is relevant to the study because it helps us to understand reasons why people cannot endure frustration and commit sucide enventually.

2.1.4 COGNITIVE BEHAVIOR THERAPY OF DEPRESSION

Aaron T. Beck and colleagues initially developed cognitive therapy as a treatment for depression. Cognitive behavioral treatment (CBT) of depression involves the application of specific, empirically supported strategies focused on depressogenic information processing and behavior. In order to alleviate depressive affect, treatment is directed at the following three domains: cognition, behavior, physiology. In the cognitive domain, patients learn to apply cognitive restructuring techniques so that negatively distorted thoughts underlying depression can be corrected, leading to more logical and adaptive thinking. Within the behavioral domain, techniques such as activity scheduling, social skills training, and assertiveness training are used to remediate behavioral deficits that contribute to and maintain depression (e.g., social withdrawal, loss of social reinforcement). Finally, within the physiological domain, patients with agitation and anxiety are taught to use imagery, meditation, and relaxation procedures to calm their bodies.

CBT is oriented towards empowering the patient. Within this specific, brief psychotherapeutic treatment modality, the emphasis is on providing patients with skills to offset their depression. One primary goal of CBT is to facilitate the use of treatment techniques outside therapy sessions to create a "positive emotional spiral" wherein can implement specific strategies to offset their depressive mood (e.g., cognitive restructuring is used to offset negative thought patterns and the consequent depressive affect, scheduling pleasant activities is used to offset decreased reinforcement secondary to social withdrawal).

2.1.4.1 Efficacy of Cognitive Behavior Therapy for Depression

Since cognitive therapy was first formulated by Beck numerous studies have demonstrated the efficacy of cognitive therapy for depression. The first landmark

study conducted by Rush and colleagues in the late seventies demonstrated that cognitive therapy was more effective than tricyclic antidepressant therapy in patients suffering from clinical depression. In contrast with previous outcome research which demonstrated that psychotherapies were no more effective than pill-placebos and less effective than antidepressant medications, the Rush et al. study was the first to show that a psychosocial treatment was superior to pharmacotherapy in the treatment of depression. Further, a follow-up study conducted twelve months post-treatment showed that relapse rates were lower among patients who received CT (39%) versus those who received anti-depressant medication (65%), although this difference did not reach statistical significance.

In the two decades since the initial trial, many controlled trials have been undertaken to replicate these findings. Although many experts now believe that the Rush study was sufficiently flawed to negate study findings, many qualitative and quantitative reviews now conclude that cognitive therapy: (1) effectively treats depression,(2) is at least comparable, if not, superior to medication treatment, and (3) may have lower rates of relapse in comparison to medication treatments . As a result, cognitive therapy has gained widespread acceptance as a first-line treatment for depression, and cognitive behavioral therapy is one of only two psychotherapies included in the guidelines for the treatment of depression published by the Agency for Health Care and Policy Research (AHCPR).

Depressed people commit many types of errors in thinking, such as jumping to negative conclusions on the basis of little evidence, ignoring good events while focusing only on negative events and exaggerating these negative events that support their negative triad. Often, these negative thoughts are so automatic that depressed people do not realized how they are interpreting situation. A wide range of studies have supported the hypothesis that depressed

people show these negative ways of thinking, and some longitudinal studies have shown that these thinking styles predict depression over time (Abramson, Alloy, Hankin, Haeffel, MacCoon & Gibb, 2002). Beck's theory led to one of the most widely used and successful therapies for depression which is cognitive-behavioral therapy.

In cognitive learning model of depression, Seligman (1974) suggested that depression may be understood as analogous to the phenomenon of learned helplessness. The basic premise of the learned helplessness theory is that an individual's passivity and sense of being unable to act and control his or her own life is acquired through unpleasant experiences and traumas that the individual tried unsuccessfully to control; this then brings on depression (Davidson & Neale 2001).

This phenomenon was first demonstrated with laboratory dogs. After exposing a number of dogs to inescapable electric shock, Seligman and his colleagues found that, when the same dogs were later subjected to escapable shocks, they either did not initiate escape response or were slow and inept at escaping. They appear to lose the ability to learn to respond in an effective way to painful stimulation (Hiroto & Seligman, 1975). On the basis of this, Seligman felt that learned helplessness in animals could provide a model for at least certain forms of depression in human beings.

The investigators concluded that, during the first phase of the experiment, when the shocks were inescapable, the dogs had learned that the shock was uncontrollable – a lesson they continued to act upon even in the second phase of the experiment when it was possible to escape the shocks (Maier, Seligman & Solomon, 1969). This formulation is consistent with the finding that, when there

is a clear precipitating event for depression, it is often an uncontrollable loss. When a person holds the belief that bad things will happen and that there is nothing one can do about it (there is lack of control over reinforcement) – he or she becomes hopeless, and it is this hopelessness that is the immediate cause of depression (Abramson, Metalsky & Alloy, 1989). In fact, Abramson and her co-workers have proposed that "hopelessness depression" constitutes a distinct subtype of depression, with its own set of causes: negative inferential styles combined with stress, and others which include; passivity, sadness, suicidal tendencies, low self-esteem and others.

2.1.5 Behavioral Theories of Depression

Behavior theory focuses on the study of observable and measurable responses of specific stimuli. Its primary task is the prediction and control of behavior. However, behavioral theories of depression center on several perspectives which include extinction and aversive social behavior. This was propounded by Watson (1913) with the goal of prediction and control of behavior. Behavioral theories of depression were discussed under two perspectives: extinction and aversive social behavior.

a. Extinction

Many behaviorists regard depression as the result of extinction of positive reinforcement (Fester, 1978; Lewinsohn, 1974). Lewinsohn pointed out that the amount of positive reinforcement a person receives depends on three broad factors which include:

1. The number of and range of stimuli that are reinforcing to that person;
2. The availability of such rein forcers in the environment;

3. The person's skill in obtaining reinforcement.

Changes in person's environment may affect any one of these factors, for example, a person diagnosed with HIV may be scared to live with the physical symptoms such as skin rashes, loss of weight, stigmatization and others. This may eventually lead to loss of social and occupational functioning. The individual may not know how to obtain reinforcement and hence withdraws to him/herself. A number of studies by Lewinshohn, Sullivan & Grosscup (1980) have produced results consistent with the hypothesis. For example, one defection to this hypothesis has been the widely held assumption that depressives are immune to reinforcement.It is not that they lack sources of pleasure but, rather that they have lost their ability to experience pleasure. Oftentimes, some depressed persons show an elevation of mood if they learn to decrease the frequency of unpleasant events and focus on issues that remind them about their pleasant experiences and encounters.

a. Aversive Social Behaviour

Some researchers have found that depressives are more likely than non depressives to elicit negative reactions from people with whom they interact (Coyne, 1990; Segrin & Abramson, 1994) and this finding has formed the basis of interpersonal theories of depression. According to this theory, depressives have an aversive behavioral style in which they constantly seek reasons to elicit "caring" behavior from people whom they feel no longer care enough. Instead of love, however, the impression the depressives are likely to get from their put-upon families and friends is shallow reassurance of rejection which simply aggravates their depression (Joiner & Metalsky, 1995).

Depression then is a cry for help, but one that rarely works. An alternative interpersonal theory is that depressives actually seek out rejection for this is more familiar and predictable to them than positive feedback (Giesler, Joseph & Swann, 1996). In support of this interpersonal hypothesis, some studies have found that rejecting responses from friends and family do tend to maintain or exacerbate depression (Joiner, 1995). For instance, Hooley and Teasdale (1989) found that the depressed patients whose spouses were critical toward them were more likely to suffer a relapse of depression in the next nine months that were those with more accepting spouses. Some evidences suggest that this style is only present during the depressed episode and disappears when the depressed person recovers (Rhode, Lewinsohn & Seeley, 1990). Whether or not it predates depression, the depressives' poor interpersonal skills probably help to maintain their depression.

This theory is relevant in explaining why some people when they loose a close friend or spouse who used to give them support, go into depression. This fact appears to be applicable in every culture including Nigeria. This is due to the fact that the one who used to give them succor is no more. In illness situation especially when the physical and psychological strengths begin to wane, the individual may begin to feel sad as well as depressed.

2.1.6 Psychodynamic Theory of Depression

This theory came as a challenge to Kraeplin's (1922) biogenic theory of mood disorder that argued that depression was not a symptom or organic dysfunction but a massive defense mounted by the ego against intra-psychic conflict. Freud described depression as a response to loss (real or imagined) but one in which the person's sorrow and rage in the face of that loss remain unconscious, thus weakening the ego.

Freud's formulation was actually an elaboration of a theory put forth by Abraham (1948). Abraham suggested that depression arises when one looses a love object toward whom one had ambivalent, positive and negative feelings. In the face of the love objects desertion, the negative feelings turn to intense anger. At the same time, the positive feelings give rise to guilt, a feeling that one failed to behave properly toward the now-lost love object.

However, because of this guilt and because of early memories in which the primary love object was incorporated by the infant, the grieving person turns his or her anger inward, thus producing despair which is depression. In recent times, there have emerged many psychodynamic perspectives of depression, yet they share a certain number of core assumptions (Bemporad, 1988; Blatt & Homann, 1992). These assumptions include: First, it is generally believed that depression is rooted in a very early defect, often the loss or threatened loss of parent (Bowlby, 1973). Second, the primal wound is reactivated by a recent blow, such as a divorce of job loss. Whatever the precipitating event, the person is plunged back into the infantile trauma. Third, a major consequence of this regression is a sense of helplessness in the face of harm. Feeling incapable of controlling his or her world, the depressive simply withdraws from it. Fourth, it is widely agreed that loss of self-esteem is a primary feature of depression. Fenichel (1945) described depressives as "love addicts", trying continually to compensate for their own depleted self-worth by seeking comfort and reassurance from others.

Considerable research has documented that people who suffer from parental loss early in life can suffer from depression. For example, in the classic Brown and Harris's (1987) study, 40 out of 458 women had lost their mother before the age of 11; however, among those 40, 42 percent developed Clinical depression in adulthood;

of the other 418 women who had lost their mother at an early age, only 14 percent had developed Clinical depression in adulthood.

Thus, the incidence of depression was three times higher in women who lost their mother before the age of 11 (Bowlby, 1982). In addition, Brown and Harris (1987) found that the type of loss (death vs. divorce or separation) also affect the severity of depression experienced in adulthood. Women who had experienced loss by death were more likely to develop severe psychotic depression, whereas women who had experienced loss by divorce or separation were more likely to develop less severe neurotic depression. However, there are a number of studies that have not found any evidence that early parental loss produces a vulnerability to depression. Gotlib and Hammen (1992) concluded that it is the quality of parenting that has greater impact on the child following parental loss. In cases of parental loss where the child continues to receive good quality care from the surrogate mother, a vulnerability to depression may not be created. However, if parental loss is followed by poor parental care, a vulnerability to depression may become obvious (Brown & Harris, 1987).

This theory is equally applicable in Nigerian context. Some orphans or children who received harsh treatment from their surrogate parents have been found to live with repressed aggression as well as depression (Clinical observation by the researcher), willingness to move through fear to confront conflicts and discomforting realities. It requires learning to face and master rather than withdraw and avoid. Any effective therapy promotes growth along the dimensions of self-acceptance, self responsibility and all the other practices that support self-esteem (Branden, 1994).

On the other hand, some researchers have identified some mediators in psychotherapy of which self-esteem is one (Steketee & Chambless, 1992; Whisman,

1993). These mediators are seen as prognostic indicators that could be used to target individuals who would likely benefit from psychotherapy and it is an important area of inquiry with both Scientific and Clinical implications.

2.1.7 Theories of group therapy

Group psychotherapy is a treatment modality, in which carefully selected persons who are emotionally ill meet in a group guided by a trained therapist and help one another effect personality change. Group therapy is a widely accepted psychiatric treatment modality that uses therapeutic forces within the group, constructive interactions between members, and interventions of a trained leader to change the maladaptive behavior, thoughts, and feelings of emotionally distressed individuals. Yalom,(1980) stated that group therapy is as helpful as individual therapy and sometimes more effective. One reason for this efficacy is that group intrinsically has many rewarding benefits such as reducing isolation and enabling members to witness the recovery of others, and these qualities draw patient into a culture of recovery.

Psychotherapeutic use of groups was made in the US by medical doctors with the aim of psycho-education. Pratt(1908, 1922) and Lazell (1921) addressed lectures to different groups of patients and their aim was to increase self- control by providing them with more information about their diseases.(group of people with tuberculosis.). These lectures were provided in small, median or large groups, and Pratt referred to his groups as Thought control classes, and Lazell as Etiology spiel.

Similar developments in Vienna were described as a guidance groups by Adler in late 1920s. He provided guidance information to group of people on their health conditions. The first psychoanalysts to bring together patients suffering from neurotic symptoms in a group (using psychoanalytic technique) was T. Burrow

(1928) in U.S. His starting point was the perception of humans as a social being and of the group as a natural focus of treatment. His aim was to make conscious, both latent and repressed materials through the 'here and now' interactions within the group. He named his method group analysis.

Also, from 1928 through to 1938, Jacob Moreno used the theatre stage for an understanding of unconscious conflicts. He offered psychodrama demonstration at Carnegie Hall. He also used psychodrama in a small group setting for psychotherapy at the Mount Sinai Hospital in New York City, and developed the method of sociometry in 1938. Moreno worked mainly with adults suffering from neurotic illnesses and recommended the exclusion of psychopaths, alcoholics, and psychotics from group.

After the Second World War, people with behavioral problems were on the increase. In 1940s, several psychoanalysts used their psychoanalytical understanding to work with patients in a group setting: Louis Wender(1940), Paul Schilder (1940) and Alexander Wolf (1949), all worked with resistance and transference processes among patients that are mainly adults, suffering from neurotic illness. They see a group therapist as a symbolic parent and other patients as representing siblings. Their aim was to provide social insights through interpersonal exchange of ideals.

They also placed emphasis on reducing patient's isolation through group interaction. All these psychoanalytically based group therapy approaches had one thing in common: they used a method of application of individual psychoanalysis to a group setting with the aim of social integration. The therapeutic emphasis was kept on the individual patient within the group setting.

Yalom (1975/ 1980) provided more eclectic or integrative approaches to group psychotherapy. Today, group therapy has many approaches such as Psycho-analytic frame of reference. Others use therapy techniques, such as transactional group therapy – devised by Eric Berne. Behavioral group therapy which relies on conditioning techniques based on learning theory. Fiedler, (1996) and Free (1999) noted that psycho educational approach is nowadays used in the application of cognitive / behavioral therapy in groups. They defined curative factors as reeducation, socialization, the imbuing of an individual with hope, the raising of morale and the emotional developments occurring during the teaching process in a group.

Rutan & Stone (2001) developed the concept of psychodynamic group psychotherapy in the U.S, which offered an integrative approach.They tried to integrate all so-called modern theories of group psychotherapy with the aim of an integrative conceptualization. Which are as follows: - Intrapsychic, (typical defenses, character formation,) interpersonal, (relational styles and roles) and social psychological components (group norms, values, and restrictions) of group psychotherapy.

2.2 Empirical Review

2.2.1 Age and Depression

There is an increasing recognition in the psychology literature that the presence of depressive disorders often start in the period of childhood and adolescence (Chang 2009), and depression which occur during this transition period often persists into adulthood (Colman et al. 2007). Adolescents who experience depression often struggle with depression throughout their lives (Lewinsohn et al. 1999), and in many cases, early onset of depression predicts more severe depression

during adulthood (Weissman et al. 1999). Detection and effective treatment of early-onset major depressive disorders can be more important than for late-onset depressive symptoms. Greden (2001) documented that early-onset depression (before the age of 21 or 22) is associated with longer first episodes, higher rates of recurrence of major depression, higher overall rates of comorbid personality disorders, and longer hospitalizations. Berndt et al. (2000) found that early-onset depression can lead to reduced educational attainment and other human capital loss, particularly for women; a randomly selected 21-year-old woman with early-onset major depressive disorder in 1995 could expect future annual earnings that were 12%-18% lower than those of a randomly selected 21-year-old woman whose onset of major depressive disorder occurred after age 21 or not at all.

Depression is not a normal part of aging. Studies show that most seniors feel satisfied with their lives, despite having more illnesses or physical problems (Chang 2009). However, when older adults do have depression, it may be overlooked because seniors may show different and less obvious symptoms. They may be less likely to experience or admit to feelings of sadness or grief. Sometimes it can be difficult to distinguish grief from major depression. More than 1 out of 20 Americans 12 years of age and older reported current depression in 2005–2006.

Empirical evidence on the effect of age on mental health is diverse. Analyses of depression in late life (i.e., above age 65) reveal a modest effect of age (Prince et al., 1999b), a strong association between the prevalence of symptoms of depression and age (Stordal et al., 2003; Castro-Costa et al., 2007) or find no overall tendency of depression to rise with age (Trollor, Anderson, Sachdev, Brotady & Andrews, 2007; Korten & Henderson, 2000), except among the oldest old (Copeland et al., 1999b).

Moreover, international comparisons reveal striking differences in depressive symptoms among countries, and even contradictory results concerning the development of mental health with age (Blazer, 1999; Prince et al., 1999b; Copeland, 1999; Copeland et al., 1999a; EORG, 2003; EC, 2004a). These studies are primarily based on national, regional and local surveys, and less often on international comparative surveys like Eurobarometer, European Study of Epidemiology of Mental Disorders/Mental Health Disability (ESEMeD/MHEDEA 2000), or Outcomes of Depression International Network (ODIN). Thus, the majority of international comparative studies suffer from the lack of comparable data (Copeland et al., 1999a; EC, 2004a). Methodological differences between studies as well as non-representativeness of the national data did not allow us to draw any conclusions about cross-cultural and geographical variation (Beekman, Copeland & Prince, 1999; EC, 2004b). Older adults also may have more medical conditions such as heart disease, stroke, or cancer, which may cause depressive symptoms. Or they may be taking medications with side effects that contribute to depression (Chang 2009). Some older adults may experience what doctors call vascular depression, also called arteriosclerotic depression or subcortical ischemic depression, Copeland et al., 1999a. Vascular depression may result when blood vessels become less flexible and harden over time, becoming constricted. Such hardening of vessels prevents normal blood flow to the body's organs, including the brain. Prince et al., 1999b). Those with vascular depression may have, or be at risk for, co-existing heart disease or stroke. Although many people assume that the highest rates of suicide are among young people, older white males age (85 and above) have the highest suicide rate in the United States, Prince et al., 1999b). Many have a depressive illness that their doctors are not aware of even though many of these suicide victims visit their doctors within 1 month of their deaths. Older adults with depression improve when they receive

treatment with an antidepressant, psychotherapy, or a combination of both (Prince et al., 1999b).

Children who develop depression often continue to have episodes as they enter adulthood. Children who have depression also are more likely to have other more severe illnesses in adulthood (Copeland et al., 1999a). A child with depression may pretend to be sick, refuse to go to school, cling to a parent, or worry that a parent may die. Older children may sulk, get into trouble at school, be negative and irritable, and feel misunderstood. Because these signs may be viewed as normal mood swings typical of children as they move through developmental stages, it may be difficult to accurately diagnose a young person with depression. Before puberty, boys and girls are equally likely to develop depression (Chang 2009). By age 15, however, girls are twice as likely as boys to have had a major depressive episode. Depression during the teen years comes at a time of great personal change—when boys and girls are forming an identity apart from their parents, grappling with gender issues and emerging sexuality, and making independent decisions for the first time in their lives. Depression in adolescence frequently co-occurs with other disorders such as anxiety, eating disorders, or substance abuse (Copeland et al., 1999a). It can also lead to increased risk for suicide. An NIMH-funded clinical trial of 439 adolescents with major depression found that a combination of medication and psychotherapy was the most effective treatment option(Chang 2009. Previous research has found that age is one of the demographic characteristics that accounts for much of the variance in the prevalence of depression. A Canadian National Population Health Survey 2011 found that the prevalence of 12-month depression varied in men from "too low to report" for men over 65 to a high of 5.2% for the 12 to 24 age group. Women's prevalence also varied by age, ranging from a low of 3.1% for women over 65 to a high of 9.6% for

the 12 to 24 age group . The Ontario Health Survey2012 found comparable variation based on age. This pattern is consistent with findings from Australia.

2.2.2 Gender and Depression

"Gender differences in depressive symptoms appear to emerge in early adolescence and then remains throughout the adult life span" (Nolen-Hoeksema, Larson, & Grayson, 1999). Consistent findings indicate that adolescent girls develop depressive symptoms at an earlier age than do adolescent boys (Ge & Conger, 2003). Emerging gender differences can be caused by individual vulnerability, life stress, and pubertal transitional challenge. Although girls and boys go through puberty at the relatively same age, it has been suggested that girls are more vulnerable to depression than boys even before adolescence (Ge & Conger, 2003). Amoran, Lawoyin and Lasebikan (2007) conducted a study with 1105 participants in Oyo State and found that 5.2% of them had depression. They also found depression to be more prevalent in women (5.7%) than in men (4.8%). In addition, they found that depression was more common in adolescents and in the rural areas.

Ge& Conger, (2003) conducted a study in which they hypothesized that;

i. Girls will demonstrate higher average levels of depressive symptoms than adolescent boys will during adolescence.

ii. The higher average level of depressive symptoms among girls, compared with boys, will become evident during early adolescence. Boys and girls with advanced pubertal status during early adolescence will manifest higher levels of depressive symptoms.

iii. Boys and girls with higher levels of depressive symptoms in early adolescence will show higher levels of depressive symptoms in mid- and late adolescence.

iv. Early depressive symptoms, the pubertal transition, and stressful life events will have interactive as well as additive main effects on risk for depressive symptoms.

v. The interactive and additive effects of early depressive symptoms, the pubertal transition, and stressful life events will explain a significant portion of the association between gender and depressive symptoms.

The choice of method was a 6-year longitudinal study of 451 families that lived in central Iowa. Interviewers visited each family and family members was asked to independently complete a set of questionnaires focusing upon individual characteristics, emotions and life events experienced by family members. These independently reported emotions and events were used to come up with the conclusion (Ge & Conger, 2003).

The results found that early depressive symptoms carry forward to mid- and late adolescence and that the interaction between gender-linked vulnerabilities (diathesis) and the new biological and social challenges of early adolescence (stress) creates greater risk for depression for adolescent girls than boys (Ge & Conger, 2003). Studies have also found higher levels of depressive symptoms in girls than in boys as young as 12 years of age and have consistently found gender differences from then on out (Nolen-Hoeksema & Girgus, 1994). However, although this conclusion was found from the experiment, this has never been reported in earlier studies of adolescent depression.

Yet again, it is difficult to determine why women are more susceptible to depression. The experiment provides conclusions and theories that have never been proven prior. It is clear that girls are more susceptible to depression even in adolescence, but there is no concrete evidence to prove why. However, depressives (girls and boys) were found to come from families in which there was marked striving for prestige with the patient as the instrument ofthis need; the family showed marked concern for social achievement and the childhood background was characterized by envy and competitiveness (Weissman & Paykel, 1974).

Being depressed as a female adolescent can have consequences in the long run in terms of social functioning, career, and enjoyment of life. Theoretically, if one were to be depressed in high school, then their grades would suffer. If their grades were to suffer, then their chances of entering a good college would dwindle. If they cannot enter a top-notch college, then they might not be able to get the career they want, and with that they would not be able to enjoy their job and feel like they have missed out on life (Nolen-Hoeksema, Larson, & Grayson, 1999).

