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THE EFFECT OF EMG-BIOFEEDBACK ON FEMALE PARTICIPANTS' MAJOR DEPRESSIVE DISORDER

By

HELI APELBAUM

A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, at the City University of New York.

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Abstract

THE EFFECT OF EMG-BIOFEEDBACK ON FEMALE PARTICIPANTS' MAJOR DEPRESSIVE DISORDER

by

Heli Apelbaum

Advisor: Professor C. T. LEE

This dissertation investigates the effect of biofeedback on depression, physiology, and homeostasis of the autonomic nervous system. To that end, both observational and experimental studies were conducted. In the observational study, the electromyograph, temperature, galvanic skin response heart rate, and blood pressure of 16 depressed and 16 non-depressed females were assessed to obtain a preliminary indicator of how the two groups rated on these physiological states. In the experimental study, 17 females suffering from Major Depressive Disorder (MDD) participated. They were randomly assigned to a control or experimental group. In the experimental group, nine participants received eight weeks of biofeedback therapy; in the control group, eight participants received a pseudo-treatment in which they underwent the same treatment procedures as those in the experimental group, but explicit feedback was withheld. Following the intervention, the experimental group's depression scores and muscle tension scores were significantly lower than those of the control group. No other result emerged from the study. On the basis of these results, a model was developed that postulates an independence of muscle tension from all other variables involved in depression symptomatology.

DEDICATION AND ACKNOWLEDGEMENTS

This research is lovingly dedicated to my husband and my children in heartfelt appreciation and acknowledgement of their support for this accomplishment.

My deepest gratitude to my mother and grandmother, who instilled in me a love for learning, and for my father, who encouraged me to persevere and work for the goals I set myself. I have been fortunate in having a dissertation committee of experts. Each offered his special skills, wisdom, and friendship, and supported me in the program.

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INTRODUCTION

This dissertation discusses the problem of depression, in particular its connection to stress. Because of the shortcomings of existing therapies this study conceptualizes a new way of treating depression. A review of the literature indicates that a person's homeostasis is disturbed by stress and how an individual interprets stress. This imbalance in the homeostasis of the organism may be a factor in causing depression. Thus it is concluded that because relaxation provides a person with a tool to handle and interpret stress, it should be helpful in the treatment of depression. It is speculated that once an individual is able to control his or her responses to stress, he or she will not suffer homeostatic imbalances, which may lead to depression. At the conclusion of this dissertation two investigations are presented, that attempt to improve the shortcomings found in studies previously conducted in this field. Age, type of depression, gender, and medications were all confounding factors in these studies, which may account for the divergent results.

The first study was conducted to determine if there is a physiological difference between various responses in the autonomic nervous system of depressed and non-depressed women. The hypothesis is tested that the autonomic responses of non-depressed persons, whose systems are in balance, are different from those of depressed persons, whose systems are not in homeostasis. An experiment will then determine if biofeedback assisted relaxation therapy improves depression. Should this treatment prove to be helpful, it should be added to existing therapies to obtain a more effective treatment of depression.

The Impact of Stress on the Etiology of Depression

Comprehensive theories have been proposed to explain the causal factors of depression. They include all of the general theoretical perspectives in the discipline of psychology: psychodynamic, behavioristic, cognitive, biomedical, phenomenological, and contextual. The cluster of symptoms, which we identify as "depression," is interpreted by each therapist or researcher according to the *Zeitgeist* of the epoch, and the culture into which the researcher is embedded. Each epoch produces a theory which fits with the spirit (*Geist*) of its time (*Zeit*). Thus, each theoretical perspective on depression's etiology is better understood as a relativistic rather than a universalistic interpretation of the disorder's causal factors.

For many decades it has been debated whether depression is a disease, that is genetically predetermined, or whether environmental factors are responsible for the onset of psychopathology. Research into the impact of environmental factors on mental health led to the formulation of a model for the effects of life events (Paykel, 1979). It states that any life event interacts with a host of other factors. This interaction of genetic factors, developmental events, physiological stressors, and psychosocially defined acute or chronic stress will eventually determine if the outcome is psychological disease and, if so, which specific illness.

The following Figure suggests a causal relationship between stress and depression:

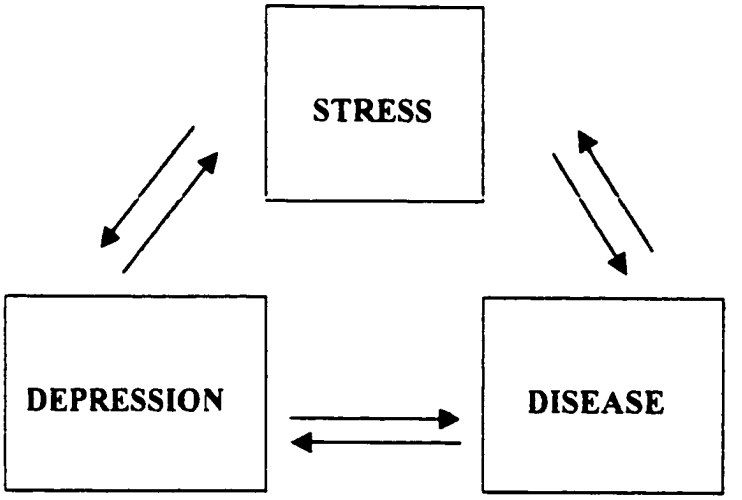


Figure 1. The Impact of Stress on the Etiology of Depression and Disease

Support for the influence of stress as a cause of depression comes from a national survey (1983) that showed that members of disadvantaged groups are more prone to develop depression. The study found that individuals who are uneducated, black, older, have experienced a recent income loss, are employed in a low prestige position, women (especially with more than three children under the age of 14), or are very poor, have the highest incidence of depression. This finding leads to the speculation that the common denominator among these