The different experiences of each gender can also be the cause of a mood disorder. The experience can vary by the age of the children, adolescences, or adults. For example, after the age of 15, females are twice as likely to become depressed as compared with men and in another study of 11-year olds, only 2.5% males met the criteria for major depression while only 0.5% females met the criteria, however in a study of 14-16 year olds, 13% of the females met the criteria while 3% of the boys did (Nolen-Hoeksema, & Girgus, 1994). This abrupt rise of depressive disorders in females during the mid-to-late adolescence years can be attributed to the more concerns a girl has as compared to boys. These concerns and worries can range from

their achievements or lack of, body dissatisfaction, sexual abuse, and low self-esteem (Lewinsohn, Gotlib, & Seeley, 1997).

Do not be mistaken that females are the only gender that that can become depressed; a good number of males can develop a unipolar mood disorder. In the average lifetime, 49% of all males will experience a depressive episode (as compared with 63% of all females). Males will become sad and dejected for different reasons, such as intimate relationships. When an intimate relationship ends, males are more likely to become depressed at the loss than females (Hankin et al., 1998). This could be attributed to the male's primal desire to have a mate so he will be able to continue his family name.

Depression may be becoming noticeable more than in years past, but that is not the only pattern researchers are noticing. Depressive symptoms are more common in female students than in their male counterparts (Barrett & Boggiano, 1991). Females are reported to have a ten percent higher chance of experiencing depressive symptoms, with an average of 50 percent of women reporting difficulty functioning in day-to-day activities ("Depression; survey shows," 2004).

A study conducted by Marty Barrett and Anne Boggiano at the University of Colorado discussed two possible reasons for increased levels of depression on female students (1991). Body image and concern over interpersonal relations are areas that receive more acknowledgements from women than men. These authors noted that females are heavily socialized to be concerned about others and establishing intimate relationships. They considered that this particular area of a female's identity might become the leading drive for social interaction and fulfillment. Moreover, 90 percent of female emerging adults reported dissatisfaction with their current body image and a desire to lose at least ten pounds. An inability to reach the ideal body type, as

portrayed by the media, can promote poor self-image and result in heightened levels of depression among women (Nolen-Hoeksema, Larson, & Grayson, 1999).

Unlike women, men tend to have a more accurate body image and a healthy physical expectation. Although men may not represent the majority of depressed college students, they do make up a significant amount of the statistic. Stressors for men tend to be related more toward academic achievement and intelligence levels (Barrett & Boggiano, 1991). Prevalence for depression has also been found to vary considerably based on gender. Consistently, women have nearly double to triple the prevalence rates for 12-month depression compared to men. There are also gender differences in both the use of outpatient treatment and response to antidepressants. "Gender differences in depressive symptoms appear to emerge in early adolescence and then remains throughout the adult life span" (Nolen-Hoeksema, Larson, & Grayson, 1999). Consistent findings indicate that adolescent girls develop depressive symptoms at an earlier age than do adolescent boys. Emerging gender differences can be caused by individual vulnerability, life stress, and pubertal transitional challenge. Although girls and boys go through puberty at the relatively same age, it has been suggested that girls are more vulnerable to depression than boys even before adolescence (Ge & Conger, 2003).

It has long been observed that women are about twice as likely to become clinically depressed as are men (Nolen-Hocksema, 2002). These differences occur in most countries around the world. In most countries, these gender differences start in adolescence and continue until about age 65 when they seem to disappear (Carson, Butcher, & Mineka, 1998). Several factors have been identified as responsible for these differences. These include:

The hormonal factor: most women know too well the physical and emotional changes that occur before menstruation; when abdominal bloating, breast tenderness, headache, anxiety, irritability or a blue mood herald premenstrual syndrome (PMS). This is as a result of hormonal changes. For some women, the symptoms associated with PMS are minor and short lived, but small percentage has such severe and disabling premenstrual symptoms of depression and hopelessness that their lives, jobs and relationships are disrupted at the period of their menstruation (Nolen-Hoeksema, Larson, & Grayson, 1999). This condition is known as premenstrual dysphoric disorder (PDD) and it may require treatment with hormone or antidepressants. Although the exact interaction between depression and premenstrual syndrome remains unclear, some researchers suggest that cyclic changes in estrogen, progesterone, and other hormones can disrupt the function of brain chemicals such as serotonin that controls mood (Nolen-Hoeksma, 1990).

Pregnancy is another factor which may be implicated in depression. The dramatic hormonal changes that occur during pregnancy along with life, work and relationship change mood and in some cases may trigger depression (Nolen-Hoeksema, 2002). Other factors that can increase the risk of depression during pregnancy include previous depression or PMD, marital strife, poor social support, especially from the spouse and ambivalence about being pregnant, miscarriage or unwanted (unplanned) pregnancy (Nolen-Hoeksema, Larson, & Grayson, 1999).

Postpartum depression is another form of depression associated with pregnancy in some women. This is a form of depression occurring occasionally in mothers between 3 days to 6 weeks after giving birth with symptoms from relatively mild to major depression (Ogun, 2006). Although, having a new baby is generally exciting, but about half of women find themselves sad, angry, irritable and prone to

tears soon after giving birth. These feelings sometimes called the "baby blues or postpartum blues" are normal and generally subside within a week or two (Ogun, 2006). If symptoms are severe with inability to care for the baby and feelings of anxiety, low self-esteem, agitation or thoughts of suicide, the individual may have postpartum depression. This is not really a matter of being unable to cope with a new baby, but rather is probably associated with major hormonal fluctuations that influence mood (Ogun, 2006).

The risk of depression may also increase during the transition to menopause when hormonal level fluctuates erratically, as well as, after menopause when estrogen levels are significantly reduced (Nolen-Hoeksema & Girgus, 1994). Most women who experience uncomfortable menopausal symptoms may not develop, but for women whose sleep is disrupted for long periods of time or who have prior history of depression, this is a vulnerable time (Ogun, 2006). Female biological make up may also influence depression. This is because hormones may alter mood through various stages of life. Psychosocial factors could interact with hormonal changes in women and result in an increased risk of depression. Drastic fluctuations in hormones can have a profound effect on a women's life, coupled with personality factors, relationship issues, socioeconomic factors and others(Ogun, 2006).. Some social factors may contribute to depression among women, for instance, women who were sexually or otherwise abused as children or even battered by their husbands are more likely to experience depression at some point in their lives than those who were not. Also, women who were raped as teenagers or young adults have a higher incidence of depression (Nolen-Hoeksema & Girgus, 1994). Although, sexual abuse occurs in boys and young men, it is more common among young girls and young women.

According to Nolen-Hoeksema, 2002 (in Press) no matter the precise cause of depression, women have different symptoms than men do. Women get married to men; leave their biological family to get initiated into another family. It could be possible that most times, it may not be so easy for a woman to be fully integrated into her husband's home due to some unfriendly attitudes of some members of her husband's family especially in Africa where extended family system is practiced.

Moreover, another factor for women to manifest higher rate of depression than men could also be attributed to differences in the way men and women cope with stress (Eaton et al., 1997). These researchers posit that when women are "down", they tend to ruminate on their state, i.e. focusing on the depression, wondering why it is happening and what it will lead to. Men take the opposite way. They try to distract themselves, with actions such as taking to smoking, even abuse psychoactive drugs. It has been found that rumination exacerbates and prolongs depression, whereas, distraction relieves it, though it may be momentary and may lead to other problems. Women therefore, are likely to have longer and more serious depression (Just & Alloy, 1997). It is part of the masculine stereotype to be active and cope rather than reflecting on one's feelings. Men learn to be less emotionally tuned in than women. By the same token, this sex-linked learning may teach women that they are by nature more emotional, therefore depressive episodes are natural and unavoidable.

Jack (1999) observed that most women suffer depression more than men due to the fact the women are generally socialized to base their self-worth and self esteem on their relationships. They are more readily prepared to silence their own need and desires in relationships in favour of maintaining positive emotional energy vested in their relationships. Many of them regret ever getting involved into such relationship; however, the remorseful feeling may induce depression in the individual.

In a study by Ezeilo (1981) on the relationship of Psychiatric diagnosis to sex and marital status in a Nigerian sample, she considered seven different mental illnesses among 969 patients of Enugu Psychiatric Hospital. These mental illnesses included neurotic anxiety, neurotic depression, psychotic depression, schizophrenia, other affective psychosis, organic psychosis and drug addiction.

The result of the findings showed that single males predominated significantly in all diagnostic categories except in organic psychosis where the difference was non-significant. Gender assessment shows that among the singles, the males predominated in all diagnostic categories at statistically significant levels, but among married ones, there tended to be more females though the differences were non-significant except in the schizophrenic group.

2.2.3 Marital Status and Depression

Depression in a spouse is an issue that most couples will face at some point in their marriage (Nolen-Hocksema, 2002). Depression is a normal and natural response to loss or grief, whether a death, separation from a loved one, job loss, loss of physical health, or relocation Jack (1999). Marital distress and relationship conflict also contribute to depression. Symptoms of depression include feelings of sadness, hopelessness, helplessness, anxiety, irritability, agitation, fatigue, low energy, and a reduced activity level are common, and there is also withdrawal from social contact and loss of interest in previously enjoyed activities, including sex. There may be changes in appetite, weight or sleep patterns, memory problems or difficulty concentrating. Often there are feelings of worthlessness or inadequacy and a lowered sense of self-esteem. In more serious cases there may be suicidal thoughts or a feeling that "life is not worth living" (Comer, 1996). Married women have higher rates of depression than unmarried women, but the reverse is true for men. Marriage seems to

confer a greater protective advantage on men than on women. Marital adjustment and depression are strongly related. In a research, collected data on 695 women and 530 men and then re-interviewed them up to 1 year later. During this a number of participants separated from or divorced their spouses though the majority reported stable marriages. Approximately 21% of the women who reported marital split during the study experienced severe depression, a rate three times higher than that from women (Comer, 1996).

Working women may be prone to depression because they bear the double burden of housework and a job outside the home. Because they have to work in two environments, one is the office environment and the other is home environment. Both are vastly different from one to another. Stress arising from marital relationships is manifested in chronic disorders such as depression, insomnia and hypertension. Since a relationship depends on the nature of the persons involved, it helps to seek the middle path when the inherent individual differences surface. It often helps to change one's attitude, go for counseling or talk openly with your spouse about problems facing your relationship. Pakistan being an Islamic country with traditional society, women are expected to stay home and do house work. Their doing job comes in conflict with the values that may cause marital maladjustment. This research would be so helpful in knowing the difficulties faced by Nigerian working and non-working women just to spend a simple married life. Because our society is man-oriented society, women have to face all problems. If they are working they are supposed to perform all duties at office as well as at home. This study will give valuable information that what are the many causes behind women's depression and stress during marital adjustment. One could argue that out of all the interpersonal cases that can contribute on the onset of a depressive disorder, the ambiance of a family has the most weight and impact on a depressed individual. In the case of spouses, the well

being of one spouse will have a notable impact on the other spouse and on the welfare of their marriage. For example, in 30% of all marriage problems, there is one spouse that can be described as clinically depressed. The reason why a spouse might have a unipolar mood disorder could be due to their relationship being "characterized by friction, hostility, and a lack of affection" (Gotlib & Hammen, 1992).

Martial distress can also be caused by the impact of having a child. When a woman is pregnant, she can experience a whole range of emotions due to the changing of interpersonal relationship with husband and the building of a new relationship with the unborn child. For example, the building of a new interpersonal relationship with the child can be very tasking and become a major stressful life event that can cause a mood disorder to develop (O'Hara, Lewis, Schlechte, & Varner, 1991).

Aside from the martial distresses of spouses, the impact of depressed parents can have an effect on their children as well. In a study on the relation between depressed adolescences and depressed mothers (Hammen & Brennan, 2001), they found that the depressed children of depressed mothers had more negative interpersonal behavior as compared with depressed children of non-depressed mothers. This is reinforced when a study (Chen & Rubin, 1995) shows that the parents of depressed children are less warm and caring and more hostile than parents of non-depressed children. Because of this negative interpersonal relation between kids and their parents, children can develop a negative view of their family. This negative view can lead to the feeling of lack of control and having a high risk of conflict, rejection, and low self-esteem (Comer, 1996).

Cummings (1995) stated that any changes in a family environment due to parental depression increase the risk of developing a mood disorder in children. The

result of this can be found as early as preschoolers and infants, due to the insecure attachment they develop with their parents. The emotional distress of children can also have an effect on their parents, causing depression that in turn will also affect the children, theoretically creating a never-ending cycle unless they seek treatment. Sometimes It is not the depressed parents that lead to the onset of depression in their children, but rather it is the change in the family environment that stems from the parents' depression that causes the children to become depressed. Some studies suggest that marital troubles are a better predicator for the onset of depression than the depression of the parents or the children themselves (Cummings, 1995).

Experiencing depression while as a child or an adolescent can also lead to reoccurring slips as an adult. Depressed persons often perform poorly in marriage and relationship with family members and they also might respond negatively to others, which have the ability to create stressful life events, which as a result might drive the person further into depression (Comer, 1996). Depressed people are dependant on other people and constantly seek reassurance in such a way that drives people away. Hammen and Brennan (2001) found that 13% of the sons and 23.6 % of the daughters who were depressed had depressed mothers as compared to 3.9% of the sons and 15.9% of the daughters who were depressed lacked a depressed mother.

Many people believe that children and parents suffer differently from depression, but not so. Depressed children can be like depressed parents, expressing sadness, anger, shame, and self- directed hostility (Brown & Siegel, 1988). Just like adults, depressed children tend to blame themselves for bad events and accredit the environment for good events--they do not give themselves credit when due (Blumberg & Izard, 1985). This is why oftentimes, children will feel guilty if their parents get divorced and they believe that they were at fault but realistically, it was

the parents' martial distress that was the cause of the divorce, not the children's depressive mood disorder.

Marital status has been found to interact with gender in accounting for variance in the prevalence of depression. In Australia, those who were separated or divorced had a high rate of anxiety disorders (18%) and affective disorders (12%). In Canada, single mothers have been found to have prevalence of 15.4% compared to 6.8% for married mothers, although this increases in rate of depression may relate to the demands of parenting rather than on marital status, per se.

2.2.4 Socioeconomic Status and Depression

Olanrewaju, Akintunde, Femi, Ibrahim and Olugbenga (2007) studied 250 patients in Ilesha and found that over half (59.6%) had various degrees of depression. Similarly, they found that patients who lived below poverty level were more likely to be depressed than those who did not.

The relationship of socioeconomic status to mental disorder differs depending on the definition and measurement of disorder. The inverse relationship is strongest for schizophrenia and anti-social personality disorder. For more general constructs of mental distress and demoralization, the evidence also is consistently in favor of a strong inverse relationship between socioeconomic status and psychological disorder, or to a score on a scale of distress or demoralization (Ortega and Corzine 1990). Many scales measuring "depression" are difficult to distinguish, theoretically and empirically, from demoralization (Link and Dohrenwend 1980). Prior to 1980, the diagnoses for disorders of mood were divided into those of psychotic intensity, such as manic depressive illness (now bipolar disorder) and affective psychosis (now major depressive disorder with psychotic features), and those without psychosis, such

as neurosis. The evidence was relatively clear, as early as the classic study of Faris and Dunham (1939), and continuing to the present, that manic depressive illness and affective psychosis were not associated with socioeconomic status and that schizophrenia was associated with socioeconomic status. For neurosis, which included the depressive subtype, there were studies that suggested an inverse relationship and studies that failed to observe it (Dohrenwend and Dohrenwend 1969). Except where explicitly noted, below we focus on depressive syndrome, which is a constellation of symptoms across a range of symptom types occurring together during a specified period of time. Shifting from sadness and demoralization to depressive syndrome is not trivial, as the depressive syndrome is more persistent and includes vegetative and somatic aspects that can lead to physical disorders (Pratt et al. 1996; Eaton et al, 1996), psychiatric treatment (Thase and Kupfer 1996), and disability (Judd et al, 1996). Most studies of socioeconomic status and mental disorder present data on prevalence rates-that is, the proportion in the population with the disorder—which leaves unresolved the question of temporal order and therefore the direction of causation. This has been dubbed the "causation-selection" issue, or the "stress-selection" issue (Dohrenwend et al. 1992). There are at least four possible explanations for the cross-sectional association of lower socioeconomic status with high prevalence of mental disorder: (1) lower socioeconomic status raises risk for mental disorder, through some sort of etiologic process ("the causation interpretation"); (2) lower socioeconomic status prolongs the duration of episodes of mental disorders through an etiologic process possibly unrelated to causation ("chronicity interpretation"); (3) the mental disorder leads to downward social mobility ("drift interpretation"); or (4) the mental disorder hinders the attainment of socioeconomic status that might otherwise be expected ("selection interpretation"). All four processes can work simultaneously to produce the prevalence result. All four

processes are important for understanding the effects of social structure and for alleviating human suffering connected to both low socioeconomic status and to mental disorders. Measurement of incidence—the rate at which new cases form—eliminates the chronicity, selection, and drift interpretation, allowing focus on etiology.

It would seem logical that mood disorders, and depressive disorder in particular, would have a strong inverse relationship to socioeconomic status. Relative position in the socioeconomic structure of society should be reflected in self-esteem, a component or close correlate of dysphoria, the cardinal symptom of depressive disorder. To the extent that persons in low socioeconomic status positions blame themselves for their status, the potential for guilt, a separate component of depressive disorder, would appear to exist (Matza 1967). Life in the lower strata of socioeconomic status is more stressful, and stress is connected to depressive disorder (Turner, Wheaton, and Lloyd 1995). (However, the type of stress with the strongest connection to onset of depression, that is, unexpected loss of spouse or other intimate other, is not as strongly connected to Socioeconomic status as other types of stress.) Studies of status hierarchies in primates reveal that status and changes in status are reflected in hormonal differences which are known to be related to depressive disorder in humans (Jones, Stoddart, and Mallick 1995). Some Darwinian psychiatrists believe that depression is functional, in an evolutionary sense, because it expresses subordination, which is useful to the survival of the species (Gilbert 1992).

Evidence on the relationship of depressive disorder to socioeconomic status is murky. In a recent review of 47 prevalence studies of Socioeconomic status and depressive disorder from the so-called “third generation” of psychiatric epidemiology (Kohn 1998), 28 of the studies had a result that was not statistically significant, and

19 showed a significant tendency for depressive disorder to have higher prevalence in the lower Socioeconomic status group. The authors concluded that "the relationship between Socioeconomic status and depression is more complex than originally suggested from the earlier studies of non-specific distress". Two examples illustrate the point. The case-control studies of Brown and his colleagues (Brown and Harris 1978) showed that lower class women in London were at higher risk for clinical depression than those of the middle class, but this difference was concentrated among women in the two class strata who had young children in the home. Curiously, a later study in the same area of London failed to replicate the social class aspect of Brown et al's work (Bebbington,1981). In the Epidemiologic Catchment Area Program, prevalence data showed weak and non-significant relationships of occupation, income, and education, to major depressive disorder in the year prior to the interview (Weissman et al. 1991). However, in that study, a relatively strong relationship of depressive disorder with financial dependence on state aid (receipt of welfare, disability, or unemployment funds) was observed. A separate analysis of Epidemiologic Catchment Area prevalence data showed a weak inverse relationship to an overall measure of socioeconomic status (Holzel, 1986). In a national survey in Holland published after the review, DSM-III-R mood disorders (major depression, dysthymia, bipolar disorder) were higher by about 50 percent among those in the lower income and education levels (Bijl, Ravelli, and van Zessen 1998). In a cross sectional study in Ontario, also not included in the review, occupational prestige was inversely related to depressive disorder (Turner 1995).

Only a few prospective studies are available on this issue. For the early years of the status attainment process, before the age of risk for depressive disorder is complete, there are four relevant studies. A prospective study in New York (Johnson,

1999) revealed weak and marginally significant relationships of parental socioeconomic status to depressive disorder. The relationships were stronger for dichotomized measures of socioeconomic status such as poverty, lack of high school education, and unskilled versus skilled occupation, reminiscent of the prevalence studies cited above. This study looked for, but found no relationship of depressive disorder to later attainment. A study in New York of a sample of respondents who had relatives who were depressed (Ritsher. 2001) found a strong relationship of socioeconomic status to early onset depressive disorder, but this relatively strong relationship might have occurred due to the high risk design, which entails a heavy weighting of depressive disorder in the parental pro bands—that is, it might not generalize to unselected populations. This study found no relationship of depressive disorder to later attainment, in spite of such sample selection. A prospective study in Dunedin, New Zealand (Miech 1999), and another in Boston, Massachusetts (Reinherz 2000), showed no effects of parental socioeconomic status on onset of depressive disorder in adolescents. The Dunedin study also demonstrated no relationship between depression and later attainment. The authors of the latter study commented that "the socioeconomic status/depression association found in some studies of adults may be specific to adulthood, reflecting the consequences of adult-specific processes" (Miech 1999).

Prospective studies of the adult years are also ambiguous. Incidence data from the Epidemiologic Catchment Area showed no relationship to baseline levels of occupation and education in an analysis by Anthony and Petronis (1991), but this analysis showed that baseline unemployment predicted onset of depressive disorder during the one-year followup. Another independent analysis showed a relationship between a measure of poverty and risk for onset of depressive disorder (Bruce,

Takeuchi, and Leaf 1991). These two findings suggest a non-linear aspect to the relationship of socioeconomic status and depression. Prospective data from the Woodlawn study also suggest the importance of welfare status in predicting psychological distress (Ensminger 1995). In a rural area of Nova Scotia, the rate of incidence of depression was about four times as high in the low socioeconomic status groups as in the high socioeconomic status groups; however, the sample had only 17 cases with depression (Murphy 1991).

Prior research has also linked low socioeconomic status with high rates of depression, but whether the association reflects an increased incidence or a longer persistence of the disorder has remained unclear.

Current findings were based on data from an epidemiologic study involving the French GAZEL cohort, which was set up in 1989 in cooperation with several departments of Électricité de France-Gaz de France to involve 20,624 volunteers aged 35 to 50 years.

Data was collected routinely over diverse dimensions and obtained from different sources, including an annual self-administered questionnaire to collect data regarding health, lifestyle, familial, social, and occupational factors. Depression levels and socioeconomic position were assessed using the Center for Epidemiologic Studies Depression scale and by occupational grade, respectively.

Using multinomial regression, Maria Melchior, ScD, Centre for Research and Population Health, Epidemiology of Occupational and Social Determinants of Health, Hôpital Paul Brousse, Villejuif, France, and colleagues 2002 ,examined the association between low socioeconomic position and 13-year depression persistence

among 12,650 community-based individuals with depression, using Generalized Estimating Equation models.

Overall, participants in intermediate and low occupational grades were up to 4.5 times more likely than those in high grades to experience persistent depression (age-adjusted odds ratio [OR], 1.40; 95% confidence interval [CI], 1.16 - 1.70; *P*< .0001), an effect that applied to both men (age-adjusted OR, 2.48; 95% CI, 1.36 - 4.54) and women (age-adjusted OR, 4.53; 95% CI, 2.38 - 8.63).

This occupational gradient decreased 21% to 59% but remained significant after adjustments for year of birth, marital status, negative life events (including divorce/marital breakup, spousal death, job loss, and self or spouse hospitalization), health behaviors such as smoking and alcohol use, body mass index, and preexisting psychological problems such as depression or sleep issues, as well as other health issues such as chronic physical illness.

According to the authors, the association between socioeconomic position and depression may reflect 2 mechanisms: health selection, whereby psychological symptoms cause people to drift down the social hierarchy, and social causation, whereby disadvantaged living conditions directly affect mental health. In addition, differential use of mental healthcare services may contribute to worse depression in lower socioeconomic groups.Study limitations include recruiting individuals with a stable job, exclusion of participants belonging to a low occupational grade and likely to be depressed, and assessment of depression using the Center for Epidemiological Studies-Depression scale, which bears the risk for false positives.Strengths included the selection of participants independent of mental health treatment status, the presence of a 13-year follow-up, and multivariate analysis that included several factors associated with depression.The study suggested that the risk of depression

follows a socioeconomic gradient, particularly when the disorder is chronic," the authors note. The main implication is that efforts aimed to reduce the burden of depression and decrease socioeconomic inequalities should address the mental health needs of the population."

Low socio-economic status, particularly when assessed by indices of material standard of living, is consistently associated with a higher prevalence of depression in cross-sectional studies (Lorant , 2003). However, such studies cannot distinguish between associations due to selection (reverse causality) or confounding (Goldberg, 2001) and those that are truly causal. A decade of research has suggested that causation has the edge over selection (Dohrenwend *et al*, 1992; Ritsher, 2001). A recent meta-analysis found that the effect of low socio-economic status on depression is greater for episode maintenance than for onset (Lorant, 2008). Since most longitudinal studies have been of short duration and have characterised socio-economic status using relatively time-invariant variables such as education or occupational social class (Lynch, 1997; Weich & Lewis, 1998), it is not yet known how changes in socio-economic status affect changes in the risk of depression over time. If socio-economic status influences depression through time-invariant mechanisms (such as personality traits), then short-term improvement of socio-economic status would not have any influence on socio-economic inequalities in depression. We thus set out to assess how changes in socio-economic status affect changes in the symptoms and caseness of depression in the general population over a 7-year period. Traditional wisdom has long held that there is an association between depression and socioeconomic status (SES). Several recent studies confirm a strong inverse relationship between SES and mental disorder(Lorant, 2008). Published research indicates that despite differences in definitions and measurements of SES,

the likelihood of depression in the lowest SES group is as much as twice that found in the highest SES group(Lorant, 2008). People in the lowest class are far more likely to suffer from psychiatric distress than those in the highest class. Lennon et al 2007. concluded that one out of every five women on welfare met standard criteria for major depression. Epidemiological studies of depression in Canada and United States found differences in the prevalence rates of depression based on SES factors. However, a review by Kohn et al 2009, found that patterns of relationships were not always consistent. Therefore, it is prudent to periodically reassess the relationship between depression and sociodemographic factors because of the changing demographic composition of Ontario.

2.2.5 Educational Level and Depression

Respondents whose education level was less than secondary school have the lowest rate of lifetime depression (9.1%); and the highest rate of lifetime depression (13.4%) is seen among those with "other post-secondary" education. A similar pattern is seen for 12-month depression. Although these results indicate that the prevalence of depression differs based on the level of education, there is no linear pattern for this relationship. For both lifetime and 12-month depression the prevalence rate was higher for "other post-secondary education" than "post-secondary education".

In another study by Bjelland et al on education and depression; they tried to establish the relationship of education to the experience of anxiety and depression and the direction of this relationship. The knowledge of this relationship is limited and inconclusive. The aim of their study was to examine (1) whether higher educational level protects against anxiety and/or depression, (2) whether this protection accumulates or attenuates with age or time, and (3) whether such a relationship

appears to be mediated by other variables. In a sample from the Nord-Trøndelag Health Study 1995--1997 (HUNT 2) (N=50,918) of adults, the cross-sectional associations between educational level and symptom levels of anxiety and depression were examined, stratified by age. The long-term effects of educational level on anxiety/depression were studied in a cohort followed up from HUNT 1 (1984--1986) to HUNT 2 (N=33,774). Low educational levels were significantly associated with both anxiety and depression. The coefficients decreased with increasing age, except for the age group 65-74 years. In the longitudinal analysis, however, the protective effect of education accumulated somewhat with time. The discrepancy between these two analyses may be due to a cohort effect in the cross-sectional analysis. Among the mediators, somatic health exerted the strongest influence, followed by health behaviors and socio-demographic factors. Higher educational level seems to have a protective effect against anxiety and depression, which accumulates throughout life.

A comparison of nondemented Parkinson's disease patients with lower, intermediate, and higher educational levels indicated an independent association between longer (better) education and less severe depressive difficulties based on the Beck Depression Inventory cognitive-affective items. Well-educated patients also had a better health-related quality of life based on the Parkinson's Disease Questionnaire-39, apparently due to beneficial effects of education on cognitive performance (attention/memory, visuospatial and executive functions) and the degree of depression. More years of education favour milder depressive difficulties and a higher self-perceived life satisfaction in nondemented Parkinson's disease patients.

2.2.6 Psychotherapy and Depression

The breadth of support for CBT is impressive, additional multicenter studies that compare medications and different forms of psychotherapy are still needed to confirm the efficacy of short-term CBT as a treatment for depression, especially in light of the results of the recent National Institute of Mental Health Treatment of Depression Collaborative Research Program study 2001 (NIMH TDCRP) which concluded that cognitive behavior therapy was not effective in the treatment of severe depression. Although many investigators now suggest that the TDCRP study was flawed in several important respects, additional studies still need to be conducted to see if these findings can be replicated. However, for any study findings to be valid, it is essential that active treatments are administered by clinicians adequately trained in the various approaches and those issues of researcher allegiance and site differences are appropriately addressed.

Further, adding controls such as a pill placebo and a placebo plus CBT control condition are imperative to enhance treatment interpretability. In order to enhance treatment effects, studies must also examine the relative efficacy of CBT in depressive subtypes that may have characteristics that are associated with poorer outcome. For example, it has been demonstrated that patients with atypical depression (AD), a new subtype of the mood disorders in the DSM-IV are less responsive to tricyclic antidepressants.

Hence, cognitive behavioral treatment for depressed patients with atypical depression may need to be modified to meet their unique symptom needs. A few of these modifications include individual and group psychotherapy.

Individual and group psychotherapy are both considered highly therapeutic forms of psychotherapy to treat psychological problems. But there are very different

ways that these modalities are therapeutically beneficial to our clients. What are some of the essential differences between individual and group psychotherapy?

Considerable evidence links the concept of therapeutic alliance with psychotherapy outcome for a "wide range of diagnoses among populations. (Klein et al (2013) Individual psychotherapy is a place for a client to work through psychological issues as these issues emerge within the context of a one on one therapeutic alliance with a psychotherapist. However, "group therapy alliance can be understood as the perception of the emotional bond held by an individual group member for the group and an agreement on the goals and tasks for the group therapy among group members with therapist" (Tasca, Balfour, Ritchie, & Bissada, 2007).

Different therapeutic factors are at work in group psychotherapy than in individual psychotherapy. Holmes & Kivlighan (2000), compared individual and group therapy and concluded that there are different factors that reflect different processes in individual and group treatment. They found that the factors of relationship-climate and other versus self-focus were emphasized more in group psychotherapy, whereas the factors of emotional awareness-insight and problem definition-change were more important to the process of individual treatment. Yalom (1995), in his review of the literature, identified 12 therapeutic factors of group therapy. Of the 12 factors identified, interpersonal input (feedback from others) was found to be the most valuable to group members. Yalom (1995) concluded that group interaction and group cohesiveness are the power of therapy groups. This is a substantially different therapeutic process involving different therapeutic factors than individual psychotherapy.

According to Bowlby (1988), in psychotherapy, the therapist "provides conditions in which his patient can explore his representational models of himself and his attachment figures with a view to reappraising and restructuring them". In

individual psychotherapy the attachment process takes place with the psychotherapist and the client's therapeutic process unfolds within the safety and protection of this one on one relationship. In Group psychotherapy, however, the attachment process takes place with the group and clients work through attachment issues as these issues are triggered by others in the group. McClosky (2002) comments that "at every level of group life, misattunement to affect will trigger insecure attachment patterns". Tasca, Balfour, Ritchie, and Bissada (2007) in their study comparing group therapy vs. individual therapy with a group of patients with eating disorders, discussed the "activation of attachment strategies" in group therapy and found that the replaying of "core relational patterns in group therapy interactions" provided the opportunity for group members to display dysfunctional patterns and then to experiment with "new ways of being with others and the self". They concluded that group psychotherapy provides more material to work with than individual psychotherapy.

Individual psychotherapy requires an intense focus on the individual client and the therapeutic work proceeds in terms of insight gained from the understanding of patterns that have emerged from unmet developmental needs or in more of a problem-solving psychotherapy, then individual problem solving strategies are developed within the context of the client-psychotherapist relationship. The group client is not center stage but must share that stage but with 6-10 other psychotherapy clients. In order to be "cured" the client must work within the community of people that comprises a group and it is the group itself, not the psychotherapist or psychotherapist-client relationship that is the primary healing agent (Yalom, 1995).

The group psychotherapy client participates in a plethora of relationships: with the psychotherapist as well as with other group members. Each member also experiences through observation and involvement in the group, the therapy of other group members. "In group psychotherapy in comparison with individual

psychotherapy, there are more people to learn from identify with, disclose to, and with whom to form significant therapeutic relationships" concluded Holmes & Kivlighan (2000).

The complexity of participating in group psychotherapy mirrors the complexity of living in the world of relationships. The therapeutic work in group psychotherapy frequently takes place along many levels at the same time. The group psychotherapy client must learn to be both client and psychotherapist and also to negotiate with others in getting his/her needs met. For example, one depressed and anxious client in group was actively learning how to express his emotional needs in the group, working through trauma issues that were triggered by the memories of another group member, participating in a discussion of the group process, and assisting his fellow group members by providing support, observations, and insight. The development and cultivation of healthy relationships requires that we are fully "present" in many roles, often at the same time.

In both individual and group psychotherapy, clients learn about the "work" and "joy" of being in relationships that have honesty and emotional depth. Group psychotherapy requires by its very nature of relationships that clients learn about a sense of community and to participate fully in all of the many complex roles and relationships that make up the fabric of life. Psychotherapy has also been compared to chemotherapy in terms of effectiveness.

2.2.8 Chemotherapy (Medications) Versus Cognitive Behaviour Therapy

Dozens of trials compare Cognitive Behaviour Therapy (CBT) to a waiting-list control group, and hundreds of trials examine how antidepressants compare to placebo. Yet not many trials directly compare CBT to antidepressant medication. Meta-analytic reviews generally find that both antidepressants and psychotherapy

offer similar efficacy in the short-term, but that after treatment discontinuation, results are better with CBT.

However, nearly all prior meta-analyses suffer from a major problem: the included trials mostly used older (vs newer) drugs. This choice could give an "unfair" advantage to psychotherapy, because the heavy side effect burden of older medications might cause early dropouts, which are less common in newer antidepressants. Olfson et al (2009) indicated that given that 90% of patients on antidepressants are taking newer drugs now, it seems dicey to assume that findings comparing psychotherapy with older medications would yield similar results to trials comparing therapy with newer medications. Cuijpers et al, (2008) showed that until recently, only one meta-analysis specifically examined how psychotherapy, mostly CBT, fared versus selective serotonin-reuptake inhibitors (SSRIs) in particular, and it found a very small but statistically significant advantage for selective serotonin-reuptake inhibitors (SSRIs)

Spielmans et al (2011) stated that while these medications had a slight advantage over therapy at the end of treatment, when therapies provided by primary care physicians, nurses, or therapists with unclear training were removed from consideration, psychotherapy and newer antidepressants had equivalent outcomes in the short term across 14 comparisons. At longer-term follow-up, psychotherapy by properly trained therapists outperformed newer antidepressants by a small, but statistically significant, margin.

Hollon et al (2005) studied 240 patients with severe depression who were randomly assigned to either CBT or a newer antidepressant (augmented with other medication, if needed). After 16 weeks of treatment, the response rates were nearly identical: 58.3% on CBT vs 57.5% for the antidepressant. So far, no big surprise—CBT and medications are usually equivalent over the short term in depression.

Patients who responded to treatment continued in the study, and were randomized to three possible conditions: Those who responded to the antidepressant were randomly assigned to continue on it, or were switched to placebo, while those who responded to CBT stopped therapy, aside from three "booster sessions," over the next 12 months. The majority of patients (76%) withdrawn from the antidepressant relapsed to depression over the year, while only 31% of those withdrawn from CBT relapsed. Patients who had prior CBT even had lower relapse rates than a group of patients randomized to continuous antidepressant treatment (relapse rate of 47%), though the difference was not statistically significant. At least for these patients, it appeared that a brief course of CBT taught them skills to fend off future misery, while medication did not.

There is preliminary evidence that patients with personality disorders may be less responsive to short-term CBT, and that optimal treatment can only be accomplished for these patients if the treatment is modified to address the personality disorder as well (25 for a review). Identifying specific populations who do not respond as well to short-term CBT will lead to the elucidation of factors that must be modified to provide more appropriate treatment. Finally, future research studies in Nigeria need to evaluate the effectiveness of CBT for depression outside of clinical research centers.

The demonstration of treatment efficacy in controlled research environments is only the first step in treatment research. Once a positive therapeutic effect has been conclusively demonstrated, generalizability becomes of paramount importance. With regard to CBT for depression, it seems fair to conclude from the reviews conducted that CBT is an effective treatment in clinical research settings. But data are not available on the efficacy of CBT for depression when delivered in non-research clinical settings to a diverse group of patients in Nigeria(This is not unique to CBT,

and applies to other empirically supported treatments as well, e.g., pharmacological approaches). Without data, one must be cautious in generalizing the results from research settings to typical clinical settings because there are several factors that might reduce the efficacy of this treatment. For example, one area of particular concern is that clinicians in research settings are likely to possess greater expertise in the administration of a particular treatment developed in that setting compared to the average clinician.

Thus, since clinician competence may be an important factor for success, one could expect a less favorable outcome in uncontrolled settings where the quality of treatment may not be as good. While caution may be warranted until data are generated specifically on CBT for depression, it is reassuring to note that data are beginning to appear that support the effectiveness of evidence based treatments outside of controlled research environments, and a recent meta-analysis of psychotherapy studies found that the effect sizes of psychotherapy in "clinically representative settings" is slightly lower (approximately 10%) but comparable to that obtained in clinical research settings.

Cognitive-behavioural therapy in depression is an active, structured approach that typically consists of a variety of cognitive restructuring, problem-solving and activity-planning interventions (Fennel, 1989). Cognitive-behavioural therapy has proven quite effective in treating depression (Nolen-Hoeksema, 2004). About 60 to 70 percent of depressed people experience full relief from their symptoms with 10 to 12 sessions of cognitive-behavioural therapy (Hollon, Hamman, & Brown, 2002; Lewinsohn, Clarke, Hops & Andrews, 1990). Cognitive-behavioral therapy has been successfully adapted for the treatment of depressed children and older persons (Garber & Horowitz, 2002).

The nature of cognitive-behavioral therapy is revealed by its hyphenated name. Client's cognitions are modified in two ways: cognitively and behaviorally. Cognitions are modified directly by teaching clients to change their maladaptive thoughts and indirectly to helping clients change their overt actions (Spiegler & Guevremont, 1993). Changing what people think in order to change what they do is the time-honoured strategy for attitude. CBT has been found to be very effective by several researchers; these include Robinson, Berman and Neimeyer (1990).

In their meta-analysis of 58 studies of psychotherapy for the treatment of depression published between 1976 and 1986 in which they compared CBT to no treatment control – a placebo control, they identified that CBT was significantly more effective than the other psychotherapies. Spence, Sheffield and Donovan (2005) found a CBT intervention to have an immediate effect in terms of reducing depressive symptoms, but these benefits were not maintained over 4 years of follow-ups. Clarke, Hawkins & Murphy, (1995) compared a control group and a CBT group and found that CBT halved the incidence of depression over a 12-month follow-up period for a group of adolescents who had elevated levels of depressive symptom rates of episode; remission tend to fall in the range of 50% to 65%, compared with about 60% in control groups. In a review and meta-analysis by Harrington, Whittaker, and Shoebridge (1998), they concluded that CBT for children is efficacious, producing significant benefit on a range of outcomes.

A recent case series investigated 12 weekly 60-minute sessions of RFCBT for 14 consecutively recruited patients meeting criteria for medication- refractory residual depression (Watkins et al., 2006). Treatment produced significant improvements in depressive symptoms and co-morbid disorders: mean reduction in

Beck Depression Inventory of 20 points, pre-to-post treatment within-subject effect size (Cohen's d) of

2.5, 50% of patients achieving full remission from depression, and a 71% reduction in co-morbid Axis I diagnosis. Importantly, RFCBT significantly reduced self-reported rumination, with rumination at pre-treatment equivalent to that found in currently depressed patients but the range of scores at post-treatment was equivalent to levels of rumination observed in never-depressed participants.

David conducted a study in 2001 to determine whether adjunctive group psychotherapy would lead to additional benefits in dysthymic patients who had already shown a response to an SSRI antidepressant medication. They found that at the end of treatment (i.e., at termination), significant differences favoring the combined treatment were found on some symptom measures (Ham-D) and general functioning measures (CGI-S and GAF). Other measures (CDRS, IIP-high score) showed differences at the trend level and demonstrated medium to large effect sizes, with combined treatment subjects showing better functioning in these areas than medication-only treated subjects. Furthermore, 33.3% of patients in combined treatment meet rigorous criteria for improvement in symptomatology, global functioning, and personality variables, whereas only 6.7% of patients treated with medication alone meet these criteria.

Butler, Chapman, & Formen (2006) recently reviewed existing CBT meta-analyses for unipolar depression. They identified 3 meta-analyses and concluded that CBT produces large within-group effect sizes for symptom improvement.

Moreover, CBT is at least comparable to antidepressant medication and other evidence-based psychological therapies, including interpersonal therapy and behavioral activation. Most outcome research relates to individual CBT, but there is evidence to suggest that group CBT is equally effective (Robinson, Berman

&Neimeyer, 1990). Importantly, the beneficial effects of CBT appear to persist up to several years post treatment and are associated with preventing relapse. There is sound evidence that CBT may be superior to continuation of antidepressants in preventing relapse.

However, a recent trial comparing patients initially successfully through either antidepressants or CBT, and then continued on antidepressants or booster CBT, suggested equivalent rates of relapse across the CBT and antidepressant conditions (Hollon, DeRubeis & Shelton, 2005). Subsequent trials have shown CBT to be equivalent to antidepressant medication in the treatment of moderate-to-severe depression (DeRubeis, Hollon, & Amsterdam, 2005) although such differences in this most recent trials suggest that therapist selection, training and supervision are likely to be especially important with clients suffering from severe depression.

CBT provides a sophisticated, empirically grounded account of depression and an evidence-based therapeutic approach for people who suffer from depression. Beyond its efficacy in treating acute depression, it has prophylactic effects and is acceptable to various populations in a range of settings. Our understanding of factors contribution to positive outcomes is growing, allowing CBT to be tailored to individual client needs (Kuyken, Dalgeish, & Holden, 2007). They therefore claimed that CBT is a mainstay approach to depression. Remaining challenges include tailoring it to different populations and settings and most importantly, ensuring that it is more readily accessible.

There are some evidences that changes in inferential style are observed at higher rates in CBT, compared with antidepressant treatment, and that these cognitive changes at least partly mediate symptom change in CBT (DeRubies, Evans & Hollon, 1990). Moreover, a study has shown that some clients who engaged in CBT for

depression show sudden gains in their symptom-response profile and that these gains are associated with cognitive changes in the preceding session (Tony & DeRubeis, 1999). However, at least one study has failed to replicate this finding (Kelly, Roberts & Ciesla, 2005). CBT for acute depression is effective and appears to offer longer term protection against relapse.

A remaining challenge is to develop evidence-based primary prevention programmes based on an emerging theory of the developmental psychopathology of depression as well as established CBT treatment principles. A further challenge is to extend the evidence base to different populations (for example, those with co-morbid diagnoses, and older adults) and to adapt CBT to make it acceptable in different settings (for example, inpatient and residential settings). A significant challenge at the level of policy is the availability of CBT; organizing services and training therapists so that people suffering from depression can access high-quality CBT is a challenge that some groups are beginning to address (Bower, & Gilbody, 2005).

The use of CBT has been extended to children and adolescents with good results. It is often used to treat major depressive disorder, anxiety disorders, and symptoms related to trauma and post-traumatic stress disorder (Kuyken, Kurzer, & DeRubeis, 2001). Significant work has been done in this area by Reinecke, Dattilio and Freeman (2003) and they also validated CBT as effective in a group setting for the treatment of youth and child anxiety. CBT has been used with children and adolescents to treat a variety of conditions with good success (Kendall, 2005). CBT is also used as a treatment modality for children who have experienced complex

2.2.9 Group Cognitive Behaviour Therapy and Depression

Cognitive–Behavioural Therapy (CBT) is a psychological treatment approach that can be delivered not only on a one-to-one basis but also to groups and in self-help formats. However, the evidence base supporting individual CBT is more extensive than the research regarding group CBT. This is likely to influence the choice of services that develop in the Improving Access to Psychological Therapies (IAPT) programme for the treatment of depression and anxiety disorders in primary care in England. This article outlines the different forms that group CBT takes, the way in which it may benefit people and the current evidence base supporting its use for anxiety and depression. It also outlines the advantages of group or individual CBT and describes those patients who appear to be best suited to a specific delivery.

Cognitive–Behavioural Therapy (CBT) delivered in group format has attracted less research than CBT delivered on a one-to-one basis. Nevertheless, no doubt influenced by the challenge of limited resources, many centres do offer group therapy based on CBT principles as an alternative or in addition to more traditional CBT delivered to individuals. In England, the Improving Access to Psychological Therapies (IAPT) programme (Department of Health 2008) aims to greatly increase the number of people treated using evidence-based approaches (as defined by the National Institute for Health and Clinical Excellence, NICE) for depression and anxiety disorders in primary care. The potential economies of scale afforded by the use of group work will be an attractive proposition for National Health Service providers working to implement IAPT.

Group psychotherapy has its origins in psycho-dynamic models of pathology and it developed before the practice of CBT was first outlined by Aaron Beck and colleagues (Beck 1979). Importantly, group psychotherapy views the interactions

between the group members as the vehicle of change. In CBT groups, it has traditionally been assumed that the cognitive–behavioural model taught to the group is more of an 'active ingredient' than the relationships between the group members (Bieling 2006). Indeed, the educational ethos is inherent in the CBT model, and this lends itself very easily to the provision of groups or classes, as does the fact that CBT is structured and directive, collaborative and time-limited (Fennell 1989).

However, group CBT cannot solely be about taught skills, and in common with individual (one-to-one) CBT, non-specific interpersonal factors play a part. As with individual CBT, these non-specific factors have not attracted the same research attention as the underlying cognitive–behavioural theoretical models (such as which underlying thoughts and behaviours are paramount in different disorders) (Bieling 2006; Tucker 2007). This is in sharp contrast to group psychotherapy, which has focused on interpersonal relationships and related mechanisms of change.