individuals is stress. Another category that is also inclined toward depression is that of affluent individuals. Again, one can speculate that wealthy people are more subject to stress because of the burdens and responsibilities associated with managing their wealth, or that persons in high managerial positions are driven by a Type A personality. However, these are only speculations. Results from animal studies further substantiate the idea that stress causes depression. When animals are subjected to a variety of acute stressors in an unpredictable sequence, they cannot adapt (Willner, 1994). Chronic stress causes (a) the activating effect of stressors in animals to decrease, (b) failure to increase fluid consumption when saccharin was added to drinking water, (c) impaired responsiveness to reward as assessed by intracranial stimulation, (d) reduced property of food to function as a reward, and

(e) the plasma level of corticosteroids to increase.

Some of these findings suggest that chronic stress produces anhedonia and a generalized insensitivity to reward. Tricyclic antidepressants normalized the rodents' decreased activating effect of stressors, and their increased level of plasma corticosteroids (except for monoamine oxidase inhibitor [MAOI], which was ineffective). Imipramine restored the failure to increase fluid consumption when saccharin was added to drinking water, and fluoxetine, maprotiline, and mianserin restored the rewarding properties of food.

The empirical evidence of the stressor's effect on depression's physiological symptoms leads to the following reconstruction of a more integrative theory to address similar issues in a broad perspective.

Change and Depression

Is stress the common denominator for the etiology of Major Depressive Disorder (MDD)? In order to answer this question, the following questions must first be considered. Why do some people become depressed while others do not? In depression, normal physical, mental and emotional growth, development, and functioning are impaired, and this impairment is exacerbated by stress. However, many individuals are subjected to the same stressors and never become depressed. Is this a quantitative problem? Does the accumulation of detrimental factors in peoples' lives bring them closer to their depression threshold? Or is it a qualitative problem? That is, are there people who are predisposed to fall ill when they encounter certain situations, compared with others who will never become depressed no matter how adverse the circumstances?

Depression is marked by a variety of symptoms, which can be caused by a variety of stressors. So far no particular stressor is found in all symptoms of depression, nor does any specific symptom always appear in patients suffering from depression. The only reliable predictor of depression is "change." The more marked the change, the higher the incidence of depression. However, the severity of the impact of a particular change is different for each individual. For example, depressed persons suffering from headaches tend to rate stressful events as more stressful, than control group participants (Ficek & Wittrock, 1995), and persons with dysfunctional psychological characteristics tend to have high psychological distress even when their physical findings are not more severe than those of other patients (Turk, Rudy, Kubinski, Zaki, & Greco, 1996). Furthermore, psychological appraisal is a crucial mediating

process in the experience of stress. Events are judged to be positive, negative, or neutral in their implications, and if they are judged negative, they are further evaluated as to whether they are harmful, threatening, or challenging (Lazarus & Folkman, 1984). Depression emerges when risk factors interact with challenging events in a person's life. A model of how gender differences emerge in depression states that girls are more likely than boys to carry risk factors for depression during childhood. These risk factors lead to depression in the face of challenges that increase in prevalence in early adolescence (Nolen-Hoeksema & Girgus, 1994). Depression can also be generated in previously normal personalities during times of war. That is, all individuals will develop depression when their level of exhaustion is reached. Thus depression will emerge when organisms fail to adapt to change. Depression signals the breakdown of an organism's ability to function in its environment.

In summary, the literature indicates that there are qualitative as well as quantitative aspects involved in the etiology of depression. Quantitative, since everybody will break down under extreme stress once a "threshold" is reached; qualitative since people are endowed with different risk factors and styles in the interpretation of events in their lives. Thus similar levels of stress are experienced differently by different individuals.

A Biopsychological Model of Mood

The next part of this paper discusses how stress can lead an organism to become depressed, with a special concern on which aspect of an individual changes in response to stress. Before the stressful event occurs, the organism is in stable condition at an optimal level, that is, in homeostasis. Support for the notion of homeostasis comes from Thayer's two-

dimensional mood theory (Thayer, Newman, & McClain, 1994). Thayer conceptualized a biopsychological model of mood which consists of two primary mood dimensions, energetic and tense arousal. Mood is closely associated with central states of general bodily arousal, with conscious components of energy (versus tiredness) and tension (versus calm). Thus people try to raise their energy levels and to reduce their tension levels to an optimum level in order to improve a depressed mood. For example, according to Thayer (1992) exercise and "giving oneself a pep talk" serve to increase energy, and relaxation or stress management techniques serve to reduce tension. Furthermore, Thayer's findings demonstrated that many body systems (cardiovascular, respiratory, skeletal-muscular and cognitive) interact in an integrated and holistic manner with a positive or a negative mood. Thus change in one system simultaneously affects other systems: mood is not purely a mental phenomenon, but involves the whole body. Thayer postulates that all moods fall into the two-dimensional space mentioned above and that further research must identify which mood-changing strategies apply for each mood.