To varying degrees, Yalom's therapeutic factors are likely to be relevant to all models of group psychotherapy. Wherever you have the formation of a group you will have patterns of relating – 'group processes' – that will affect both the group as a whole and the extent to which group members benefit from attendance (Burlingame, 2004). Some of Yalom's factors – such as the corrective recapitulation of the primary family group – might be seen as less relevant to CBT groups. However, researchers have translated aspects of the concept into CBT principles – so that the group might be seen to modify maladaptive relational patterns through observing others and trying out new styles of interacting (Bieling 2006).

White & Freeman describe the two important elements that need to be present for an effective CBT group as the group's cohesiveness and task focus. They define cohesiveness as 'the degree of personal interest of the members for each other'

(White 2000). All CBT groups should be task-focused, in that there should be defined goals to be achieved. Other mechanisms that probably play a part in effecting change in group CBT are listed below: as can be seen, many incorporate concepts inherent in Yalom's therapeutic factors.

The group provides ready circumstances for behavioural experiments challenging automatic thoughts and their underlying core beliefs and assumptions. For example, the prediction 'If I say something with a strong accent or use a wrong word then people will look down on me and laugh' can be tested in the group. Beliefs can be gently challenged not only by the therapist but also by the other group members, who are to a degree acting as co-therapists (Heimberg 1993).

When group members observe that other members have similar experiences, worries and emotional responses it illustrates for them that they are not unique in thinking and behaving as they do. This can be a powerful normalising experience which helps to reduce associated stigma and shame. The mechanism by which this occurs is the disproving of beliefs such as 'I'm alone' and 'I'm defective and different from others'. The normalization process can therefore be seen as another method of cognitive restructuring.

a. Therapeutic relationships

Individuals sometimes take comments made by one group member to another more seriously than those made by the therapist. This is probably because the other group members are viewed as more impartial than the therapist. The relationships within the group (therapist to group member and between the group members) must be as collaborative and as non-threatening as possible, so that questioning and suggestions are not perceived as attacking and undermining (White 2000).

b. ‘In-vivo’ exposure

Exposure to a feared stimulus is often helped by having a group of people to hand. Social phobia, for example, is a natural candidate for group CBT. Feared situations such as public speaking can be recreated within the group setting to allow individuals to habituate to their fears. Note how this links in with cognitive restructuring through the testing out of beliefs mentioned earlier. Practitioners of CBT have devised group protocols that deliver cognitive–behavioural principles in innovative and diverse formats. One way of classifying the range of group formats is on a continuum from ‘small groups’ through to ‘large-group psychoeducation’ (Morrison 2001). The characteristics of the ‘therapy’ delivered at these two extremes. In reality, most groups will have elements from both ends of the continuum. The psychoeducational format has been defined as ‘high volume’ and ‘low contact’: large numbers of attendees can be ‘treated’ at one sitting, although the interaction between the attendees is minimal. Sessions are delivered in a more traditional didactic teaching style (Cuijpers 2005).

Examples include the stress control programme developed to treat large numbers of people with anxiety in primary care (White 2004; Kellett 2007a) and all-day workshops for stress and self-confidence (Main 2005; Brown 2008). The large-scale stress workshops of Brown and colleagues contained 20–25 participants. When you consider that these workshops allowed self-referral and were offered during the weekend in a leisure centre, then the potential to offer services with reduced stigmatization can be appreciated. Of particular interest is the fact that over 70% of attendees who referred themselves fulfilled criteria for an ICD–10 diagnosable disorder. Many of these attendees’ mental health problems were not known to their general practitioners (Brown 2005). As the lowered anxiety ratings achieved in the

workshops were maintained at 2-year follow-up (Brown 2008), the potential of such interventions to deliver significant public mental health improvements can be appreciated (Brown 2005).

There are advantages to having more than one therapist in group CBT sessions. A second therapist has the flexibility to leave the room with a distressed patient or to note changes in a patient's mental state that may not be apparent to a therapist working alone. This monitoring can be more easily carried out when the group is smaller. Although it is harder to tailor the content of group therapy sessions to meet the needs of individual members' formulations it is not impossible, particularly in smaller groups. However, group therapy may not be able to follow an individual's formulation completely (as each patient's formulation is different) as would be the case in individualised therapy (Morrison 2001).

Most groups CBT will attempt to incorporate the key characteristics of individually delivered CBT. The therapist aims to promote a group culture that encourages gentle challenging and questioning of preconceptions. In smaller groups this style allows group members to interact in such a way that they serve as *de facto* therapists to each other (Heimberg 1993). Homework tasks are an integral and essential component of CBT, so that the principles addressed during sessions can be generalized outside them. The therapist should review the outcomes of the homework, although the extent to which this can happen for all group members is questionable in very large psycho-educational groups.

It is clear that the experience of attending an 'intimate' small CBT group of five will be very different from that of attending a large psycho-educational didactic group with little interaction between group members. This has implications in terms of the active ingredients of the particular form of CBT delivered. It can be

hypothesised that some of the factors outlined in the previous section will be particularly important in small groups – such as the ability to use other group members as co-therapists. Other factors, such as the normalising effect of having the same problem as so many other people who make up the group, may be more active in larger psycho-educational groups (Kellett 2007). However, this remains to be proven.

In addition to whether there is an evidence base supporting the use of group CBT for a particular condition or problem, other patient factors need to be considered. There are factors that are believed to influence therapeutic success in individual CBT (Moorey 1996).

The presence of any of these variables need not preclude CBT: a patient may have multiple Axis I diagnoses or significant Axis II problems and still gain great benefit from CBT. It is simply that these factors need to be taken into account when a clinical decision is made about whether to progress with therapy.

There is a substantially larger body of evidence supporting the use of individually delivered CBT compared with the evidence supporting CBT in groups. However, some clinicians have erroneously taken the evidence supporting the former as implicitly supporting the latter. This ignores the likelihood that the format of the therapy, in addition to the underlying model, affects its effectiveness. From the summary of the current evidence base relating to depression and anxiety disorders outlined below, it can be seen that we cannot unequivocally conclude that group and individual CBT interventions have equivalent outcomes. Tucker & Oei have analysed the evidence base for group and individual CBT to calculate which is the most cost-effective (Tucker, et al2007). They tentatively deduce that group CBT is more cost-effective for depression, but less cost-effective for anxiety and social phobia. They

point out that the available evidence has significant methodological shortcomings (including whether the efficacy trials can be generalised into naturalistic settings) and conclude that 'It cannot be summarily or definitively stated that group CBT is a cost-effective treatment'.

The recently updated NICE guidelines on depression (National Collaborating Centre for Mental Health (2009) state that there is a place within the stepped care model of treatment for group CBT based on the 'Coping with Depression' approach (Lewinsohn 1989; Kuehner 2005). This approach, which uses the concepts of traditional CBT, has a strong psycho-educational element and consists of twelve 2-hour sessions over 8 weeks (sessions are twice weekly for the first 4 weeks). The guidelines comment that this traditional CBT group approach has a medium effect size for mild depression. However, because they find that group CBT is less cost-effective than low-intensity approaches (such as bibliotherapy and computerised CBT) they recommend that the latter be the first-line treatment in the majority of cases. Interestingly, because of this evidence supporting the use of low-intensity interventions and traditional group CBT for milder depression, the guidelines have now removed the previous recommendation for counselling as a first-line treatment for mild to moderate depression. Specifically, the guidelines recommend that group CBT should be considered: 'for people with persistent subthreshold depressive symptoms or mild to moderate depression who decline low-intensity psychosocial interventions' (p. 250).

In addition to traditional CBT groups, the updated NICE depression guidelines also continue to recommend mindfulness-based cognitive therapy, which is generally provided in group format (Segal 2002). Groups meet for eight weekly 2-hour sessions with four follow-up sessions in the following year. Mindfulness has

developed from Buddhist principles of meditation and has a specific remit in this context of reducing the relapse rate rather than treatment of depression during an episode (Teasdale 2000). Consequently, it is a treatment for recurrent depressive disorder and the guidelines recommend it for people who have had at least three episodes of depression.

The NICE guidelines for depression in adults with a chronic physical health problem (National Collaborating Centre for Mental Health 2009b) appear to recommend group CBT for a greater range of presentations than is the case for the generic depression guidelines (National Collaborating Centre for Mental Health 2009a). Also, in contrast to the OCD guidelines (National Collaborating Centre for Mental Health 2006) they define group CBT as a 'high-intensity' psychological intervention. They recommend group-based CBT as an initial option for moderate depression associated with a chronic physical health problem and for people with milder presentations who have not responded adequately to 'low-intensity' interventions such as peer support groups or computerized CBT.

2.4 HYPOTHESES

1. Age, gender, marital status, employment status and educational level wouldindependently and jointly significantly predict depression.
2. Participants who received combined (individual and group) CBT would significantly report lower scores on depression than those who did not receive any cognitive behaviour therapy.
3. Participants who received only group cognitive behaviour therapy would significantly report lower scores on depression than those who did not receive any cognitive behaviour therapy.
4. Participants who received only individual cognitive behaviour therapy would significantly report lower scores on depression than those who did not receive any cognitive behaviour therapy.
5. Participants who received combined (individual and group) CBT would significantly report lower scores on depression than those who received only individual CBT.
6. Participants who received combined (individual and group) CBT would significantly report lower scores on depression than those who received only group CBT.

2.5 OPERATIONAL DEFINITION OF TERMS

2.5.1 AGE

The length of time during which a being has existed: usually measured by years from birth.

2.5.2 GENDER

This is the biological differences that could be attributed to nature between a male and female**.**

2.5.3 MARITAL STATUS

It is a state of being married, single, separated or widowed.

2.5.4 EMPLOYEMENT STATUS

It involves whether an individual is doing something productive or not.

2.5.5 EDUCATIONAL LEVEL

This relates to the academic attainment achieved by an individual such as Primary, secondary or higher level of learning.

2.5.6 DEPRESSION

Is a being sad, gloomly and0.having pessimistic ideation with loss of interest or pleasure in normally enjoyable activities. This feeling is often accompanied withsymptoms of anorexia and consequent weight loss, insomnia, feeling of worthlessness or guilt, diminished ability to think or concentrate or recurrent thought of death or suicide. In this study, it is the participant's scores on the beck depression scale. The higher the score the higher the level of depression.

2.5.7 COGNITIVE-BEHAVIOURAL THERAPY

This refers to the techniquesthat are used to help people with depression overcome their problems. This include techniques such as cognitive restructuring through monitoring and identification of dysfunctional thoughts and disputing them as well as behavioral techniques such as relaxation training, behavioral recordings by the clients.

2.5.8 EFFICACY

The ability of an intervention to produce the desired beneficial effect in expert hand.

CHAPTER THREE

3.0 METHODS

3.1 (a) Design

This is an intervention study and multi-staged as with clinical research which encompasses assessment preceding the experimental intervention research. The design is with two stages as follows. The first stage (Cross sectional survey using ex-post facto design) was an assessment of depression in patients. The independent variable is cognitive behaviour therapy – group and individual and the dependent variable is depression.

The second phase which was an experimental research designadopted a pre and post-test comparisons between the treatment and control groups. It aimed at finding out the efficacy of group Cognitive Behavioural Therapy among patients with depression. Only individuals who participated in the first phase of the study and met the criteria for being selected for second stage were selected for this stage. This stage employed pre-test and post- test control quasi-experimental research design. This method became necessary in order to find out the effectiveness of group Cognitive Behavioural Therapy on participants with depression. The participants were divided into four groups;

Group 1 - Combined group and individual CBT

Group 2 - Individual CBT

Group 3 - Group CBT

Group 4 - The control group.

All groups were given pre- test but only the first three groups received the cognitive behavioural intervention. The control group only benefitted once from a health talk which was open to all patients visiting the hospital. Post test was administered to all the groups immediately after the termination of the intervention to measure the immediate impact of the intervention.

At the start of each recruitment process, the patients were carefully told the study purpose, right to continue or withdraw, benefits of the study and the likelihoodof falling into any of the groups. In all the consent for those who participated in the studyt was sough for and obtained.

3.2 Setting

The research was carried out at Federal Neuro- Psychiatric Hospital, Yaba, Lagos. The setting was considered appropriate for this study because it is one of the leading treatment Centres in Nigeria with a large turnout of patients. The Centre caters for patients who come from various cities in the western region to receive treatment and care.The hospital is located at no 8 Harvey roads under Yaba Mainland Local Government. The Hospital is a major tertiary institution in Nigeria with renowned experts in the management of psychiatric cases. The hospital has an annex in Oshodi where the child and adolescent center is located and adult patients are also admitted in to the adult ward.The Yaba setting has 520 beds for inpatients and see on each clinic days (Monday, Tuesday, Thursday and Friday) an average of 500 patients. The management team of the hospital comprises of Doctors, Nurses, Pharmacist, Clinical Psychologist, Social workers and Occupational therapist and Administrative staff. This environment was considered appropriate because the participants are familiar with this environment which is very conducive, save and accessible to them.

The venue also guaranteed freedom from any form of interference or breach of privacy. Consulting rooms and halls were available for intervention sessions.

3.2.1 Sample Size

The population for this study was selected from depressive patients seen at the out-patient clinic of Federal Neuro Psychiatric Hospital Yaba, Lagos. Participants were drawn from those who had been receiving treatments with medications for more than six months prior to the study. A structured questionnaire was administered to one hundred and fifty participants who met the inclusion criteria.

From these numbers, only one hundred and sixteen questionnaires were returned and analysed. Others were excluded because of incomplete information. Among the 116 participants, wereof 66 males (56.9%) and 50 females (43.1%), 92 employed (79.3%), 24 unemployed (20.7%), 51 married (44%) and 65 single (56%).

The collected and analyzed questionnaires showed that only forty of the participants met the criteria for selection into the second stage as their scores fell within modreate depression (inclusion criteria include: be diagnosed with depression , be mentally stable , indicate willingness to participate in the study and sign the informed consent form to participate).

The sample size of this study is 40 participants which represented 30% of the sample size in the first phase. The size was determined based on the participants that met the inclusion criteria and the size the researcher has the capacity to finance and adequately manage.

3.3. Sampling Techniques

Participants for the first phase of the study were purposely selected from the outpatient clinic on their various clinic days. The patients were identified through their appointment registers with the assistance of the record personnel.

3.4. Participants

The population for this study was selected from depressive patients seen at the out-patient clinic of Federal Neuro Psychiatric Hospital Yaba, Lagos. Participants were drawn from those who had been receiving treatments with medications for more than six months prior to the study. A structured questionnaire was administered to one hundred and fifty participants who met the inclusion criteria.

The second phase comprised of patients receiving treatment for depression. All the participants were selected from those who participated in the first stage of the study, and met criteria for being selected for second stage. Forty participants comprising of 22 males and 18 females participated in the second stage and were purposively divided into four groups. 10 were in the combined group and individual CBT, 10 were in only Individual CBT group, 10 were in only group CBT group and 10 participants were in the control group.

3.5 Instruments

Section A: socio-demographic variables: Respondents' socio-demographic and background variables were measured under this section. These include age, sex, level of education, marital status, employment status, illness stage, illness duration,

Section B – Depression Scale: Beck Depression Inventory (BDI-II). The BDI-II (Beck, & Steer, 1978) is a self-report measure composed of 21 items that reflect the

cognitive-affective and somatic symptoms of depression. The original version was published in 1961 and subsequently revised. It had been extensively used especially in assessing and monitoring changes with cognitive therapy. The items are rated using a four-point modified likert scale ranging from 0 to 3, with 0 reflecting no experience of depressive symptom and 3 reflecting the experience of a significant degree of depressive symptom in question. Respondents are required to rate the items considering their experience within the last two weeks. The BDI-II score is the total of the rating for each item, which has a range from 0 to 63. <10 represents minimum or no depression; 10-18 indicate mild to moderate depression; 19-29 show moderate; 30- 63 is severe depression. Reliability studies showed a test retest correlation having ranged from 0.48 - 0.90 (beck et al. 1988). Beer and Steer report Crombach alphas of 0.92 in the clinical sample and 0.83 in the healthy sample.

Section C –Center for epidemiological studies- depression scale (CES-D) was developed by Radloff (1977). It is a self-report 20 item measure that covers depressed and positive affect, interpersonal symptoms and somatic activity. According to Radloff (1977) the split-half correlations of .90 was found for the patient group and .85 for the community sample.

3.6 Procedure

For a research of this nature, it is important that all the ethical procedures are followed. The researcher submitted a copy of this proposal to the institution's Ethical and Research Committee of Federal Neuro-Psychiatric Hospital, Yaba, Lagos. A formal approval was given by the committee. Patients who met the inclusion criteria mentioned were given consent forms to sign and participate in the study. The first stage of the study involved administration of questionnaires to each of the participants after the consent paper had been duly signed.Based on the criteria that must be

fulfilled to be considered to participate in this study, purposive sampling method was adopted for the selection of participants for the second stage of the study. All the inclusion criteria for phase one of the studies were maintained in the second stage of the study. The intervention programme began with securing a formal approval from the management of F.N.P.H.Y for the use of their consulting rooms and psychotherapy hall for the intervention programme. The approval for the intervention stage was part of the ethical approval earlier secured for the study. The intervention started with an introduction of the intervention and what will be required from the participants within the period of the intervention was discussed. The participants were made to sign another consent form for this stage. After discussion, participants were randomly assigned to treatment and control groups using simple random sampling of odd and even number technique.

Because the design of this stage is Pre-test Post Quasi experimental control group design, both the treatment and control groups were given pretest to establish baseline. Only the treatment group was however exposed to intervention. They met once a week (Saturdays) and the sessions lasted for eight weeks. Two clinical psychologists with clinical experiences were co-opted to also help handle the individual sessions. The modules of intervention were strictly followed so as to be properly guided. During the sessions there were occasional miss of session by the participants. The control group was not exposed to intervention but met only once and was given health talk. This was done in order to maintain contact and relationship with them. The efficacy of the interventions was first measured on the participants' post test score which was administered to both Intervention and Control groups. They were thereafter followed up eight weeks after the intervention to know the efficacy of the intervention on their adherence level. Provision was made for refreshment and transportation for participants during the intervention programme.

3.7. Inclusion /Exclusion Criteria

Inclusion

a) be diagnosed with depression
b) be mentally stable
c) indicate willingness to participate in the study
d) sign the informed consent form to participate

Exclusion

The exclusion criteria are as follows:

i. Patients who are not willing to participate.
ii. Patients who are not mentally stable.
iii. Patients that did not meet the diagnosis of depression.

3.7.1. Ethical consideration:

i. Theresearcher obtained Ethical approval from the research and ethical committee of F.N.P.HY.
ii. The participants were be briefed on the purpose of the study and that the data that were collected from them was meant for research purposes only.
iii. Confidentiality, anonymity and privacy of participants were given great consideration.
iv. Participants were not exposed to any form of harm or exploitation before, during and after the study.

3.7.2. Control of Extraneous Variables

In order to ensure that all extraneous variables were controlled, the following steps were taken

1) Randomization: Participants were matched as explained above
2) The researcher conducted the experiment personally so as to control for experiment effect.
3) Avoidance of Intrusion: The cooperation of medical personnel on duty was sought so as to avoid intrusion throughout the duration of intervention.

a. Randomization of participants

In order to avoid the influence of various unaware factors that could influence the results of the study, the participants were purposively divided into both the treatment and control group using simple random technique.

b. Matching: Matching was ensured in that all the participants in all the groups were literate enough to understand the ingredients of the intervention.

c. Welfare Package: To ensure that favoritism was avoided and participants were treated equally, all the participants were given equal incentives.

3.8. Statistical Analysis

Responses to the questionnaires were coded and entered into the SPSS (version 16) and SPSS was used in most of the analyses except the descripitive statistics.Hypothesis 1 was tested with the use of multiple regressions.Hypotheses 2 - 6 were with the use of t-test for independent measures.

3.9 Intervention Modules

Session 1: The researcher greeted the participants and introduced himself in a warm and professional manner. He explained the model of the therapy and the process by which the therapy was to be conducted. The reason for coming together as a treatment group or being seen on an individual basis and reason for selection were also explained to them. The nature and psychological implications of depression was explained. An agenda that would guide the sessions were set. Each patient gave a brief review of his/her psychiatric history. Feedback was obtained from the group members to access their impression about the session so far. At the end of the session's interaction, an assignment was given to participants to write about their knowledge, beliefs and experiences of stigmatisation.

Session 2: The session started with a recapitulation of the last session. Recurring themes in history and assignment were identified. These include dysfunctional and negative thoughts and beliefs, unhappy feelings and withdrawal from the society and feelings of worthlessness. The connection between thoughts, feelings and behaviour was explained to the participants since thoughts influence feelings and feelings likewise affect behaviour. Other psychological factors like self-esteem associated with self-stigma and perceived stigma were introduced. Individual/Group members were told that each factor will be discussed in details during subsequent sessions. Feedback was obtained from members to assess their understanding so far.

Session 3: individual/Group members gave a review of the last session. The cognitive model was further explained especially the reciprocal and interactive nature of thoughts, feelings and behaviours. In addition, the researcher further explained the role of negative thoughts and distorted cognitive processes as mediator of behaviour

using example. Next, Daily Record of Dysfunctional Thoughts-DRDT was introduced to the participants and the recording was fully explained. An assignment was given for them to record the DRDT.

Session 4: The session started with a summary of the previous session. The assignments were reviewed and discussed. Individual/Members who completed the assignment were verbally reinforced and those with problems with their assignment were assisted. The need to practicalize session activities in everyday life was emphasized. An assignment based on working on dysfunctional thought, feeling, and behaviour was given.

Session 5: A recapitulation of the previous session was given by the individual /group members. The homework assignments were reviewed and those who successfully completed their assignments were verbally reinforced. The main activity of the session was that each member gave a personal account of how he/she was able to influence his/her behaviour by taking cognizance of personal thoughts and feelings. For homework assignments members were required to further identify automatic negative thoughts and the effect they have on their feelings and behaviour.

Session 6: Participants gave a recap of the previous session. The homework assignments were reviewed. The researcher emphasised on the need to correcting and clarifying the process of challenging thoughts by the participants. The researcher started preparing the participant/group members for termination of sessions. Homework assignment was given.

Session 7: Participants gave a recap of the previous session. The homework assignments were reviewed. The researcher emphasised on the need to correcting and clarifying the process of challenging thoughts by the participants. Homework assignment was given. Participants were informed that there were two sessions to end the therapy. They were also reminded that the next session would be ending the therapy session.

Session 8: The homework assignments were reviewed - A recapitulation of the sessions. Emphasis was laid on how to deal with dysfunctional thoughts and how to handle psychological concomitants in the future. They were also told that they could apply the same principles to so many aspects of their lives. The need to also comply with their medication and out-patient clinic was emphasis Individual patients were made to demonstrate practically how he/she applied the principles taught during the therapy in real life situations. They were also reminded that the next session would be ending the therapy session.

3.10 The Post-Treatment Assessment: In order to assess the level of progress made by participants after intervention, the instruments were re-administered to the participants for post intervention assessment. The control group was also re-assessed along with the experimental group. Posttestwas carried out immediately after the eight sessions of the intervention. Participants in the experimental and control groups were reassessed.

CHAPTER FOUR

RESULTS

4.1 Socio-demographic characteristics of the respondents

Table 4. 6: Socio-demographic characteristics of Respondents

	Frequency (n)	Percentage (%)
Gender		
Male	22	55.0
Female	18	45.0
Age (Years)		
Below 25	3	7.5
25 – 35	18	45.0
35 – 45	13	32.5
45 and Above	6	15.0
Employment Status		
Employed	37	92.5
Unemployed	3	8.6
Marital Status		
Single	19	47.5
Married	21	52.5
Education		
Primary	3	7.5
Secondary	21	52.5
OND	2	5.0
HND/Bsc	14	35.0

There was a preponderance of male respondents (55.0%) with (45%) been female and majority of the respondents were aged between 25 and 35 years (45%). 7.5% were age below 25 years,32.5% aged between 35 and 45 years and 15% were 45 years and above. Majority of respondents were employed 37 (92.5%) while 7.5% were unemployed. Married respondent constitutes about 52.5% of the respondents and 47.5% were single.

Most of the respondents (21) had secondary school education (52.5%) while 40.0% had tertiary education, and only 7.15% had primary education.

Figure 5: Pie Chart showing the gender distribution

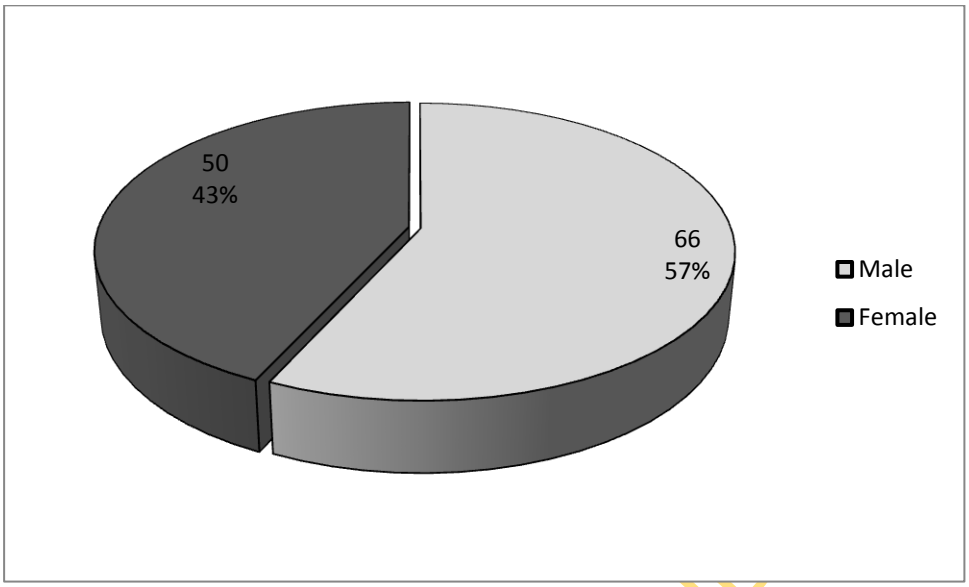


Figure 6: Bar Chart showing the age distribution

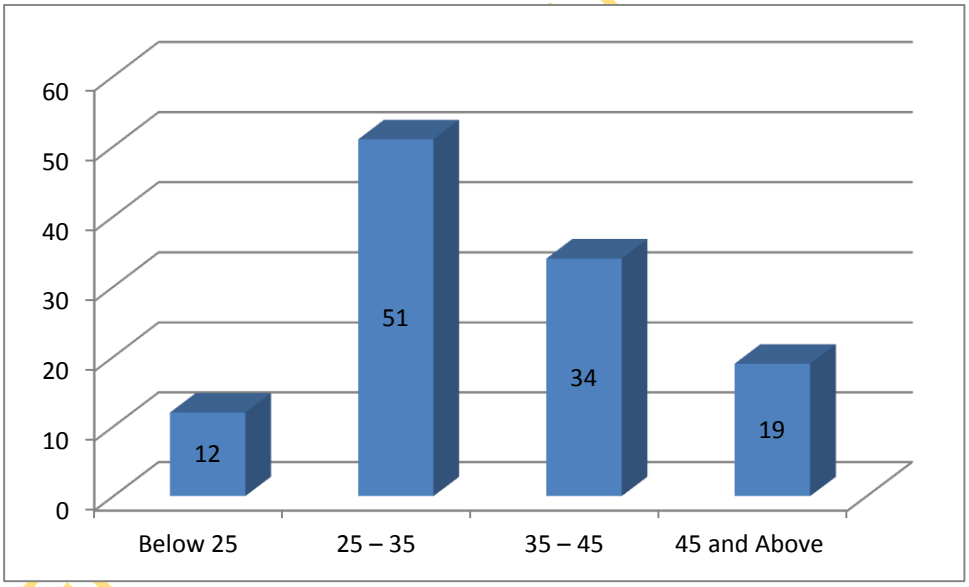


Figure 7: Histogram showing the frequency distribution of the ages

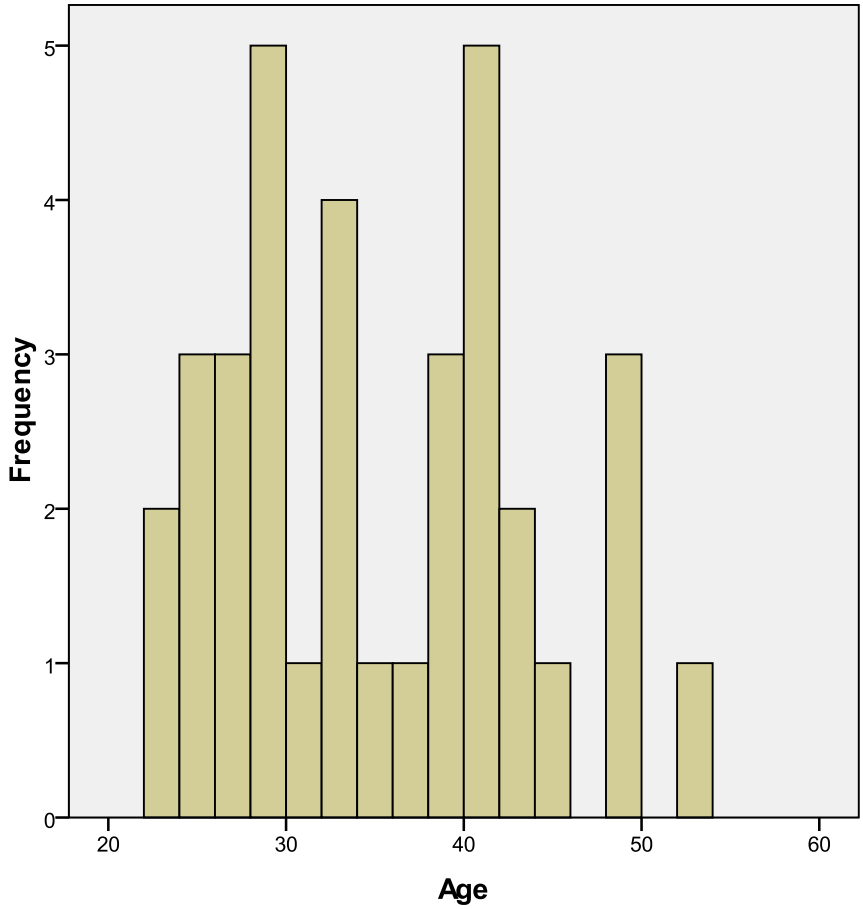


Figure 8: Pie Chart showing the employment status distribution

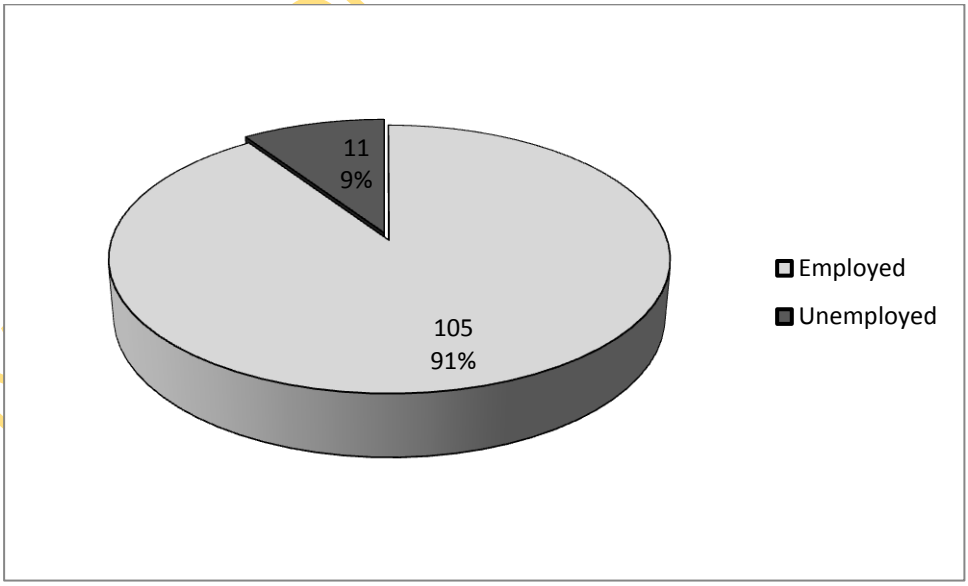
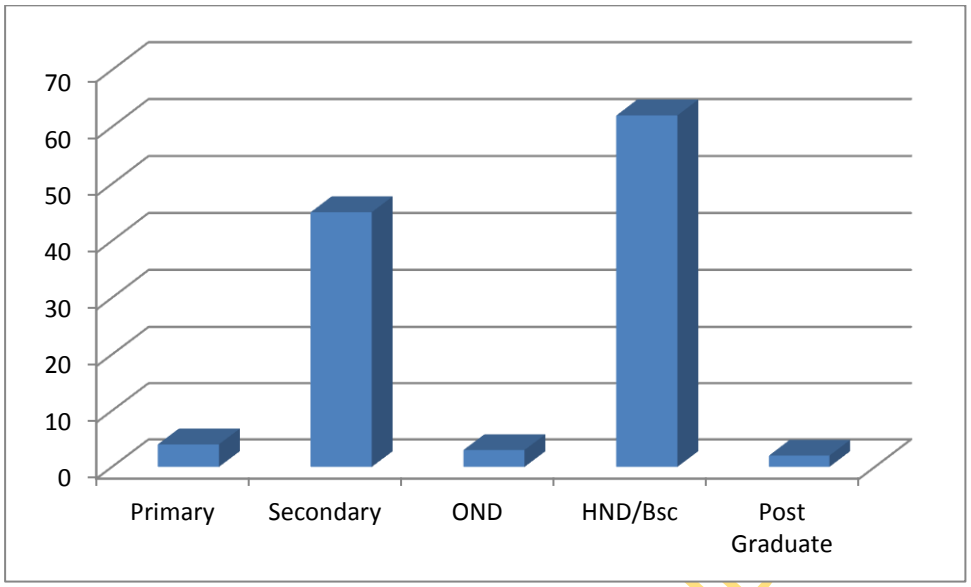


Figure 5: Bar Chart showing the level education distribution



4.2 COMPARISON OF DEPRESSION AMONG THE FOUR GROUPS (PRE AND POST ASSESSMENTS)

Table 4.7: Comparison of Depression among the Four Groups (Pre and Post Assessments)

S		PRE-ASSESSMENT		POST ASSESSMENT	
Therapy Group		Mean	Std. Deviation	Mean	Std. Deviation
A - Individual & Group Therapy	BDI Score	30.60	11.157	7.70	8.473
	CESD_Score	37.90	5.820	3.30	3.498
B - Individual Therapy	BDI Score	38.30	4.270	12.00	11.304
	CESD_Score	40.60	4.812	20.60	10.416
C – Group Therapy	BDI Score	38.70	7.409	19.60	10.239
	CESD_Score	30.80	11.063	24.40	16.794
D - Control Group	BDI Score	34.20	7.193	29.20	6.746
	CESD_Score	30.00	6.549	26.10	5.915

S		PRE-ASSESSMENT		POST ASSESSMENT	
Therapy Group		Mean	Std. Deviation	Mean	Std. Deviation
A - Individual & Group Therapy	BDI Score	30.60	11.157	7.70	8.473
	CESD_Score	37.90	5.820	3.30	3.498
B - Individual Therapy	BDI Score	38.30	4.270	12.00	11.304
	CESD_Score	40.60	4.812	20.60	10.416
C – Group Therapy	BDI Score	38.70	7.409	19.60	10.239
	CESD_Score	30.80	11.063	24.40	16.794
D - Control Group	BDI Score	34.20	7.193	29.20	6.746
	CESD_Score	30.00	6.549	26.10	5.915

Table 4.2 shows the comparison among the groups before and after the intervention. It was observed that of those given individual and group therapy there was a mean score of 30.6 (SD = 11.157) using BDI and a mean score of 37.9 (SD = 5.82) using CES-D before the intervention. However after the intervention the mean score reduced to 7.7 (SD = 8.473) using BDI and a mean score of 3.3 (SD = 3.498) using CES-D.

For those given individual therapy, there was a mean score of 38.3 (SD = 4.27) using BDI and a mean score of 40.6 (SD = 4.812) using CES-D before the intervention. However after the intervention the mean score reduced to 12.0 (SD = 11.304) using BDI and a mean score of 20.6 (SD = 10.416) using CES-D.

For those given group therapy, there was a mean score of 38.7 (SD = 7.4096) using BDI and a mean score of 30.8 (SD = 11.063) using CES-D before the

intervention. However after the intervention the mean score reduced to 19.6 (SD = 10.239) using BDI and a mean score of 24.4 (SD = 16.794) using CES-D.

For those in the control group, there was a mean score of 34.2 (SD = 7.193) using BDI and a mean score of 30.0 (SD = 6.549) using CES-D before the intervention. However after the intervention phase, the mean score reduced slightly to 29.2 (SD = 6.746) using BDI and a mean score of 26.1 (SD = 5.915) using CES-D.

Figure 6: Panelled Histogram Showing the level of depression (BDI score)

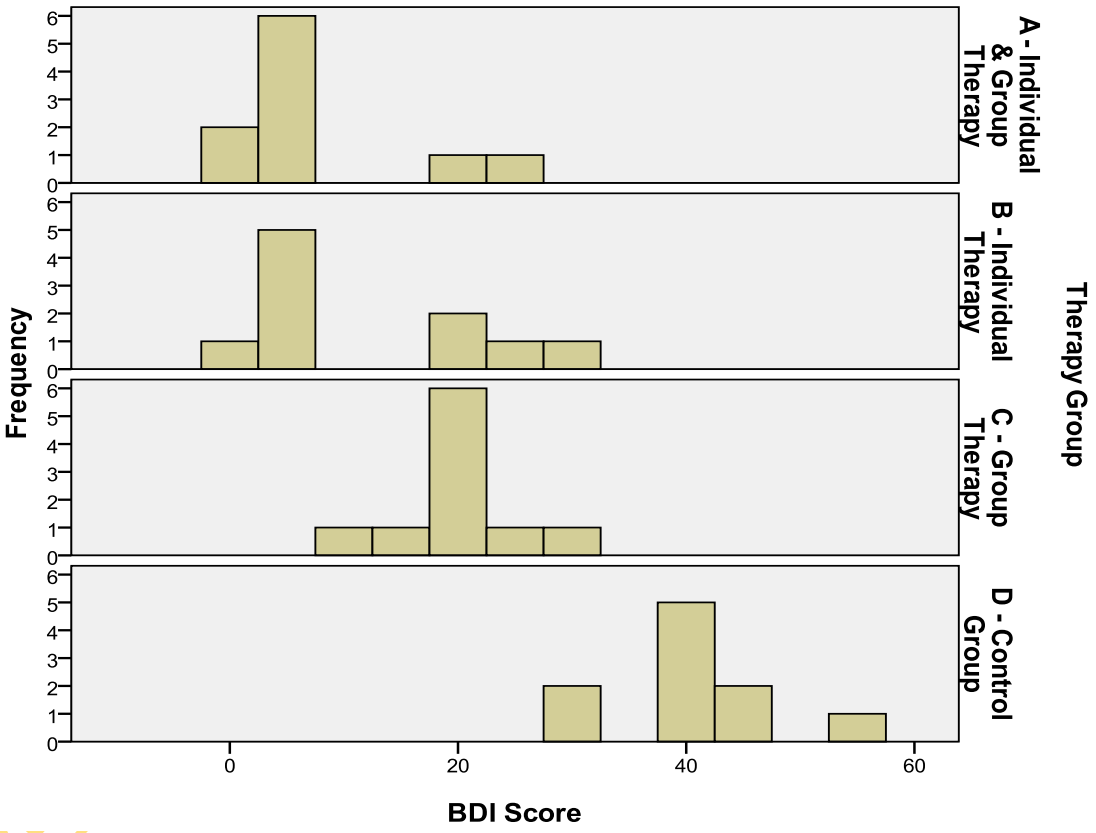
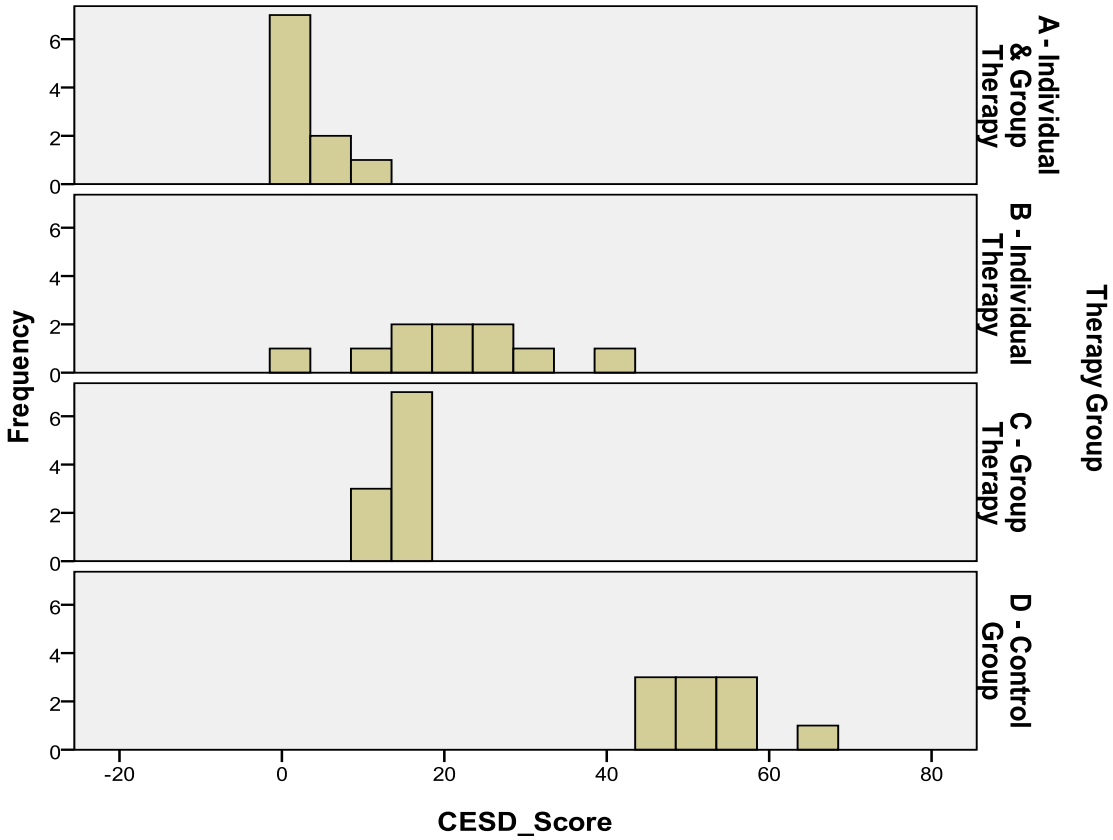


Figure 7: Panelled Histogram Showing the level of depression (CESD score)



4.3 TABLE 3 COMPARISON OF DEPRESSION LEVELS AMONG GROUPS

Table 4. 8: Depression Level among Groups Using BDI & CES-D (Post Test)

Therapy Group		Frequency (n)	Percentage (%)
BDI			
A - Individual & Group Therapy	Minimal	8	80.0
	Moderate	2	20.0
	Total	10	100.0
B - Individual Therapy	Minimal	6	60.0
	Moderate	3	30.0
	Severe	1	10.0
	Total	10	100.0
C – Group Therapy	Minimal	2	20.0
	Moderate	5	50.0
	Severe	3	30.0
	Total	10	100.0
D - Control Group	Moderate	1	10.0
	Severe	9	90.0
CES-D			

Therapy Group		Frequency (n)	Percentage (%)
BDI			
A - Individual & Group Therapy	Minimal	8	80.0
	Moderate	2	20.0
	Total	10	100.0
B - Individual Therapy	Minimal	6	60.0
	Moderate	3	30.0
	Severe	1	10.0
	Total	10	100.0
C – Group Therapy	Minimal	2	20.0
	Moderate	5	50.0
	Severe	3	30.0
	Total	10	100.0
A - Individual & Group Therapy	Minimal	10	100.0
B - Individual Therapy	Minimal	2	20.0
	Mild	2	20.0
	Moderate	3	30.0
	Severe	3	30.0
	Total	10	100.0
C – Group Therapy	Minimal	2	20.0
	Mild	4	40.0
	Severe	4	40.0
	Total	10	100.0
D - Control Group	Moderate	2	20.0
	Severe	8	80.0

Table 4.3 shows the depression level of the respondents among the various groups using the BDI and CES-D instruments. It was observed that majority of the respondents under the individual and group therapy had minimal depression (80.0%) with 20% being moderate using the BDI while the CES-D reports 100% minimal depression for this therapy group. None from this group had severe depression.

For those who received individual psychotherapy, 60% had mild depression, 30% were moderately depressed and 10% had severe depression using the BDI while CES-D reports 40% to be minimal and mildly depressed, 30% were moderately and 30% were severely depressed.

For those who received group therapy, 20% were minimal,65% were moderate while 30% had severe depression. From the CES-D, 20% were minimal while 40% had mild and severe depression respectively.

For those in the control group who had no intervention, 10% and 20% had moderate depression using the BDI and CES-D respectively while 90% and 80% respectively had severe depression using the BDI and CES-D.

4.4 COMPARISON OF CBT AMONG GROUPS

Table 4.4 shows a one-way ANOVA which tests if there is an overall significant difference among the four intervention groups.

Table 4. 9: A One-Way ANOVA testing The Difference Among The 4 Groups

On the Measures of Depression

		Sum of Squares	Df	Mean Square	F	P-Value
BDI Score	Between Groups	6234.275	3	2078.092	30.684	<0.001
	Within Groups	2438.100	36	67.725		
	Total	8672.375	39			
CESD_Score	Between Groups	13139.800	3	4379.933	110.744	<0.001
	Within Groups	1423.800	36	39.550		
	Total	14563.600	39			

From the table it was observed that there was significant difference among the four groups ($F=30.684$; $df = 3,36$; $p<0.01$) for BDI and ($F=110.744$; $df = 3,36$; $p<0.01$) for CES-D.

Since the F-test shows a significant difference among the different interventions, it is therefore important to see the significant difference between different pairs of interventions.