A NEW THERAPY OUTLOOK

The fact that depression usually presents itself in connection with a host of other ailments and complaints supports Thayer's theory that a depressed person's system is neither in balance nor at an optimum level. Many medical conditions are associated with depression. Depression, in turn, is associated with high risk in patients afflicted with cardiopulmonary diseases (Berkow, 1987) such as myocardial ischemia, a temporary constriction of the coronary arteries that is associated with heart attacks (Gulette, 1997). In these cases, it is important that one not look for a cause and effect relationship (for example, does depression cause nausea, or does urinary frequency cause depression), but rather, one should take the whole organism into consideration. The depression occurs in relationship with an intricate and interactive multidimensional set of biologic, personality, and environmental factors. One or several components in a patient's total existence may have changed, causing an imbalance which leads to the malfunction of the whole system. Changing one component may result in a chain reaction which will alter the level at which the whole organism functions.

Table 1 gives an overview of the prevalence of depression in selected medical conditions:

Table 1

Prevalence of Depression in Selected Medical Conditions (Elliot, 1995)

Medical Conditions	Approximate Frequency of Depressive Syndromes (%)
ENDOCRINOPATHIES	
Hypothyroidism	40
Hyperthyroidism	30
Cushing's Syndrome	67
NEUROLOGIC CONDITIONS	
Stroke	50
Parkinson's Disease	40
Multiple Sclerosis	30
Head Injury	30
HIV Encephalopathy	30
Tumors	20
Alzheimer's Disease	40
CANCERS	
Pancreatic	50
Gastrointestinal	20
Gynecological	23
END-STAGE RENAL DISEASE	5
MYOCARDIAL INFARCTION	20
DIABETES MELLITUS	11
CHRONIC PAIN	32

Stress and Depression

A healthy person is not merely one who is not depressed at the moment, but one who is in a state of complete physical, mental, and social well-being. Stress interferes with that state of well-being. The particular stressor for each depressed person may not be obvious, but is discernible. In an attempt to find pure endogenous depression, 185 depressed persons were interviewed using a 61 item list (Paykel, 1979). Ninety-three percent responded that they had experienced a stressful life event within six months prior to the onset of the depression. Paykel then formulated a model for the effects of life events which states that any life event interacts with a host of other factors. This interaction of genetic factors, developmental events, physiological stressors, and psychosocially defined acute or chronic stress, will eventually demonstrate, not only if the outcome is psychopathology but also will determine the specific illness.

Over the years, researchers have found that people who experience various forms of stress often suffer from depression (Resident Services Committee, 1988; Trelawny-Ross, 1987). The functions of their immune systems are different from those of non-depressed persons (Altshuler, Plaeger-Marshall, Richeimer, Daniels, & Baxter Jr., 1989; Bartrop, Lazarus, Luckhurst, & Kiloh, 1975; Darko et al, 1986, 1987; Kiecolt-Glaser et al., 1987; Kronfol, House, Silva Jr., Greden, & Carroll, 1984; Linn, & Jensen, 1984; Schleifer, Keller, Camerino, Thornton, & Stein, 183, 1984, 1989). Their physiological variables, such as heart rate (Dawson, Schell, & Catania, 1977, Lader, & Wing, 1969), temperature (Nikitopoulous, & Crammer,

1976; Pflug, Erikson, & Johnsson, 1979, 1981), and galvanic skin response (GSR) (Dawson et al., 1977; Donat, & McCullough, 1983; Gilberstadt, & Maley, 1965; Iacono, Lykken, Peloquin, Lumry, Valentine, & Tuason, 1983; Lader, 1969; Mirkin, & Coopen, 1980; Noble, & Lader, 1972; Stern, Sila, & Word, 1960; Storrie, Doerr, & Johnson, 1981; Thorell, 1987, Thorell et al., 1987) are different from non-depressed persons. Also, the occurrence of depression with, for example, hypertension, a disease related to stress, may lead to cardiovascular disease and strokes. Thus depression is considered a marker for hypertension mortality (Simonsick, Wallace, Blazer, & Berkman, 1995).