The following are the results obtained from the hypotheses tested:

Hypothesis one which states that age, gender, marital status, employment status and educational level will significantly, jointly and independently predict depression was tested with multiple regression. The result is presented in the table 4.5

Table 4.5: Multiple Regression Analysis showing influence of Socio-demographic Characteristics on Depression

	BDI			CES-D		
	R	R Square	ANOVA (p-Value)	R	R Square	ANOVA(p-Value)
	0.337	0.114	0.487 >0.05	0.387	0.150	0.670 >0.05
Predictors	Beta	T	P-Value	Beta	T	P-Value
(Constant)	-10.594	-.330	.745	-22.201	-.598	.557
Gender	-.002	-.010	.992	.273	1.128	.274
Age	.121	.471	.643	.170	.677	.507
Employment status	.243	.998	.331	.292	1.223	.236
Marital status	.097	.369	.716	-.097	-.380	.708
Education	.123	.544	.593	-.029	-.131	.897

P>0.05: Not significant at the level of 0.05

From table 4.5 above it could be seen that the predictors could only explain 11.4% of the variations that exist in the depression of the respondents (R-Square = 0.114) using BDI. By implication, the remaining 88.6% variations in depression level observed from the respondents could be explained by other factors not considered among the predictors. In addition, the predictors could only explain 15% of the variation that exist in the depression of the respondents (R-Square = 0.150)

using CES-D. By implication, the remaining 85% variation in depression level observed from the respondents could be explained by other factors not considered among the predictors. The results presented show that gender, age, employment status, marital status and educational level have no significant influence on depression: [F (40) =0.487; P>0.05 for BDI and [F (40) =0.670; p>0.05 for CES-D. Therefore, hypothesis one was not supported. This implies that gender, age, employment status, marital status and educational level have no significant influence on depression according to this study.

Second hypothesis which states that participants who receive group cognitive behaviour therapy will significantly report lower scores on depression than those who did not receive any cognitive behaviour therapy (Control) was tested with independent t-test. The result is presented in table 4.6.

Table 4.6: Independent Sample T-test Comparing Group CBT And Control Group on the measures of depression

	Therapy Group	Mean	Std. Deviation	T	Df	P
BDI Score	C - Group Therapy	19.60	10.239	2.218	18	<0.001
	D - Control Group	29.20	6.746			
CESD_Score	C - Group Therapy	24.40	16.794	-2.256	18	<0.001
	D - Control Group	26.10	5.915			

P: Significant at the level of 0.001

From table 4.6 above it was observed that there was a mean depression score of 19.6 (SD 10.239) from participants who benefited from only the group CBT using the BDI and a mean depression score of 24.4 (SD 16.794) from participants who

benefited from the group CBT using the CES-D. However, the results obtained from participants who benefited from only the group CBT using the BDI and CES-D were found to be significantly lower than those participants who did not receive any CBT (control group) having a mean depression score of 29.2 (SD 6.746) using BDI and a mean depression score of 26.1 (SD 5.915) using CES-D: [t (18) =-2.218,p<0.001] & : [t (18)=-2.256, p<0.001] for BDI and CES-D respectively Therefore, hypothesis 2 was supported. Participants who received group cognitive behaviour therapy significantly reported lower scores on depression than those who did not receive any cognitive behaviour therapy (Control).

Hypothesis three which states that participants who receive only individual cognitive behaviour therapy will significantly report lower scores on depression than those who did not receive any cognitive behaviour therapy (Control) was tested with independent t-test. The result is presented in the table 4.7.

Table 4. 7: Independent Sample T-test Comparing Individual CBT And Control Group on the Measures of Depression

	Therapy Group	Mean	Std. Deviation	T	Df	P
BDI Score	B - Individual Therapy	12.00	11.304	-6.774	18	<0.001
	D - Control Group	29.20	6.746			
CESD_Score	B - Individual Therapy	20.60	10.416	-8.316	18	<0.001
	D - Control Group	26.10	5.915			

P: Significant at the level of 0.001

From table 4.7 above, it was observed that there was a mean depression score of 12 (SD 11.304) from participants who benefitted from only individual CBT using the BDI and a mean depression score of 20.6 (SD 10.416) from participants who

benefitted from only individual CBT using the CES-D. These results were found to be significantly lower than the mean depression score obtained from participants from the control group 29.2 (SD 6.746) using BDI and 26.1 (SD 5.915) using CES-D :[t (18)=-6.774,p<0.001] & : [t (18)=-8.316, p<0.001] for BDI and CES-D respectively. Therefore, hypothesis three was confirmed. Participants who received only individual cognitive behaviour therapy significantly reported lower scores on depression than those who did not receive any cognitive behaviour therapy (Control).

Hypothesis four which states thatparticipants who receive combined (individual and group) CBT will significantly report lower scores on depression than those who will receive only individual CBT was tested with independent t-test. The result is presented in the table 4.8.

Table 4.8: Independent Sample T-test Comparing Group & Individual CBT Against Individual CBT on the measures of depression.

	Therapy Group	Mean	Std. Deviation	T	Df	P
BDI Score	A - Individual & Group Therapy	7.70	8.473	-.963	18	>0.05
	B -Individual Therapy	12.00	11.304			
CESD_Score	A - Individual & Group Therapy	3.30	3.498	-4.979	18	<0.001
	B - Individual Therapy	20.60	10.416			

P: Significant at the level of 0.001

From table 4.8 above, it was observed that there was a mean depression score of 7.7 (SD 8.473) from participants who benefitted from the combined (individual and group) CBT using the BDI and a mean depression score of 3.3 (SD 3.498) from participants who benefitted from the combined (individual and group) CBT using the CES-D. These results were not found to be significantly lower than the mean

depression score of those who received only the individual therapy 12.0 (SD 11.304) using the BDI: [t(18)=-.963,p<0.05 but were found to be significantly lower the mean depression score of those received only the individual therapy 20.6 (SD 10.416) using the CES-D :[t (18)=-4.979,p<0.001]. Therefore, hypothesis four was confirmed based on results obtained on the CES-D . Participants who received individual and group cognitive behaviour therapy significantly reported lower scores on depression than those who received only individual cognitive behaviour therapy (Control).

Hypothesis five which states that participants who received combined (individual and group) CBT will significantly report lower scores on depression than those who will receive only group CBT was tested with independent t-test. The result is presented in the table 4.9.

Table 4.9: Independent Sample T-test Comparing Group & Individual CBT against Group CBT on Measures of Depression.

	Therapy Group	Mean	Std. Deviation	t	Df	P-Values
BDI Score	A - Individual & Group Therapy	7.70	8.473	-9.779	18	<0.001
	C – Group Therapy	19.60	10.239			
CESD_Score	A - Individual & Group Therapy	3.30	3.498	-6.655	18	<0.001
	C - Group Therapy	24.40	16.794			

P: Significant at the level of 0.001

From table 4.9 above it was observed that there was a mean depression score of 7.7 (SD 8.473) from participants who benefitted from the combined (individual and group) CBT using the BDI and a mean depression score of 3.3 (SD 3.498) from participants who benefitted from the combined (individual and group) CBT using the CES-D. These results were found to be lower than the mean score of those who

received only the group CBT, which was 19.6 (SD 10.239) using the BDI and a mean depression score of 24.4 (SD 16.794) using the CES-D: [t (18)=-9.779,p<0.001] & : [t (18)=-6.655, p<0.001] for BDI and CES-D respectively. Therefore, hypothesis five was confirmed. Participants who received combined (individual and group) CBT significantlyreported lower scores on depression than those who received only group CBT.

Hypothesis six which states that participants who received combined (individual and group) CBT will significantly report lower scores on depression than those who will not receive any cognitive behaviour therapy (Control) was tested with independent sample t-test. The result is presented in the table 4.10.

Table 4.10: Independent Sample T-test Comparing Combined CBT (Group & Individual) And Control Group on the Measures of Depression

	Therapy Group	Mean	SD	T	Df	P
BDI Score	A - Individual & Group	7.70	8.473	-9.489	18	<0.001
	D - Control Group	29.20	6.746			
CESD_S	A - Individual & Group	3.30	3.498	-22.457	18	<0.001
	D - Control Group	26.10	5.915			

P: Significant at the level of 0.001

From table 4.10 above it was observed that there was a mean depression score of 7.7 (SD 8.473) from participants who benefited from the combined CBT (individual & Group) using the BDI and a mean depression score of 3.3 (SD 3.498) from participants who benefited from the combined CBT (individual & Group). These results were found to be significantly lower than the mean depression scores obtained from participants who did not receive any intervention (control group) 29.2 (SD 6.746) using BDI and 26.1 (SD 5.915) using CES-D: [t (18) =-9.489, p<0.001] &: [t (18) =-22.46, p<0.001] for BDI and CES-D respectively.

Therefore, hypothesis six was confirmed. Participants who received combined (individual and group) CBT significantly reported lower scores on depression than those who did not receive any cognitive behaviour therapy (Control).

CHAPTER FIVE

5.0 DISCUSSION AND CONCLUSION

5.1 DISCUSSION

The study examined the efficacy of group cognitive-behavioural therapy on Depression among Patients of Federal Neuro- Psychiatric Hospital, Yaba Lagos. The first stage of this study examined the level of depression among the participants both in the treatment and the control groups, while the second stage assessed the efficacy of cognitive behaviour therapy (combined individual and group, individual alone, Group alone and control) on depression among the patients with depression.

Six hypotheses were formulated in this study and were all tested with their significance proven, five of the hypotheses were confirmed while one hypothesis was not confirmed among the participants. From the results presented above, all the hypotheses were variously supported. Generally, several findings were implicated in this study and below are presentation of the discusszion of the results presented within the results section above.

5.1.1 Hypothesis one which posited that age, gender, marital status, employment status and educational level will significantly, jointly and independently predict depression was not supported. Result obtained revealed that gender, age, employment status, and educational level have no significant influence on depression. This goes to show that age, gender, employment status and educational level does not influence the development and maintenance of depression both jointly and independently.

5.1.2 Hypothesis two which posited that participants who received only group cognitive behaviour therapy will have significantly lower scores on depression than those who did not receive any intervention (control) was accepted. The result reveals that the participants that received group administered CBT achieved significantly lower scores than those who did not receive any intervention.

This result is consistent with the finding of David et al; (2001) that at the end of adding group psychotherapy to medication treatment in dysthymia (i.e., at termination), significant differences favoring the combined treatment were found on some symptom measures (Ham-D) and general functioning measures (CGI-S and GAF). Other measures (CDRS, IIP-high score) showed differences at the trend level and demonstrated medium to large effect sizes, with combined treatment patients showing better functioning in these areas than medication-only treated patients. Furthermore, 33.3% of patients in combined treatment meet rigorous criteria for improvement in symptomatology, global functioning, and personality variables, whereas only 6.7% of patients treated with medication alone meet these criteria.The above finding is consistent with those of Keller et al., (2000) that combined treatment was indeed more efficacious than medication alone in the treatment of chronic nonpsychotic major depression.

This result is consistent with the finding of Wilkinson and Goodyer (2008) that there were significantly greater reductions in ruminations in individuals who had CBT group than in individuals who did not receive any form of CBT in any form.

However, the above findings is not in line with the findings of Wikoso and Gover 2008 that there was no significant difference in the reduction of self-reported depressive symptoms between the control and the group that received group CBT.

They concluded that adding CBT to the treatment regimen causes a greater reduction in mood-related ruminative responses in depressed individuals. This result also conforms to the finding of Austin et al (2007), that group administered CBT was able to reduce the incidence of post natal depression by over 50% over time.

Their findings revealed that the effect sizes for GCBT over the control conditions range from small (0.1) to large (2.87) with the mean effect size of 1.10. Other findings indicated that GCBT yielded outcomes better than no-treatment controls and was comparable with other treatments (including both bona fide and non-bona fide comparison treatments). It was concluded that GCBT was effective for the treatment of unipolar depression and thus can be used with confidence.

Group CBT is now gaining grpound more in the treatment of depression. There is now an urgent need to develop and evaluate a coherent GCBT theory, in particular the roles of group processes in GCBT, before further major advancement in this area can be made.

5.1.3 Hypothesis three which posited that individuals who receive only individual CBT will significantly report lower scores on depression than those who did not receive any intervention. The result shows that individuals who received individually administered CBT scored significantly lower than those who did not received any intervention. This result is consistent with the finding of De Rubeis, et al. (2005) that the combination of both Cognitive Therapy and Antidepressant medications produced better efficacy in reducing depressive symptoms compared to placebo or pills at the 8-week assessment point.

Similarly, it is consistent with finding of Watkins et al; (2006) that Rumination Focused Cognitive behaviour therapy produced significant improvements in

depressive symptoms and co-morbid disorders: 50% of patients achieving full remission from depression, and a 71% reduction in co-morbid Axis I diagnoses. Importantly, RFCBT significantly reduced self-reported rumination, with rumination at pre-treatment equivalent to that found in currently depressed patients but the range of scores at post-treatment equivalent to levels of rumination observed in never-depressed participants.

It is also in agreement with the finding of Keller et.al (2000) that there was a greater remission rate in the patients who received both an antidepressant medication and a modified form of CBT compared with either treatment on its own .This result is consistent with the finding DeRubeis R.J, et al (2005) that the combination of both Cognitive Therapy and Antidepressant medications produced better efficacy in reducing depressive symptoms compared to placebo or pills at the 8-week assessment point.

Similarly, it is consistent with the finding of Watkins et al; (2006) that Rumination Focused Cognitive behaviour therapy produced significant improvements in depressive symptoms and co-morbid disorders with a mean reduction in Beck Depression Inventory of 20 points. Importantly, RFCBT significantly reduced self-reported rumination, with rumination at pre-treatment equivalent to that found in currently depressed patients but the range of scores at post-treatment equivalent to levels of rumination observed in never-depressed participants.

Cognitive behavioural therapy over decades has helped in understanding the thought process involed in the development of depression and how to do a cognitive restructuring to help such individuals. Administering CBT to a set of participants showed the importance of this technique in the management of depression. It is also in agreement with Dobson (1989) that cognitive therapy for depression was superior

to pharmacotherapy, behavior therapy, "other" psychotherapies, and a wait-list condition. The finding also give support to the findings of Bishop, Glen, Whalley, & Christie, 1981; Hollon, DeRubeis, Evans, et al., 1992; Murphy, Simons, Wetzel, & Lustman, 1984) that cognitive therapy is as effective as pharmacotherapy regardless of the severity of the depression.

Similarly, the above finding is consistent with the finding of Blackburn, Eunson & Bishop, 1986; Evans, Hollon, DeRubeis, et al., 1992; Kovacs, Rush, Beck, & Hollon, 1981; Shea, Elkin, Imber et al., 1992; Simons, Murphy, Levine, and Wetzel, 1986 that cognitive therapy of depression is more effective than pharmacotherapy alone in preventing relapse.

Furthermore, the finding is in line with the finding of Hollon, Thase, and Markowitz (2002) that the effects of cognitive behavioural therapy tend to be enduring. According to the authors, "several studies have shown that patients treated to the point of remission with cognitive therapy are only about half as likely to relapse following termination of treatment as are patients who enter remission after treatment with medications". Whether cognitive behavioural therapy is used contingently with medications or alone, its enduring effects on relapse and recurrence rates are an indication of the success of this method.

5.1.4 Hypothesis four which states that individuals who received combined (Individual and Group) CBT will significantly report lower scores on depression than those who received only individual CBT was similarly accepted. The result shows that although there was reduction in the post test result of both groups of participants, the group that had a combination of group and individually administered CBT had

significantly lower post test scores compared to the group that had only individually administered CBT.

The efficacy of a combination of both group and individually administered CBT over individually administered CBT was also established in several studies. The above finding gives support to the finding of Schein et al (2008) that individually administered CBT was effective while combinedgroup and individually administered CBT hadmore superiorresults. Although the study was conducted among army veterans undergoing a drug rehabilitation program, they observed that the combination appeared to have a "double edged sword" effect that adequately addressed the psychological issues presented.

Also the finding is in agreement with the finding of & Oei (2007) and Vos, Corry, Haby, Carter,& Andrews (2005) that delivering CBT for depression in a group format is a cost-effective alternative to individual treatment. They also suggested that group therapy may provide further advantages, as patients may benefit from group cohesion and normalization effects; hence using the group as an arena to engage in behavioral experiments, learning from others and functioning as co-therapists.

Similarly, the finding corroborates the finding of McDermut, Miller, and Brown (2001) shows that different forms of group therapy effectively reduce depressive symptoms.

The authors found an overall effect size of 1.03 in this study with CBT group therapy being more efficacious than psychodynamic group therapy. In a systematic review of 34 studies on group therapy for depression in 2008, Oei and Dinglefound measures of cognitions, behaviors and general health in addition to depression severity in their analyses and found an average effect size of 1.11 in favor of group CBT. Analyses of

21 uncontrolled studies showed an average effect size of 1.30 for comparisons between pre-treatment and post-treatment scores in the review. Hence, the review concluded that group CBT for depression is as effective as other bona fide treatments as defined by Wampold, Minami, Baskin, and Callen Tierney (2002). Also the finding is supported by the finding of Huntley, Araya, and Salisbury (2012) that there was a significant effect of group CBT over usual care at post-treatment and medium- to long-term follow-up; the standardized mean differences (SMDs) reported by the authors were -.55 and -.47, respectively.

The above finding is consistent with the finding of Alyson et al. (2012) that there was a significant effect in favour of group CBT immediately post-treatment (standardised mean difference (SMD) –0.55 (95% CI –0.78 to –0.32)). There was some evidence of benefit being maintained at short-term (SMD = –0.47 (95% CI – 1.06 to 0.12)) and medium- to long-term follow-up (SMD = –0.47 (95% CI – 0.87 to –0.08). They were able to conclude that group CBT confers benefit for individuals who are clinically depressed over that of indicidual CBT.

Though much still have to be understood and studies conducted, when it comes to the dynamism of group therapy. It is however clear that group intervention is highly effective when it comes to the management of depression against individual therapy.Group therapy provides further advantages, as patients may benefit from group cohesion and normalization effects and may also be able to use the group as an arena for engaging in behavioral experiments, learning from others and functioning.

5.1.5 Hypothesis five posited that participants who received combined (individual and group) will significantly report lower scores on depression than those who received only group CBT.

The result also shows that there was a reduction in the post test result of both groups, however the group that had a combination of individual and group administered CBT had a significantly lower score than the group that received only group therapy.

The above finding gives support to the findings of Nolen-Hoeksema (2004); Hollon, Hamman, & Brown, (2002); Lewinsohn, Clarke, Hops & Andrews, (1990); and Garber & Horowitz, (2002) that combined use individual and group CBT is effective in the management of depression than any other form of therapy. However, with reference to the population studied, many studies have not been done in this area of managing depressed persons with cognitive behavioural therapy. There is need therefore to borrow from this finding the CBT technique in the management of depression.

5.1.6 Hypothesis six which posited that participants who received combined (individual and group) CBT will significantly report lower scores on depression than those who will not receive any cognitive behaviour therapy (Control) was accepted. The results from the independent t-test showed that there was significant difference in the score of individuals who received combined cognitive behavioural therapy and the control group that did not have any therapy.

The above finding is in line with the finding of Abercrombie et al (2006) that individuals who received both the individual and group administered CBT performed better than other groups who did not receive any form of CBT. The findings also give support to the finding of Andre (2011) that OCD can be treated effectively with a group format of CBT as well as individual CBT, thus sparing some therapist resources.

5.2. CONCLUSIONS

5.2.1 Conclusion

The first stage of this study examined the level of depression among the participants both in the treatment and the control groups, while the second stage assessed the efficacy of cognitive behaviour therapy (combined individual and group, individual alone, Group alone and control) on depression among the patients with depression.

Six hypotheses were formulated in this study and were all tested with their significance proven, five of the hypotheses were confirmed while one hypothesis was not confirmed among the participants.

From the results presented above, all the hypotheses were variously supported. Generally, several findings were implicated in this study and below are presentation of the discussion of the results presented within the results section above.

It was also found that Participants who received either individual or group cognitive behavioural therapy had lower scores on depression than those who did not receive any intervention (control).From the findings of this study, Participants who received combined (individual and group) CBT had lower scores on depression than those who did not receive any cognitive behaviour therapy (Control). In addition, it was confirmed from this study that Participants who received combined (individual and group) CBT scored lower on depression than those who received either individual CBT or group cognitive behavioural therapy.

There are clinical advantages delivering CBT in group format with regards to the findings of this study. Some patients appear to benefit most from individualized approaches, whereas others appear to do very well in group CBT. The potential of attracting individuals who do not want the stigma of using formal psychiatric services

but who are willing to attend a psycho-educational large group in a community setting such as a leisure centre has real public mental health implications (Brown, 2005). Thus, there is a case for offering a choice of CBT delivery where this can be practically accommodated. There may also be a particular role for group CBT for people who have a very specific problem in common – such as postnatal depression (Milgrom 2005) – or for the treatment of depression associated with chronic physical ill health (National Collaborating Centre for Mental Health ,2009).

It is worth noting, however, that it is not clear which conditions, and equally which personal characteristics, indicate whether a group or individual approach is likely to be most helpful for a given individual. Questions remain, not only about the relative effectiveness of group CBT compared with individual therapy (Tucker 2007), but also about their relative acceptability for patients and drop-out rates (Heimberg 1993; Sharp ,2004; Semple ,2006).

It has been noted that there is no clear evidence yet for the use of group CBT with people from different cultures (Oei 2008). One critical issue pointed out by Oei & Dingle is that there is currently no coherent theory for group CBT that encapsulates both the content of the CBT model and the interpersonal processes that are occurring in the group. This may go far in explaining why the outcome research is at times conflicting – perhaps it is measuring different processes in different populations. When the theory is clearer we may be able to firmly determine the role of group CBT.

Innovative ways of delivering CBT in groups have been developed, as outlined in this study.

Also patients do well in group CBT and it is highly effectious in reducing symptoms of depression.

5.3 Implications and Recommendations

This study has brought to the fore the efficacy of combined group and individual cognitive behavioural therapy in the reduction of dysfunctional thoughts among patients with depression. Similarly participants who received psychological treatments experienced reduction in depression when compared to the control group. Hence, it is therefore recommended that patients undergoing treatment for depression should be exposed to cognitive behavioural interventions alongside with their medications, as the study revealed that participants in the control group still had high elevation on depression at post -test when compared to the treatment group.

The findings of this research have some implications which could be applied in many therapeutic and clinical settings. Much evidence has shown the efficacy of some psychological intervention especially combined approach with the use of group CBTwith Individual CBT. Depression has been reported as one of the main factor of psychiatric condition. Findings from available studies and this present study showed a substantial and contributory factor of group CBT in the treatment of Depression.

The study however confirms the role of group CBT as adjunct form of intervention in dealing with patients or clients who have been identified not to benefit from individual therapy. The group setting would enable such person to feel at ease bearing in mind that he is not only the sufferer. It creates an avenue for the individual to share his or her experiences with others. Cognitive Behavioural Therapy has shown to be an effective treatment on depression and improvement of self-esteem.

Moreover, adherence to intervention modalities involves a sequence of complex cognitive factors and behavioural skills. Patients must learn to be actively involved in their own care.