Since it appears that excessive stress may lead to disease and depression, an effective depression therapy should include a stress management tool. Relaxation therapy has been known to counteract the detrimental effects of stress (Benson, 1975). The following section examines the relationship relationship between relaxation and depression:

The Effect of Relaxation on Depression

Relaxation is defined as a state of being in which there is freedom from physiological and psychological tension (Rees, 1995). Most cognitive-behavioral therapies incorporate relaxation exercises in the treatment of depression, but relaxation strategies alone are not considered appropriate to treat depression. As early as 1979 it was observed that when different therapies for depression were compared, relaxation seemed to be more helpful than psychotherapy or antidepressants (McLean & Hakstian, 1979). The success of the relaxation could not be explained and it was assumed that it must have been due to a placebo effect. These findings gave rise to the idea that relaxation therapy could be a helpful tool in the treatment of mood

disorders. Several studies have since supported the notion that relaxation alleviates depression: Relaxation (progressive relaxation) was found to be superior to the used antidepressants, and equally successful as cognitive behavior therapy (Murphy, 1995, Reynolds & Coats, 1986). Yet it was also found that a cognitive behavioral intervention (CBT) of only five to eight weeks was more fruitful than relaxation; however, by follow up, the relaxation group had achieved almost the same results as the CBT group (Wood, Harrington, & Moore, 1996). In other studies, relaxation (through meditation and progressive relaxation) improved the depression scores of anxious patients (Kabat-Zinn, et al., 1992; Miller, Fletcher, & Kabat-Zinn, 1995), of patients suffering from high blood pressure (McGrady, 1994), of persons with HIV (Eller, 1995), of persons in cardiac rehabilitation (Bohachick, 1984), and those of primiparas (Rees, 1995). It was also found that exercise, relaxation techniques, putting feelings in perspective, and controlling ones thoughts were the most fruitful strategies employed by patients to effect mood changes (Thayer et al., 1994).

Interestingly, during relaxation in autogenic training, persons showed a series of symptoms, called autogenic discharges (Luthe, 1969). These discharges are particularly evident in people with psychosomatic symptoms, psychodynamic disorders, multiple psychophysiologic reactions, or chronic affective or heterosexual deprivation. Table 2, below, illustrates the similarity between discharges, which occur in response to relaxation, and symptoms displayed by depressed patients.

Table 2 indicates that similar symptoms are found in both groups, that is the group of depressed persons, and the group of relaxing persons. Not only is every major system of the

person affected, but each is affected in a similar fashion. Major discrepancies in the reported symptoms for example, relaxing persons report certain sensations that are not reported by depressed persons, are probably due to the fact that these symptoms were compiled and reported by independent researchers in different studies. Since the symptoms are so similar, one must suspect that there may exist another link between relaxation and depression.

Upon completion of autogenic training, respondents report an overall improvement in their well-being (Luthe, 1969). The symptoms arising during the sessions are called "emotional discharges" (Luthe, 1969). Should the symptoms of depressed persons also be "emotional discharges," then depression may not be a disease, but rather a necessary stage in which a reorganization of the organism is taking place. This stage is reminiscent or similar to *Trauerarbeit*, the German expression that connotes "the work of mourning." Mourning is not merely a pathological symptom requiring suppression with antidepressants or other means. Mourning is a stage that a person must work through, in order to function again.

Trauerarbeit is a psychic discharge, necessary for the normal functioning of the organism. When channeled, it will not become self destructive or overwhelming. During *Trauerarbeit*, the therapist's tasks are to:

- prevent a person from committing suicide or starving to death;
- give the person more opportunities to have minor "discharge" periods during relaxation sessions, which will allow for the discharge to occur in a more controlled environment; and
- increase the person's awareness of overall health so that future discharges will not be necessary.

Table 2

Comparison of Symptoms Reported When Depressed and When Relaxing

Affected System	*Symptoms reported by:*	
	Depressed persons	*Relaxing persons*
Gastrointestinal	loss, or increased appetite overeating, weight gain, or loss, constipation, diarrhea, indigestion, nausea, urinary problems	hunger, salivation, sucking, pain in abdomen, and in stomach, nausea, vomiting, nausea, urination, the sensation of a varnishy, wooden taste
Cardiologic	palpitations, chest sensations, chest pain	palpitations, tachycardia, cramp like sensations of the heart, and pressure in the chest
Vascular	hypothermia, and headaches	feeling: cool, warm, burning, pulsations, blood flow, pain due to swelling, and tingling
Muscular	psychomotor retardation, and agitation	twitching, jerking, trembling, involuntary movements, muscular tension, stiffness, and feeling lame as if paralyzed
Sexual function	decreased or increased libido	erection, ejaculation, orgasm, vaginal contraction
Emotional Reactions	feeling blue, sad, hopeless, unworthy, like crying, anxious, irritable, unreal, and guilty	depression, crying, anxiety, fear, longing love and affection, loneliness, and euphoria
Cognitive	ruminations over the past, present, and future, poor concentration and memory, forgetfulness, indecisiveness, suicidal thoughts and death wishes, dizziness, weakness, general feeling of apathy	experience of intruding thoughts, memories, plans, loss of ability to concentrate, senses disagreeable and odors, sees colors and cloud like formations, shadows, forms which may be static or dynamic, faces, images, film and cinema strips, and hears voices.

The person will then reach a more stable state of homeostasis, with a heightened awareness of both his psychological and physiological needs. Fulfillment of these needs will help to maintain overall health.

In the homeostasis model it seems that during relaxation the person can be likened to a pendulum, swinging between two extremes for example, the state of too much and too little arousal. Both extremes are unpleasant and render the person incapable of function. The opposing sensations (described by Luthe as heaviness, sleepiness and warmth on the one hand, and flying, floating, fear, and coldness on the other) support the concept that the organism reaches a point of equilibrium at which it can function optimally by swinging back and forth between excessive states. Once the organism attains a state of balance, the person can cope with stress, which may otherwise have led to a breakdown of the body systems and to depression. This movement of the organism can be seen as analogous to the movement of a pendulum. The state of equilibrium or homeostasis is realized when the pendulum stops. I the pendulum is pulled back (cause by the introduction of a new stressor) and released, the pendulum will swing back and forth through an arc, which becomes shorter and shorter, until the pendulum stops again.

In order to reach and maintain the above mentioned state of equilibrium, one's position in the arc of the pendulum must be ascertained. If the present position is located near the extremes it has to be readjusted to an optimum level. Biofeedback is an excellent too to accomplish this readjustment, because it feeds back information about bodily functions to a person. Once a person is aware of how her body is functioning at that particular point in time, she can attempt

to control it. The following section describes the history of biofeedback and its success as a therapeutic device.

The History of Biofeedback in the Treatment of Stress-Related Illness

Because traditional medical approaches were insufficient for managing and treating many chronic diseases, conditions, and health damaging or maladaptive behaviors, behavior medicine emerged in the late 1970s as an outgrowth of psychophysiology, behavior therapy, and psychosomatic medicine. Behavior medicine recognizes the importance of stress, life-style, habits, and environmental variables in the etiology of diseases. It places much emphasis on the patient's role in prevention and recovery from organic diseases and conditions. Biofeedback is considered a major specialty within the broader field of behavioral medicine. It is a form of applied psychophysiology in which psychophysiologists manipulate psychological variables and observe the physiological effects. During biofeedback people alter their behaviors by using feedback from their physiology. These include muscle activity, peripheral blood flow, cardiac activity, sweat gland activity, brain electrical activity, and blood pressure.

It produces changes in bodily processes, which relieve the effects of stress. Thus many stress management systems and relaxation therapies now incorporate biofeedback into their treatment regimens (Schwartz, 1995).

Hence, biofeedback is a useful tool to teach relaxation, and to counteract the effects produced by psychological stress (Basmajian, 1989). It has been successful in the treatment of various stress-related diseases, including:

- anxiety (Lavallee, Lamontagne, Annable, & Fontaine, 1982),

- chronic fatigue syndrome (James, 1996),
- headaches (Blanchard et al., 1982; Levine, 1984),
- hypertension (Nakao et al., 1997),
- insulin-dependent diabetes (McGrady, Graham, & Balley, 1996),
- myopia (Rupolo, 1997),
- pain due to repetitive strain injuries (Moore & Wiesner, 1996),
- panic disorder (Oest & Westling, 1995),
- Raynaud's disease (Sedlacek, 1996),
- rheumatoid arthritis (Achterberg et al., 1981; Burke, Hickling, Alfonso, and Blanchard, 1985; Bradley, 1987, Mitchell, 1986), and
- temporomandibular disorder (Turk et al., 1996).

Moreover, it has been found that biofeedback improved not only the disease, but also the depression accompanying the disease (Burke et al., 1985; Frediani, 1988; Lavallee et al., 1982; Rupolo, 1997). Studies have also demonstrated that biofeedback is useful in the direct treatment of depression of a depressed businessman (Kumano, Horie, Shidara, Kuboki, Suematsu, & Yasushi, 1996), in chronic pain patients (Newton-John, Spence, & Schotte, 1994; & Peniston, Hughes, & Kulkosky, 1986), and in children (Labbe, 1993). Biofeedback normalizes the physiological values of depressed patients (Reda, Arciero, & Blanco, 1986) and eliminates performance deficits associated with depression on an escape/avoidance task (Klee & Meyer, 1981).

Because biofeedback is beneficial in the direct treatment of depression an observational Study and a controlled experiment were conducted to determine if biofeedback has an effect on depression scores and to examine which physiological responses, if any, changed in response to the biofeedback treatment as well as how the various responses are related to each other. Figure 2 illustrates how stress, depression, physiological responses and disease are interrelated:

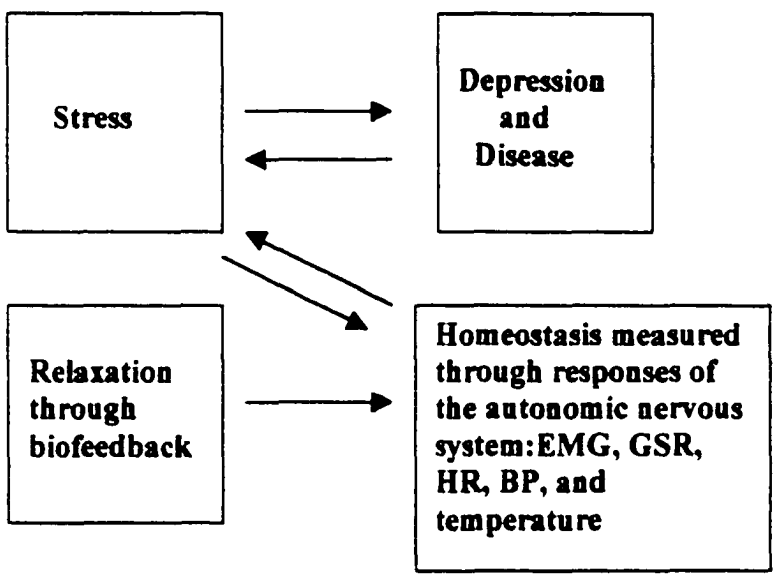


Figure 2. The Impact of Biofeedback on the Organism's Response to Stress

TWO INVESTIGATIONS CONDUCTED TO EXAMINE THE RELATIONSHIP BETWEEN PHYSIOLOGICAL VARIABLES, AND DEPRESSION

Physiological Measurements Taken in both Studies

The following section describes the physiological responses which were monitored, and measured during both investigations.