Given the efforts that patients on with this condition must remain compliant in spite of the reality of their lives; health care providers are under a particular obligation to assist in every way they can (APA Women's Health Conference Advisory Committee, 1996). Hence, mental health evaluation should be integral health care component of all patients receiving medical care. An increased awareness of these factors by professionals attending to these clients must entail, recognizing and potentially treating them in order to live quality life, function social and relate appropriately with people around them.

The study contributed to knowledge in the area of mental health, specifically;

1. The study demonstrates that both individual / group psychotherapy enhances positive mental health.
2. It shows that group cognitive therapy is highly effective in reducing symptoms of depression.
3. It reveals that group cognitive behaviour theapy is essential for the enhancement of positive cognition among the depressed patients.

5.4 Limitations

This study was restricted to patients at Federal Neuropsychiatric hospital Yaba, which is just one centre. Hence the result could not be generalized to patients in other psychiatric hospitals in the country. Another limitation of this study is on the sample size of the participants, a larger sample size may further be needed in the future which will further enhance the external validity of the result among patients undergoing treatment for depression.

Moreover, much study has not been done on the effect of CBT in depression among Nigerians; however, this study was limited only to the persons with

Depression. There is need for further research in this area to consider using a different population among the Nigerian samples.

REFERENCES

Abraham, E. (1948).*Note on psychoanalytic investigation and treatment of manic-depressive insanity and applied conditions*. London: Hogarth press.

Abramowitz, J., Foa, E.,& Franklin, M. (2003). Exposure and ritual prevention for obsessive-compulsive disorder: effects of intensive versus twice weekly sessions. *Journal of Consulting and Clinical Psychology*, *71*, 394–8.

Abramson, L. Y., Alloy, L.B., Hankin, B.L., Haeffel, G.J., MacCoon, D.G.,& Gibb, B.E. (2002). Cognitive vulnerability-stress models of depression in a self- regulatory and psychological context. In I.H. Gotlib and C.I. Hammer (Eds.) *Handbook of Depression (294-298).* New York: Guilford Press.

Abramson, L.Y., Metalsky, G.I.,& Alloy, L.B. (1989). Hopelessness Depression: A theory-based subtype of depression. *Psychological Review*, *96*, 358-372.

Amoran, O., Lawoyin, T.,& Lasebikan, V. (2007). Prevalence of depression among adults in Oyo State, Nigeria: A comparative study of rural and urban communities. *Australia Journal of Rural Health, 15(3),*211-215.

Anderson, R.,& Rees, C. (2007). Group versus individual cognitive–behavioural treatment for obsessive–compulsive disorder: a controlled trial. *Behaviour Research and Therapy*, *45*, 123–37.

Anthony, E.L. & Petronis, U.B.(1991).Gender, creativity, depression, and attributional style in adolescents with high academic ability. *Journal of Abnormal Child Psychology*, *21*, 455-467.

Asarnow, J. R., Carlson, G. A., & Guthrie, D. (1987). Coping strategies, self-perception, hopelessness, and perceived family environment in depressed and suicidal children. *Journal of Consulting and Clinical Psychology*, *55*, 361-366.

Asnis, G. M., McGinn, L. K., & Sanderson, W. C. (1995). Atypical depression: Clinical aspects and noradrenergic function. *American Journal of Psychiatry*, *152*, 31-36.

Barret, E.,& Boggiano, M.(1991).Intimate relationships and depression: Is there a causal connection? *Journal of Consulting and Clinical Psychology, 62*, 1033-1043.

Bebbington, P., Hurry, J., Tennant, C., Sturt, E.,& Wing, J.K. (1981). Epidemiology of Mental Disorders in Camberwell. *Psychological Medicine, 11(3),* 561–79.

Beck, A. T. (1963). Thinking and depression: Idiosyncratic content and cognitive distortions. *Archives of General Psychiatry*, *9*, 324-333.

Beck, A.T, Rush, A.J., Shaw, B.F., & Emery, G. (1979).*Cognitive therapy of depression.* New York: Guilford Press.

Beck, A. T. (1983). Cognitive therapy of depression: New perspectives. In P.J. Clayton & J. E. Barett (Eds.), *Treatment of depression: Old controversies and new approaches (pp.265-284)*. New York: Raven Press.

Beck, A.T., Ward, C., Mendelson, M, Mock, J., &Erbaugh, J. (1961). *An Inventory for measuring depression*. New York: Guilford Press.

Beekman, A. T. F., Copeland, J. R. M. & Prince, M. J. (1999). Review of community prevalence of depression in later life. *British Journal of Psychiatry, 174*, 307-311.

Bemporad, J. (1988). Long term analytic treatment of depression. In E.E. Beckham & W.R Leber (Eds), *Handbook of depression: Treatment assessment and research (pp. 82 – 99)*. Hanewood IL: Dorsey Press.

Bieling, P.J, McCabe R.E,& Antony, M.M. (2006).*Cognitive Behavioral Therapy in Groups*. New York: Guilford Press.

Bijl, R.J., Ravelli, A.,& van Zessen, G. (1998). 1998; Prevalence of Psychiatric Disorder in the General Population: Results of the Netherlands Mental Health Survey and Incidence Study (NEMESIS).*Social Psychiatry and Psychiatric Epidemiology,33,* 587–95.

Bisson, J, &Andrew, M. (2007). Psychological treatment of post-traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews*, *Issue 3*: CD003388).

Blatt, S.J., & Hamman, E. (1992). Parent-child interaction in the etiology of dependent and self-critical. *Clinical Review, 12,* 47 – 91.

Blumber,& Izard, (1985). 'Poverty and psychiatric status: Longitudinal evidence from the New Haven Epidemiologic Catchment Area study'.*Archival Gen enral Psychiatry, 48,* 470–474.

Bower, P.E &Gilbody,S. (2005). Stepped care in psychological therapies: Access, effectiveness and efficacy: Narrative literature review. *British Journal of Psychiatry, 186 (1*), 11-17.

Bowlby, J. (1973).*Attachment and Loss*. London: Hogarth Press.

Bolwby, J. (1982).*Attachment and Loss* (2nd Edition). New York: Basic Books.

Bowlby, J. (1988). *A secure base:Parent-Child Attachment and Healthy Human Development.* New York:Basic Books.

Branden, N. (1994).*Six Pillars of Self Esteem: The Definitive work on self-esteem*. New York: Bantam.

Brantley, P.J., Mehan, D.J. Jr., & Thomas, J.L. (2000). The Beck Depression Inventory (BDI) and the Center for Epidemiological Studies-Depression Scale (CES-D). In M.E. Maurish (Ed), *Handbook of Psychological Assessment in primary care setting*. London: Laurence Erbaum Associates Publication.

Berndt, E.R., Koran, L.M., Finkelstein, S.N., Gelenberg, A.J., Kornstein, S.G., Miller, I.M., Thase, M.E., Trapp, G.A., &Keller, M.B. (2000). 'Lost Human Capital From Early-Onset Chronic Depression'.*American Journal of Psychiatry, 157*, 940-947.

Blazer, D. (1999). EURODEP Consortium and late-life depression. *British Journal of Psychiatry, 174*, 284-285.

Brown, J. (2005). Are self-referrers just the worried well? A cross-sectional study of self-referrers to community psycho-educational stress and self-confidence workshops. *Social Psychiatry and Psychiatric Epidemiology, 40*, 396–401.

Brown, J. (2008). Can the effects of a 1-day CBT psycho-educational workshop on self-confidence be maintained after 2 years? A naturalistic study. *Depression and Anxiety, 25*, 632–40.

Brown, G.W., & Hanis, T.O. (1987).*Social origins of depression*. London: Tanistock.

Burlingame, G., Fuhriman, A., &Johnson, J. (2004). Small-group treatment: Evidence for effectiveness and mechanisms of change. In Bergin and Garfield's *Handbook of Psychotherapy and Behaviour Change* (5th edn) (eds MJ Lambert, AE Bergin, SL Garfield): 647–96.

Butter, A.C., Chapman, J.E., &Forman, E.M. (2006). The empirical status of Cognitive-Behavioral Therapy: A review of meta-analysis. *Clinical Psychology Review, 26(1)*, 17 – 31.

Callahan, C.M., Hendrie, H.C., Dittus, R.S., Brater, D.C., Hui, S.L., & Tierney, W.W. (1994). Improving treatment of late depression in primary care: A randomized clinical trial. *Journal of American Geriatric Society, 42*, 839-846.

Carson, C.R., Butcher J.N., & Mineka, S. (1998). *Abnormal Psychology and modern life.* New York: Longman.

Castro-Costa, E., Dewey, M., Stewart, R., Banerjee, S., Huppert, F., Mendonca-Lima, C., Bula, C., Reischies, F., Wancata, J., Ritchie, K., Tsolaki, M., Mateos, R., & Prince, M. (2007). Prevalence of depressive symptoms and syndromes in later life in ten European countries. The SHARE study. *British Journal of Psychiatry, 191*, 393-401.

Chang, W. (2009). *'Epidemiology, comorbidity, and course of adolescent depression.' Treatments for adolescent depression: Theory and practice. (pp. 3-25).* New York: Oxford University Press. xii, 328 pp.

Chen, J, (2007). Group cognitive behavior therapy for Japanese patients with social anxiety disorder: Preliminary outcomes and their predictors. *BMC Psychiatry, 7*, 69.

Clark, (eds), *Cognitive Behaviour Therapy for Psychiatric Problems*. Oxford: Oxford University Press. pp. 169-234.

Clark, G.N., Hawkins, W., & Murphy, M. (1995). Targeted Prevention of Unipolar Depressive Disorder in at risk sample of high-school adolescents – a randomized trial of group cognitive intervention. *Journal of American Child Adolescence Psychiatry, 34 (3),* 312 – 321.

Colman, I., Wadsworth, M. E. J., Croudace, T. J., &Jones, P. B.(2007). 'Forty-year psychiatric outcomes following assessment for internalizing disorder in adolescence.' *The American Journal of Psychiatry, 164(1),* 126-133.

Comer, A.G. (1996). The Australian National Survey of Mental Health and Well-Being. *The British Journal of Psychiatry, 177*, 325-330.

Cordioli, A. (2003). Cognitive–behavioral group therapy in obsessive-compulsive disorder: A randomized controlled trial. *Psychotherapy and Psychosomatics journal , 72*, 211– 216.

Corrigan, P.W., Watson, A.C. & Barr, L. (2006).*Journal of Social and Clinical Psychology, 25, (8),* 875-884.

Cromarty, P.(2004). Cognitive therapy and exercise for panic and agoraphobia in primary care: Pilot study and service development. *Behavioural and Cognitive Psychotherapy, 32*, 371– 374.

Copeland, J. R. M., Beekman, A. T. F., Dewey, M. E., Jordan, A., Lawlor, B. A., Linden, M., Lobo, A., Magnusson, H., Mann, A. H., Fichter, M., Prince, M. J., Saz, P., Turrina, C., & Wilson, K. C. M. (1999b). Cross-cultural comparison of depressive symptoms in Europe does not support stereotypes of ageing. *British Journal of Psychiatry, 174*, 322- 329.

Coyne, J.C. (1990). Interpersonal processes in depression. In G. Keitner (Ed), *Handbook of depression*. Washington, D.C.: American Psychiatric Press.

Cuijpers, P., Smit, F., &Voordouw, I. (2005). Outcome of cognitive behaviour therapy for minor depression in routine clinical practice. *Psychology and Psychotherapy: Theory, Research and Practice 78*, 179–88.

Cumming, M. E. (1995). Security, emotionality, and parental depression: A commentary. *Developmental Psychology, 31*, 425-427.

Davidson, G. C., & Neale, J. M. (2001). *Abnormal Psychology*. New York: John Wiley.

David, J. H., Suzanne, A. S., Wallner,L.S., Sarai, B., Christopher, M., Michael, F., David, K., Richard, N. R., & Arnold, W., (2001).Adding group psychotherapy to medication treatment in dysthymia.*Journal of Psychotherapy Practice and Research, 10*(2), 93–103.

Department of Health (2008).*Improving access to psychological therapies. Implementation plan: National Guidelines for Regional Delivery*. Department of Health.

Derogative, L.R., Lipman, R.S., Richels, K., Uhlenhuth, E.H. &Covi, L. (1974a). *The Hopkins Symtom Checklist (HSCL).* In P.Pinchot (Ed*), Psychological Measurements in Psychopharmacology (pp.79-11).* Basel: Karger.

DeRubeis, R.J., Evans, M.D., & Hollon, S.D. (1990). How does cognitive therapy work? Cognitive change and symptom change in cognitive therapy and pharmacotherapy for depression. *Journal of Consulting Clinical Psychology, 67 (6),* 862 – 869.

DeRubeis, R.J., Hollon, S.D., & Amsterdam J.D. (2005).Cognitive therapy vs. medications in the treatment of moderate to severe depression. *Archives of General Psychiatry, 62(4),* 409 – 416.

Dobson, K. (1989). A meta-analysis of the efficacy of cognitive therapy for depression. *Journal of Consulting and Clinical Psychology, 57*, 414-419.

Dohrenwend, B.P., &Dohrenwend, B.S. (1969). *Social status and psychological disorder: A causal inquiry.* New York: Wiley.

Dohrenwend, B.P., Levav, I., &Shrout, P.E.(1992). 'Socioeconomic status and psychiatric disorders: The causation-selection issue.' *Science, 255*,946–952.

Eaton, W. W., Anthony, J.C., Gallon, J., Cai, G., Romanski, A., Lykos, C., &Chen, L.S. (1997). Natural History of Diagnostic Interview Schedule/DSM-IV Major Depression. The Baltimore Epidemiological Catchment area follow-up. *Archives of General Psychiatry, 54*,993-999.

Ellis, A. (1958). Rational Psychotherapy. *Journal of General Psychology, 50*, 35-49.

Ellis, A. (1962). Reason and emotion in psychotherapy, and evaluation. *Journal of Consulting Psychology, 16*,319-324.

Elkin, I., Shea, T., &Watkins, J. (1989). National Institute of Mental Health of Depression Collaborative Treatment Program. *Archives of General Psychiatry, 46*, 971-982.

Engel, G. L. (1977). "The need for a new medical model: A challenge for biomedicine".*Science, 196*,129–136.

Engel, G. L. (1980). "The clinical application of the biopsychosocial model". *American Journal of Psychiatry, 137(5),* 535–544.

Ensminger, M. E.(1995). Welfare and psychological distress: A longitudinal study of African American urban mothers. *Journal of Health and Social Behavior, 36,* 346–59.

Ezeilo, B.N. (1981). Relationship of psychiatric diagnosis of sex and marital status in a Nigerian sample. *IRCS Medical Science: Psychology Psychiatry,Social Science and Occupational Medicine, 9,* 183-185.

Faris, B.,& Dunham, H.(1989).*Mental disorders in urban areas*. Chicago: University of Chicago Press.

Fenichel, O. (1945).*The Psychoanalytic theory of neurosis*. New York: W.W. Norton.

Fennell, M. (1989). Depression. In K. Hawton, P. Salkovskis, J. Kirk, & D.M. Clark (Eds.), *Cognitive behaviour therapy for psychiatric problems: A Practical Guide* (pp. 169–234). Oxford:Oxford University Press.

Fennel, M.J,V. (1989). Depression. In K. Hawton, P.M. Salkovskis, J.Kirk & D.M Clark (Eds.), *Cognitive-behaviour therapy for psychiatric problems: A practical guide.* Oxford: Oxford University Press.

Fester, C.B. (1978). A functional analysis of depression. *American Psychologist, 28,* 857-870.

Garba, J.,& Horowite, T.L. (2002). Depression in children. In I,H.Gottib, & C.L. Hammen (Eds), *Handbook of depression*. New York: Guilford Press.

Ge, X., & Conger, R. D. (1999). Adjustment problems and emerging personality characteristics from early to late adolescence. *American Journal of Community Psychology, 27,* 429-459.

George, B. (2008). 'Mental health, educational and social role outcomes of adolescents with depression.' *Archives of General Psychiatry, 59,* 225-231.

Giesler, R.B., Joseph, R.A., & Swann, W.B., Jr. (1996). Self-verification in clinical depression: The desire for negative evaluation. *Journal of Abnormal Psychology, 105*,358-368.

Gilbert, P.(1992).*Depression: The evolution of powerlessness.* Hove, UK: Lawrence Erlbaum Associates.

Gold, J.L.& Chorousos, V. (2002).New evidence on the relationship between income and health.' *Journal of Health Economics, 15,* 67–85.

Goldberg, D. (2001). Vulnerability factors for common mental illnesses. *British Journal of Psychiatry, 178 (suppl. 40),* s69–s71.

Gotlib, I. H., & Hammen, C. L. (1992). *Psychological aspects of depression: Toward a cognitive-interpersonal integration.* New York: Wiley.

Gove, J. & Shin, O.O. (1989).*Treatment of depression: An interpersonal system approach*. New-York: Pregamon.

Greden, J. (2001). 'The burden of recurrent depression: Causes, consequences, and future prospects.' *Journal of Clinical Psychiatry, 62(Suppl. 22),*5–9.

Gureje,O. (2009).*Self-Esteem in patients who recovered from psychosis: Profile and relationship to quality of life*. Australian: New Zealand.

Hammer, E.U. & Bremmer, T.T.(2001). Vulnerability factors for common mental illnesses. *British Journal of Psychiatry, 178 (suppl. 40),* s69–s71.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., Mcgee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107*, 128-140.

Harrington, R., Whittaker, J., & Shoebridge, O. (1998). Psychological treatment of depression in children and adolescents – a review of treatment research. *British Journal of Psychiatry 173*, 291 – 298.

Heim, P.B. (2005).*The interactional nature of depression: Advances in interpersonal approaches*. Washington, DC: American Psychological Association.

Hiroto, D. S., & Seligman, M.E.P. (1975). Generality of learned helplessness in man. *Journal of personality and Social Psychology, 31*, 311-327.

Hollon, S. D., & Beck, A. T. (1986). Cognitive and cognitive-behavioral therapies.
In S. L. Garfield, & A. E. Bergin, (Eds.), *The handbook for psychotherapy and behaviorchange: An empirical analysis, 2nd ed.(pp. 443-482).*New York: Wiley.

Hollon, S. D. (1981). Comparisons and combinations with alternative approaches.
In L. P. Rehm (Ed.). *Behavior therapy for depression (pp. 33-71).* New York: Academic Press.

Hollon, S.D, DeRubeis, R.J., & Shelton, R.C (2005).Prevention of relapse following cognitive therapy vs. medication in moderate to severe depression. *Archives of General Psychiatry, 62(4),* 417 – 422.

Hollon, S.D., Hamman, K.I, & Brown, L.L. (2002). Cognitive behavioral treatment of depression. In I.H. Growth & C.L. Hammen (Eds.). New York. *Journal of Psychiatry, 38*, 334-338.

Hollon, S. D., Shelton, R. C., & Loosen, P. T. (1991). Cognitive therapy and pharmacotherapy for depression. *Journal of Consulting and Clinical Psychology, 59*, 88-99.

Hollon, S. D., & Najavits, L. (1988). Review of empirical studies on cognitive

Therapies. In A. J. Frances, & R. E. Hales (Eds.), *American Psychiatric Press Review of Psychiatry, (Vol. 7, pp.643-666)* Washington, DC: American Psychiatric Press.

Holmes,E.,& Kivlighan, Jr.(2000). Comparison of Therapeutic Factors in Group and Individual Treatment Processes. *Journal of Counseling Psychology, 4*, 478-484.

Holzer, P.T. (1986).Is Depression a Risk Factor for Dementia or Cognitive Decline? A Review. *Gerontology, 46,* 219-227.

Jack, D.C. (1991). *Silencing the self: Women and depression.* New York: Harper Perennial.

Jacobson, N. S., & Hollon, S. D. (1993). Cognitive-behavior therapy versus pharmacotherapy: Now that the jury's returned its verdict, it's time to present the rest of the evidence. *Journal of Consulting and Clinical Psychology, 64*, 74-80.

Jacobson, N. S., & Hollon, S. D. (1993). Prospects for future comparisons between drugs and psychotherapy: Lessons from the CBT-versus pharmacotherapy exchange.*Journal of Consulting and Clinical Psychology, 64*, 104-108.

Joiner, J.E. (1995). The price of soliciting and receiving negative feedback. Self-verification theory as a vulnerable to depression theory. *Journal of Psychology, 104*, 364-372.

Jomer, T. E., & Metalsky, G.I. (1995). A perspective test of an integrative interpersonal theory of depression: A naturalistic study of college roommates. *Journal of Personality and Social Psychology*, *69*, 778- 788.

Jones, T., Stoddart, S.,& Mallick, W. (1995).Self-cognitions, stressful events, and the prediction of depression in children of depressed mothers. *Journal of Abnormal Child Psychology, 16,* 347-360.

Judd, L., Paulus, M., Wells, K., &Rapaport, M.(1996). Socioeconomic burden of subsyndromal depressive symptoms and major depression in a sample of the general population. *American Journal of Psychiatry,153,* 1411–17.

Karjalaine, P.L. (2003). The response-styles to depression. *Journal of Abnormal Psychology, 106*, 221-229.

Kaplan, H.I.& Sadock, B.J. (1998). *Synopsis of psychiatry: Behavioral sciences/clinical psychiatry (8th ed.).* New Delhi: B.I. Waverly Pty.Ltd.

Keller, J. (2000). The acute treatment of chronic depression: a comparison study of nefazadone, cognitive behavioral analysis system of psychotherapy, and theircombination. *New England Journal of Medicine, 342*, 1462-1470.

Kelly, M.A.R., Roberts, J.E., & Ciesla, J.A (2005). Sudden gain in cognitive-behavioural treatment for depression: When do they occur and do they matter? *Behavioural response therapy*, *43* (6), 703-714.

Kendall, P.C. (2005).*Child and adolescent therapy: Cognitive-behavioural procedures(3rd Ed).* New York: Guilford press.

Kindler, S.,Gardner, E.,&Prescott,Y.(2002).Vulnerability to depressive symptomatology: A prospective test of the diathesis-stress and causal mediation components of the hopelessness theory of depression. *Journal of Personality and Social Psychology*, *63*, 667-675.

Kennedy,C.A., Skurnick,J.H., Foley, M.,& Louria, D.B. (1995). Gender differences in HIV- related psychological distress in heterosexual couples. *AIDS Care*, *7*, 33-38.

Klein, D. E. (1996). Preventing hung juries about therapy studies. *Journal of Consulting and Clinical Psychology*, *64*, 81-87.

Klein, A., Markowitz, J., Rothbaum, B., Thase, M., Fisher, A., & Kocsis, J. (2013). The Relationship between the therapeutic alliance and treatment outcome in two distinct psychotherapies for chronic depression. *Journal of Consulting and Clinical Psychology*,*4*, 627-638.

Klosko, J. S., & Sanderson, W. C. (1999). *Depression: Clinical application of empirically supported psychotherapy.* Northvale, NJ: Jason Aronson.

Kodama, M., Katayanaji, H., Shimada, H., Sakano, (2008). The associations among stress coping, automatic-thought, state anxiety, and depressive symptoms in university students. Human Science Research and Wadeda. *The Journal of Nervous and Mental Disease, 196.*

Kohn, E.P.(1998).Is Depression a Risk Factor for Dementia or Cognitive Decline? A Review. *Gerontology, 46*, 219-227.