Electromyography (EMG)

When a muscle contracts, it tries to bring its two ends together. This results in a "muscle contraction," which involves a force and a movement. Because it is difficult to measure the grams of pull between the two ends of the muscle, one instead measures an electrical correlate of this activity. Electrical signals that are carried by "motor unit cells" cause the muscle fibers that comprise a muscle to contract simultaneously. The electrical activity corresponds to the muscle contraction in that muscle and can be measured with a fine-wire electrode. The electrode can either penetrate the skin above the muscle, or make contact with the skin above the muscle. This procedure of monitoring the electrical aspect of muscle contraction is called electromyography (EMG). It is measured in microvolts (a millionth of a volt), a unit of electrical pressure. Values above 10 microvolts indicate excessive muscle activity, and values below 5 microvolts indicate relaxation.

Frequent, excessive, and sustained psychophysiological tension and overarousal cause or exacerbate many health problems. It is therefore of interest to detect and manage these states of arousal (Schwartz, & Schwartz, 1995). Hitherto, the EMG values of depressed persons have never been compared with those of non-depressed individuals.

Temperature

Smooth muscle activity or peripheral vasoconstriction is another physiological process commonly associated with overarousal. Yet it is not feasible to simply measure the changing diameter of peripheral blood vessels, or the smooth muscle activity that is responsible for these adjustments. Here again, a correlate is used to monitor the process. Dilated vessels pass more warm blood than constricted vessels. Thus, the temperature of the surrounding tissue changes according to smooth muscle dilation and provides a good correlate of vascular diameter. The effect can best be detected in the extremities (such as fingers and toes), where the vascular diameter changes are more noticeable because the amount of surrounding tissue is relatively small and warms and cools rapidly in response to changes in the blood supply. Temperature is read out in degrees Fahrenheit which is an indirect measure of vasoconstriction (Schwartz et al., 1995). Peripheral temperatures below 80° Fahrenheit are considered low; above 90° are considered high (White & Tursky, 1982).

Differences in the temperature of depressed persons were reported (Avery, 1986). Patients had higher ear canal temperatures when they were depressed than during recovery. Six manic depressive patients experienced temperature fluctuations (Nikitopoulos, 1976; Pflug, 1979, 1981). They had a disorganized circadian rhythm with a higher mean temperature than normal individuals, and earlier daily temperature maxima. The overall temperature of the depressed group and the control group did not differ (Souetre et al., 1989), but the circadian rhythm was abnormal. The group of depressed patients had a significantly lower diurnal and a

higher nocturnal peak of body temperature than the control group. Unfortunately, the authors did not distinguish between unipolar and bipolar depression.

Blood Pressure (BP)

Blood pressure is a physiological variable that can be influenced by stress. Studies reveal that blood pressure levels tend to rise over time and may lead to hypertension in patients leading stressful lives. Cardiac output and total peripheral resistance determine blood pressure. Cardiac output equals heart rate (beats per minute) times stroke volume output, which is the amount of blood ejected with each beat of the heart. Factors controlling heart rate and stroke volume include sympathetic and parasympathetic nerve activity. Total peripheral resistance is the resistance or impediment to the flow of blood in the arteries and veins. Sympathetic activity, circulating substances in the blood, and tissue conditions influence the total peripheral resistance. If sympathetic activity increases, arterioles constrict and th· ·sistance to the blood flow becomes larger, raising blood pressure. Hormonal, neural and kidney factors influence blood pressure. Stretch receptors in the walls of the heart and in the blood vessels monitor blood pressure. They respond to distention caused by increased pressure by sending an electrical signal through nerves to the vasomotor center in the medulla of the brain. Nerve responses then begin, and within a few seconds blood pressure decreases.

Blood volume determines blood pressure in the long run. When a person consumes too much fluid, the pressure rises. This signals the kidneys to excrete fluid until blood pressure decreases.

Aldosterone is a hormone released by the adrenal cortex that also influences blood pressure. Aldosterone is released in response to decreased plasma levels, sodium is then reabsorbed by the kidneys, fluid loss decreases, and fluid balance is restored.

Blood pressure is expressed as systolic/distolic in millimeters of mercury. Systolic is the maximum pressure during ejection of blood from the heart, and diastolic is the minimum pressure during cardiac relaxation. Mean arterial pressure is the average blood pressure driving blood through tissues. A diastolic pressure below 85mmHg is considered to be normal; between 85 and 89 is high normal; 90 to 104 is mild hypertension; 105 to 114 moderate hypertension; 115 or greater is severe hypertension. When the diastolic pressure is below 90 mmHg, a systolic pressure below 140 mmHg indicates normal blood pressure; between 140 and 159 is borderline isolated systolic hypertension; 160 or higher is isolated systolic hypertension. A pressure of 200/140 is considered malignant hypertension (Wilson et al., 1991).

Standardized and repeated blood pressure measurements are important to establish the actual blood pressure value. The arm should be positioned at heart level and the patient should be quietly sitting with his back supported for five minutes.

After reviewing several studies, Stein (1994) reported that blood pressure levels in depressed persons are comparable with those of controls. Blood pressure is measured here in this study to determine if there is a relationship between it and the other physiological variables.

Heart Rate

The heart rate comprises impulses generated in the wall of the right atrium. These impulses establish the rate at which the heart beats. Changes in heart rate are dictated by the balance between the acceleration function of the sympathetic fibers and the deceleration function of the parasympathetic innervation from the vagus nerve. Heart rate is influenced by stress, exercise, muscle tension, personality, motivation, emotion and organic pathology (Basmajian, 1993). A heart rate of between 50 and 100 beats per minute is considered normal (Bia et al., 1981).

It is not clear whether the heart rates of depressed individuals differ from those of normal controls. Some researchers have found no difference between the heart rates of depressed and non-depressed individuals (Donat, & McCullough, 1983). Others (Dawson et al., 1977; Iacono et al., 1983) found heart rates in depressed individuals to be higher as well as less responsive to changes in stimuli, compared with the heart rate of non-depressed persons. It should be pointed out that these findings include persons using antidepressants. Antidepressants elevate heart rate, but heart rate variability, which is controlled by the parasympathetic nervous system, decreases significantly (Stein, 1994; Rechlin, 1995).

Galvanic Skin Response (GSR)

Sweat gland activity is a good indicator of autonomic nervous system arousal. The amount of sweat a gland releases is an indicator of a person's arousal level. Since one cannot tell whether a sweat gland is "on," how much sweat is being secreted, or how many such glands are active, one cannot directly measure sweat gland activity. Hence, this must be determined

indirectly through measuring a person's skin conductance. Sweat contains electrically conductive salts which make sweaty skin more conductive to electricity than dry skin; thus skin conductivity corresponds to sweat gland activity. This is known as GSR, "galvanic skin response." A skin conductance device applies a small voltage to an area of the skin where there are many sweat glands. The palm surface of the hand is generally used to measure the amount of electrical current that the skin will allow to pass. The current is read out in units of electrical conductance called "micromhos." The number of micromhos indirectly measures the amount of sweat released by the gland, and therefore the activity of the autonomic nervous system. Conductance values of above 5-10 micromhos are considered to be relatively high, whereas those below 1 micromho are thought to be low (Schwartz et al., 1995).

Higher skin conductance levels in depressed persons than non-depressed psychiatric subjects and healthy persons were reported (Lewinsohn, 1974). However, several studies (Dawson et al., 1987; Donat & McCullough, 1983; Gilberstadt et al., 1965; Iacono et al., 1983; Lader & Wing, 1969; Mirkin & Coppen, 1980; Noble & Lader, 1972; Stern et al., 1960; Storrie et al., 1981; Thorell, 1987, 1987) suggest that GSR may be a marker for depression, since depressed individuals tend to have significantly lower GSRs. Thorell (1987) describes the GSR of suicidal depressed patients to be even lower than the GSR of non-suicidal depressed patients, thus indicating that more severe symptoms may be correlated with lower GSR. It is not clear if treatment and recovery correlate with a rise in GSR (Dawson, 1977; Stern et al., 1960) or if GSR levels remain lower, even after recovery, than in never before depressed controls (Iacono et al., 1983; Storrie et al., 1981; Thorell, 1987).

Reda's Study and its Shortcomings

It is of interest how physiological measurements relate to each other. Reda et al. (1986) compared the correlation between EMG, GSR, and temperature and found that in normal subjects whose bodies are most likely to be in a state of homeostasis there was a positive correlation between EMG and GSR values; that is, the more muscle tension, the higher the galvanic skin response. EMG and temperature were negatively correlated; that is, the more muscle tension, the lower the body temperature. And, as expected, GSR also negatively correlated with body temperature. Reda et al. trained a group of normal subjects to relax with the help of biofeedback; as the subjects learned more and more about how to relax the correlations between the different values became more clear cut. For example: at the beginning of the training, the correlation between EMG and GSR was 0.40; after 30 sessions, the correlation was 1.00. EMG and temperature correlation was –0.30 at the first recording, and after 30 sessions it was 0.90. Reda also looked at these correlations in depressed patients and found that their values differed from the values found in normal subjects. Depressed patients had no clear-cut distinction between the correlations of EMG-GSR, which was 0.10 at the beginning of the training; the correlation between GSR and temperature was 0; and between EMG and temperature was –0.15. At the end of 30 sessions, their values started to resemble the values of non-depressed persons with about 5 sessions of biofeedback training. It should be noticed that in contrast to schizophrenic patients, whose readings were totally erratic, the pattern of correlation measurements taken during the training was the same for normal and depressed individuals: the positive and negative correlations between the different variables

were progressively increasing. The trend was such that, had the training for depressed subjects continued, they would have eventually reached the same correlations as those of non-depressed persons.