Korten, A., & Henderson, S. (2000). The Australian National Survey of Mental Health and Well-Being. *The British Journal of Psychiatry*, *177*, 325-330.

Kovacs, M., Rush, A. J.,& Beck, A. T., (1981). Depressed outpatients treated with cognitive therapy or pharmacotherapy. *Archives of General Psychiatry*, *38*, 33-39.

Krieger, E.U.(1997). Male gender role conflict, depression and anxiety: Clarification and generalizability to women. *Journal of Counseling Psychology. 50*, 167-174.

Kuyken,W., Dalgeish,T.,&Holden,E.R. (2007). Advances in cognitive – behavioral therapy for unipolar depression. *Canadian Journal of Psychiatry. 52*, 5 – 13.

Kuyken, W., Kurzer, N., & DeRubeis, R.J. (2001). Response to cognitive therapy in depression: The role of maladaptive beliefs and personality disorders. *Journal of consulting clinical psychology, 36,* 560-566.

Kvaglin, E. (1922).*Manic depressive insanity and paranoia*.Edinburg: E & S Livingstone.

Lewinsohn, P. M. (1974). Clinical and theoretical aspects of depression. In K.S. Calhoun, H.E. Adams, & K.M. Mitchell (Eds.), *Innovative Treatment Methods of Psychopathology.* New York: Wiley.

Lewinsohn, P.M, Clarke, G.N., Hops, J.N, & Andrews, J. (1990). Cognitive-behaviour treatment for depressed adolescents. *Behaviour Therapy, 21*, 385-401.

Lewinsohn, P. M., Gotlib, I. H., & Seeley, J. R. (1997). Depression-related psychosocial variables: Are they specific to depression in adolescents? *Journal of Abnormal Psychology, 106,* 365-375.

Lewinsohn, P. M., Munoz, R., Youngren, M., & Zeiss, A. M. (1986). *Control your depression*. New York: Fireside.

Lewinsohn, P.M., Rhode, P., Klein, D.N., &Seeley, J.R. (1999). 'Natural course of adolescent major depressive disorder, I: continuity into young adulthood.' *J Am Acad Child Adolescence Psychiatry, 38,* 56-63.

Lewinsohn, P.M., Sullivan, J.M., &Grossaip, S.J. (1980). Changing reinforcing events: An approach to the treatment of depression. *Psychotherapy: Theory Research and Practice, 17*,322-334.

Lewinsohn, P. M. (1975). The behavioral study and treatment of depression. In M. Hersen, & R. M. Eisler (Eds.), *Progress in behavior modification (pp. 19-64).*New York: Academic Press.

Link, J.I. & Dohrenwend, A.L. (1980). "Components of depressed mood in married men and women: The center for epidemiologic studies' depression scale." *American Journal of Epidemiology, 119,* 997-1004.

Lorant, E. (2003).A prospective study of risk factors for unipolar depression. *Journal of Abnormal Psychology, 97,* 251-264.

Luyten, P., Blatt, S. J., Van Houdenhove, B., & Corveleyn, J. (2006). Depression research and treatment: Are we skating to where the puck is going to be? *Clinical Psychology Review, 26,* 985-999.

Lynch, J.W., Kaplan, G.A., Shema, S.J.(1997). Cumulative impact of sustained economic hardship on physical, cognitive, psychological, and social functioning. *N Engl J Med, 337,* 1889–95.

Maier, S. F., Seligman, M.E.P., & Solomon, R.L. (1969). Pavlovian fear conditioning and learning helplessness. In B.A. Campbell & R.M Church (Eds.), *Punishment.* New York: Appleton.

Maruish, M.E. (2000). Application of the symptom Assessment- 45 Questionnaire (SA-45) in primary care settings. In M.E. Maurish, (Ed), *Handbook of psychological Assessment in primary care settings.* London: Lawrence Erlbaum Associates publishers.

Mark, F.L. &Marks, N. (1996). Flying solo at midlife: Gender, marital status, and psychological well -being. *Journal of Marriage and Family, 58(4),* 917-933.

Matze,E.U. (1967).'Childhood emotional and behavioral problems and educational attainment.' *American sociological review, 69(5),* 636-658.

McCluskey, U. (2002).The dynamics of attachment and systems-centered group psychotherapy. *Group Dynamics:Theory,Research and Practice ,6,*131-142.

McLaren, N. (2002). "The myth of the biopsychosocial model". *Australian and New Zealand Journal of Psychiatry,*36 (5), 701–703.

McGinn, L. K., Asnis, G. M., & Rubinson, E. (1996). Clinical and biological validation of atypical depression. *Psychiatry Research, 60,* 191-198.

McGinn, L. K., (1998). *Prevalence implications of axis II disorders in patients with atypical depression*. Paper presented at the annual convention of the Association for the Advancement of Behavior Therapy (AABT, Washington, DC.

McGinn, L. K., Young, J. E., & Sanderson, W. C. (1995). When and how to do longer-term therapy without feeling guilty. *Cognitive and Behavioral Practice, 2,* 187-212.

Miller, R. C., & Berman, J. S. (1983). The efficacy of cognitive behavior therapies: A quantitative review of research evidence. *Psychological Bulletin, 94,* 39-53.

Miech, R.A., &Shanahan, M.J. (2000).Socioeconomic status and depression over the life course. *J Health Soc Behav, 2000,41,* 162–76.

Mosely, T.A.(2002).Differential responses to male and female depressive reactions. *Journal of Consulting and Clinical Psychology, 45,* 994–1001.

Murphy, J.M. (1995). Diagnostic schedules and rating scales in adult psychiatry. In: Tsuang M, Tohen M, Zahner G, (Eds.),*Textbook in psychiatric epidemiology* (253-72). New York, NY: John Wiley and Sons, Inc.

National Institute of Mental Health (NIMH) 2005, Depression (09-07) Paykel et.al, 1999; Scott et al, (2000). Prevention of relapse by cognitive therapy in residual depression, a control trial. *Archives of General Psychiatry, 56,*829-853.

Nevello,O.P.(1999).*Sex differences in depression*. Stanford, CA: Stanford University Press.

Nolen-Hoeksema, S. (2002). Gender differences in depression. In H.G. Gotlib &C.L. Hammen (Eds.), *Handbook of depression*. New York Guilford press.

Nolen-Hoeksema, S., & Girgus, J.S. (1994). The emergence of gender differences in depression during adolescence. *Psychological bulletin, 115*, 424-443.

O'Hara, M. W., Lewis, D. A., Schlechte, J. A., & Varner, M. W. (1991). Controlled prospective study of postpartum mood disorders: Psychological, environmental, and hormonal variables. *Journal of Abnormal Psychology, 100*, 63-73.

Olanrewaju, A., Akintunde, A., Femi, F., Ibrahim, B. & Olugbenga, A, (2007). Pattern of Depression and Family Support in a Nigerian Family Practice Population. *The Internet Journal of Family Practice. 6:* 1.

Orley, J.A. (1972). Gender role in psychopathology. *East African Medical Journal, 49*, 16-2.

Ostelo, C.C. (2005). Linking employment status, maternal psychological well -being, parenting, and children's attributions about poverty in families receiving government assistance. *Family Relations, 51*, 112-120.

Ortega, S.T., &Corzine, J. (1990). *Socioeconomic Status and Mental Disorders. In: Greenley JR, editor. Research in community and mental health: A research annual: Mental disorder in Social Context, 6 (pp. 149-82).* Greenwich, CT: JAI Press.

Pearlin, L.I. & Johnson, J.S. (1977). Marital status, life -strains and depression. *American Sociological Review, 42*, 704-715.

Prince, M. J., Beekman, A. T. F., Deeg, D. J. H., Fuhrer, R., Kivela, S.L., Lawlor, B. A., Lobo, A., Magnusson, H., Meller, I., van Oyen, H., Reischies, F., Roelands, M., Skoog, I., Turrina, C., & Copeland, J. R. M. (1999b). Depression symptoms in late life assessed using the EURO-D scale. Effect of age, gender and marital status in 14 European centres. *The British Journal of Psychiatry, 174*, 339-45.

Radloff, L.S. (1977). The CES-D Scale: A self-report depression scale for research in general population. *Applied Psychological Measurement, (3*), 385-401.

Reinche, M.A., Dattilio, F.M.,& Freeman, A. (2003). *Cognitive therapy with children and adolescents: a casebook for clinical practice* (2nd ed). New York: Guilford press.

Reinherz,G.(2000).*Women and depression: Risk factors and treatment issues.* Washington, DC: American Psychological Association.

Ritsher,E.P. (2001).Union formation and depression:Selection and relationship effects. *Journal of Marriage and Family, 65(4),* 953-962.

Rhode, P.C., Lewinsohn, P.M., & Seeley, J.R. (1990). Are people changed the experience of having an episode of depression? A further test of the Scar hypothesis. *Journal of Abnormal Psychology, 99,* 264-271.

Robinson, L.A, Berman, J.S, & Neimeyer, R.A. (1990). Psychotherapy for the treatment of depression: A comprehensive review of controlled outcome research. *Psychology Bulletin, 105*, 1 – 20.

Ronanow,D.G.(2003).The impact of the family on health: The decade in review. *Journal of Marriage and the Family, 52,* 1059-1078.

Rush, A., Beck, A. T., Kovacs, M., et al. (1979). Comparative efficacy of cognitive therapy and imipramine in the treatment of depressed patients. *Cognitive Therapy and Research, 1,* 17-37.

Saisan, N.O. (2006). Poverty and economic hardship in families. In D.H. Demo, K.R. Allen, M.A. Fine (Eds.), *Handbook of family diversity (pp. 293-315).* New York, NY: Oxford Univ ersity Press.

Samson, J. A., Mirin, S.M., Hauser, S.T., Fenton, B.T., & Schildkrant, J.J. (1992). Learned helplessness ad urinary MHPG levels in unipolar depression. *American Journal of Psychiatry, 149*, 806-809.

Sanderson, W. C., Raue, P. J., & Wetzler, S. (1998). The generalizibility of cognitive behavior therapy for panic disorder. *Journal of Cognitive Psychotherapy, 12*, 323-331.

Scascighini, R.B.(2008).*Health psychology (pp. 180-212).* Hoboken, NJ: John Wiley &Sons, Inc.

Schneider, F. (1996). Cerebral blood flow changes in limbic regions induced by unsolved anagram tasks. *American Journal of Psychiatry, 153*, 206-212.

Schotte, C.K., van Den Bossche, B., de Doncker, D., Claes, S., &Cosyns, P.(2006) . A biopsychosocial model as a guide for psychoeducation and treatment of depression. *Depress Anxiety, 23(5),*312-24.

Segrin, C., &Abramson, M.Y. (1994). Negative reactors to depressive behaviors: A communication theories analysis. *Journal of Abnormal Psychology, 103*, 655- 668

Seligman, M. E. P., Abramson, L. Y., Semmel, A., & von Baeyer, C. (1979). Depressive attributional style. *Journal of Abnormal Psychology, 88*, 242-247.

Seligman, M.E.P. (1974). Depression and learned helplessness. In R.T. Friedman & M.M. Katz (Eds.), *The psychology of depression: contemporary theory and research*. Washington, DC: Winston-Wiley.

Seligman, M.E.P. (1975). Learned helplessness, depression, and the perception of reinforcement. *Behavioural Research and Therapy, 14*, 7- 17.

Sirey, J. (2004). Perceived stigma as a predictor of treatment discontinuation in young and older outpatients with depression. *American Journal of Psychiatry, 158*, 479–481.

Skeketee, G. & Chambless, D.L. (1992). Methodological issues in prediction of treatment outcome. *Clinical Psychology Review, 12*, 387 – 400.

Smeets, S.B. (2006).'Socioeconomic status, depression disparities, and financial strain: What lies behind the income-depression relationship?' *Health Economics, 14,* 1197–1215.

Stack, S.,& Eshleman, J.R. (1998). Marital status and happiness: A 17-nation study. *Journal of Marriage and the Family, 60(2),* 527-536.

Stone, A.A. (2002). Law, science, and psychiatric malpractice: A response to Klerman's indictment of psychoanalytic psychiatry. *American Journal of Psychiatry,14*, 419-427.

Stordal, E., Krüger, B. M., Dahl, N. H., Krüger, O., Mykletun, A., & Dahl, A. A. (2001). Depression in relation to age and gender in the general population: The Nord-Trøndelag Health Study (HUNT). *Acta Psychiatrica Scandinavica, 104(3),* 210–216.

Takeuchi, S.F. &Leaf, B.V. (1996). *Marital communication and decision making.* New York: Free Press.

Tasca, G., Balfour, K., Ritchie, L., & Bissada, H. (2007). The relationship between attachment scales and group therapy alliance growth differs by treatment type for women with binge-eating disorder. *Group Dynamics, Theory, Research, and Practice,11*, 1-14.

Thase, M. E.,& Kupfer, D. J.(1996). Recent developments in the pharmacotherapy of mood disorders. *Journal of Consulting and Clinical Psychology,64,* 646–59.

Tony, T.Z., &Derubis, R.J. (1999). Sudden gains and critical sessions in cognitive behavior therapy for depression. *Journal of Consulting Clinical Psychology, 67* (6), 894-904.

Trollor, J.N., Anderson, T.M., Sachdev, P.S., Brotady, H., & Andrews, G. (2007). Age shall not weary them: Mental health in the middle aged and the elderly. *Australian and New Zealand Journal of Psychiatry, 41(7),* 581-589.

Turner, R.J, Wheaton, B., & Lloyd D.A. (1996).The Epidemiology of Social Stress. *American Sociological Review,60,*104–25.

VanHook, M.P. (1996). Challenges to identifying and treating women with depression in rural primary care. *Social Work in Health Care, 23(3),*73- 92.

Urquhart, A.F. (2008).Marital status transitions and psychological distress: Longitudinal evidence from a national population sample. *Psychological Medicine, 29,* 381– 389.

Waite, E.B. (2000).The effects of marital and nonmarital union transition on health. *Journal of Marriage and the Family, 64,* 420– 432.

Watson, J.B. (1913). Psychology as the behaviourists viewed it. *Psychological Review, 20,* 158-177.

Weich, M.N. & Lewis, O.D.(1998).Poverty, unemployment, and common mental disorders: Population based cohort study. *BMJ, 317,* 115– 119.

Weissman, O.F. (1991).'Helpless attributions and depression in adolescents: The roles of anxiety, event valence, and demographics.' *Journal of Adolescence, 26,* 169-183.

Weissman, M.M., Wolk, S., &Goldstein, R.B., (1999). 'Depressed adolescents grown up.' *JAMA, 281,*1707-1713.

Weissman, O.F. & Paykel, A.S. (1974). 'Assessing depressive symptoms in five psychiatric populations: A validation study.' *American Journal of Epidemiology, 106,* 203–214.

Wolfe, B. E. (1994). Adapting psychotherapy outcome research to clinical reality. *Journal of Psychotherapy Integration, 4,* 160-166.

Williams, D.R., Takeuchi, D.T., & Adair, R.K. (1992). Marital status and psychiatric disorders among blacks and whites. *Journal of Health and Social Behavior, 33,* 140-157.

Wills, F. & Sanders, D. (1997).*Cognitive therapy*: *Transforming the Image.*London: Sage

Wishman, M.A. (1993). Mediators and moderators of change in Cognitive therapy of depression. *Psychological Bulletin, 114(2),* 248 -265.

Yalom, L.D (1983).*Existential Psychotherapy*. New York: Basic Books.

Yalom, I.D. (2005). *The Theory and practice of group psychotherapy.* (5th ed.), New York:Basic Books.

Zung, W.W.K., Broadhead, W.E., & Roth, M.E. (1993). Prevalence of depressive symptoms in primary care. *Journal of Family Practice, 37,* 337-344.

APPENDIX I

CONSENT TO PARTICIPATE IN A RESEARCH

You are hereby invited to participate in a study titled Efficacy of Group Cognitive-Behavioural Therapy among Patients with Depression in Federal Neuro- Psychiatric Hospital, Yaba Lagos. The study will entail you responding to some set of questionnaires which would help the researcher find out certain information and you might also be picked to participate in some psychotherapy session.

This study aims at finding out the efficacy of group cognitive behaviour therapy among depressed patients of the hospital. CBT is safe and has no risks associated with it. Participation is voluntary. Information about you will remain confidential. There will be no identifiers on any of the documents used in the final analysis of the data obtained from you.

I have read and understood the information contained in the Consent Form

- I agree to participate in the study
- I do not want to participate or
- I may withdraw before the end of the study

(Please tick as appropriate)

_____ _____

Signature/thumbprint of the participant **Signature of the Researcher**

APPENDIX II

QUESTIONAIRE

DEPARTMENT OF PSYCHOLOGY, UNIVERSITY OF IBADAN

Respondent,

This questionnaire is designed to gather a post-graduate research work. There is no right or wrong answers and your names are not required. Therefore your responses will be strictly confidential.

SECTION A

1. AGE actual age……..

2. SEX: Male ☐ **Female** ☐

3. EDUCATIONAL LEVEL: Primary ☐ **Secondary** ☐ **Tertiary** ☐

Others (Specify)…….

4. MARITAL STATUS: Single ☐ **Married** ☐

5. OCCUPATION: Employed ☐ **Unemployed** ☐ **Self Employed** ☐

6.DURATION OF ILLNESS…….

APPENDIX III

SECTION B

SECTION B – BECKS DEPRESSION INVENTORY

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the one statement in each group that best describes the way you have been feeling during the past two weeks, including today. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including item 16 (Changes in sleeping pattern) or item 18 (changes in appetite).

1. Sadness

0 I do not feel sad.

1 I feel sad much of the time.

2 I am sad all the time.

3 I am so sad or unhappy that I can't stand it.

2. Pessimism

0 I am not discouraged about my future.

1 I feel more discouraged about my future than I use to be.

2 I do not expect things to work out for me.

3 I feel my future is hopeless and will only get worse

3. Past Failure

0 I do not feel like a failure.

1 I have failed more than I should have

2 As I look back, I see a lot of failure

3 I feel I am a total failure as a person.

4. Loss of Pleasure

0 I get as much pleasure as I ever did from the things I enjoy.

1 I don't enjoy things as much as I used to.

2 I get very little pleasure from the things I used to enjoy.

3. I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

0 I don't feel particularly guilty.

1 I feel guilty over many things I have done or should have done.

2 I feel quite guilty most of the time.

3 I feel guilty all of the time.

6. Punishment Feelings

0 I don't feel I am being punished.

1 I feel I may be punished.

2 I expect to be punished

3 I feel I am being punished

7. Self-Dislike

0 I feel the same about myself as ever.

1 I have lost confidence in myself

2 I am disappointed in myself.

3 I dislike myself.

8. Self-Criticalness

0 I don't criticize or blame myself more than usual.

1 I am more critical of myself than I used to be.

2 I criticize myself for all of my faults.

3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

0 I don't have any thoughts of killing myself.

1 I have thoughts of killing myself, but I would not carry them out.

2 I would like to kill myself.

3 I would kill myself if I had the chance.

10. Crying

0 I don't cry anymore than I used to.

1 I cry more than I used to.

2 I cry over every little thing.

3 I feel like crying, but I can't.

11. Agitation

0 I am no more restless or wound up than usual.

1 I feel more restless or wound up than usual.

2 I am so restless or agitated that it's hard to stay still.

3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.

1 I am less interested in other people or things than before.

2 I have lost most of my interest in other people or things.

3 it's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.

1 I find it more difficult to make decisions than usual.

2 I have much greater difficulty in making decisions than I used to.

3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.

1 I don't consider myself as worthwhile and useful as I used to.

2 I feel more worthless as compared to other people.

3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.

1 **I have less energy than I used to have.**

2 **I don't have enough energy to do very much.**

3 **I don't have enough energy to do anything.**

16. Change in Sleeping Pattern

0 **I have not experienced any change in my sleeping pattern.**

1a **I sleep somewhat more than usual.**

1b **I sleep somewhat less than usual.**

2a **I sleep a lot more than usual.**

2b **I sleep a lot less than usual.**

3a **I sleep most of the day.**

3b **I wake up 1-2 hours early and can't get back to sleep.**

17. Irritability

0 **I am no more irritable than usual.**

1 **I am more irritable than usual.**

2 **I am much more irritable than usual.**

3 **I am irritable all the time.**

18. Changes in Appetite

0 **I have not experienced any change in my appetite.**

1a **My appetite is somewhat less than usual.**

1b **My appetite is somewhat greater than usual.**

2a **My appetite is much less than before.**

2b **My appetite is much greater than usual.**

3a **I have no appetite at all.**

3b **I for crave food all the time.**

19. Concentration Difficulty

0 **I can't concentrate as well as ever.**

1 **I can't concentrate as well as usual.**

2 **It's hard to keep my mind on anything for very long.**

3 **I find I can't concentrate on anything.**

20. Tiredness or Fatigue

0 **I am no more tired or fatigued than usual.**

1 **I get more tired or fatigued more easily than usual.**

2 **I am too tired or fatigued to do a lot of the things I used to do.**

3 **I am too tired or fatigued to do most of the things I used to do.**

21. Loss of Interest in Sex

0 **I have not noticed any recent change in my interest in sex.**

1 **I am less interested in sex than I used to be.**

2 **I am much less interested in sex now.**

3 **I have lost interest in sex completely.**

APPENDIX IV

SECTION C CES-D SCALE

Below are statements that describe the way people feel.

Pleas indicate the extent to which these statements apply to you in the past by ticking in the appropriate column.

		RARELY	SOMETIMES	OFTEN	ALWAYS
1.	I was bothered by things that usually don't bother me.				
2.	I did not feel like eating, my appetite is poor.				
3.	I feel that I could not shake off the blues even with the help of my family and friends.				
4.	I felt that I was just as good as other people				
5.	I had trouble keeping my mind on what I was doing.				
6.	I felt depressed.				
7.	I felt that everything I did was an effort.				
8.	I felt hopeful about the future.				
9.	I thought my life has been a failure.				
10.	I felt fearful.				

11.	My sleep was restless.				
12.	I was happy.				
13.	I talked less than usual.				
14.	I felt lonely.				
15.	People were unfriendly.				
16.	I enjoyed life.				
17.	I had crying spells.				
18.	I felt sad.				
19.	I felt that people disliked me.				
20.	I could not get going.				

CBT Modules and Sessions

SESSION	CONTENT
Session One	Introduction & review of depression:-this session served as an introductory session of the treatment group. The therapist explained the psychological implication of Depression and the reason for the psychotherapy sessions.
Session Two	CBT models- goals for therapy: - The therapist introduced the goals for the therapy which include: (a) Establishing collaborative treatment alliance. (b) Identification and reduction of dysfunctional thoughts feelings and behaviour. (c) Setting an agenda.
Session Three	This session entailed working on agenda, daily activity of negative thoughts/schedule was introduced in order to monitor activities of participants. -home-work/assignment was given after each session.
Session Four	The session entailed the use of techniques in dealing with problems identified and correcting irrational beliefs/ thoughts. (Cognitive restructuring): -home-work
Session Five	Participants were encouraged to engage in enjoyable activities and also enhance problem solving skills. (Behavioral activation).:-home-work
Session Six	Review, more activities to enhance and correct irrational thoughts/be (Home-work).
Session Seven	This session entailed recapitulation of the previous sessions, evaluation of activities with review of participants and also preparing participants for termination.
Session Eight	Review, evaluation and termination.