Unfortunately, Reda et al. did not report if the patients' depression abated as their values reached normal levels. If we assume that the mind and body interact closely, we must speculate that the depression did improve, because the body's physiological responses were so drastically changed.

The following investigation is an attempt to improve the shortcomings found in the studies reviewed above.

Firstly, patients diagnosed as suffering from bipolar disorder were excluded.

Secondly,Patients, taking antidepressants were excluded. The physiological measurements of persons suffering from bipolar disorder, or who are taking antidepressants, may be different from patients suffering from major depressive disorder (MDD) (Iacono et al., 1983; Lader et al., 1969; Stern, 1960).

Thirdly, only persons younger than 50 years old were included because age is negatively correlated with the mean temperature level in normal persons (Souetre, 1989), and GSR is affected by the age of depressed persons (Stern, 1960, & Thorell et al., 1987). Another indicator for the influence of age on the functioning of the physiology of depressed persons comes from a study on the effects of stress on the responses of the immune system. As depressed patients age, their immune systems' responses to stress become more and more different. The largest change occurs around the age of 50; before 50, the immune system

becomes more active in response to stress; after 50, the response drops after a stressful event (Schleifer, 1989).

Fourthly, only women who were younger than 50 years old participated to avoid confounding by an age or gender factor. The pattern of depressive symptoms in older women is different from younger ones (Newman, Engel, & Jensen, 1991). Younger women suffer from what is conventionally considered MDD, but older women suffer from a quieter form of depression, which is termed "depletion syndrome." This finding is reminiscent of "involutional melancholia," which is Kraeplin's classification of depressive symptoms in older women with onset during the menopausal age. Furthermore, there is a gender difference associated with emotional processing in women. Depression in women interferes significantly with the right hemisphere's arousal and vigilance system, which is connected to the autonomic nervous system (Liotti & Tucker, 1992).

Fifthly, EMG biofeedback was chosen because a drop in muscle tension correlated with an improvement of depression in persons enrolled in a running program (Hannaford, Harrell, & Cox, 1988).

Sixthly, an observational study assessed if there was a difference between the physiological responses of depressed and non-depressed women.

Seventhly, A controlled experiment shed light on whether EMG-biofeedback assisted relaxation could directly lead to a reduction in depression, and how it affected the physiological variables and the homeostasis of the autonomic nervous system in depressed patients.

An Observational Study to Examine How Physiological Measurements of Depressed and Non-Depressed Women Differ

In the following study, the physiological variables of women scoring in the depressed range of the Beck's Depression Inventory, the Zung's Self-Report Depression Scale, and the Clinician Evaluation Guide were compared with those of women scoring in the normal range. Previous studies have failed to clarify the relationship between depression and physiological variables, such as GSR, temperature, blood pressure (BP), pulse (P), and EMG. The women's EMG, P, BP, GSR, temperature, and depression scores were gathered and compared with the scores of non-depressed women. The hypothesis here was that if the physiological variables between depressed and non-depressed women differed, this would reveal an underpinning physiology of depression.

Method

Participants. Sixteen women between the ages of 18-40 years were randomly recruited from a medical office and a college. Eight who scored in the depressed range on the Beck Depression Inventory, on the Zung Self-Rating-Depression Scale and on the PRIME-MD, and eight who scored in the normal range were asked to participate in the study. All depressed women were classified as suffering from MDD by the PRIME-MD scale. The overall, (i.e., of depressed and non-depressed women) correlation between Zung's Self-Report Depression Scale and Beck's Depression questionnaires was $r = .90$, $p < .05$.

Two women were excluded from the experiment: the first scored in the normal range but was taking an antidepressant and could therefore not qualify for the non-depressed group; a second participant scored in the depressed range but was excluded because she suffered from

bipolar disorder. Volunteers were not paid for participating and were treated in accordance with the "Ethical Principles of Psychologists and Code Conduct" (American Psychological Association, 1992).

Materials. The women's physiological variables were measured with a J&J Physiological Data system I-410 BCS, is a computerized system which measures the participants' temperature, GSR, and muscle tension. The electrodes used to measure the EMG and GSR are Ag/AgChl surface electrodes, which are used with a conductive paste in the electrode cup. In order to remove skin debris, which may interfere with signal detection, the skin surface was slightly abraded prior to the application of the electrodes. BP and P were measured with an AND-Ua-701-digital blood pressure meter. The depression scores were obtained with three questionnaires:

Firstly, the Beck Depression Inventory (BDI): It has a test-retest correlation of .90 (Lightfoot & Oliver, 1985), a content validity of .67 (Moran & Lambert, 1983), a concurrent validity of .72 between clinical ratings of depression and the BDI for psychiatric patients, and .60 for non-psychiatric clients (Beck, Steer, & Garbin, 1988). The correlations among the BDI, MMPI-D Scale, and Zung Self-Rating Depression Scale is more than .55 (Schaefer et al., 1985). Although the BDI was not designed to discriminate among patients with different psychiatric diagnoses, it can discriminate between Dysthymia, MDD, and Mental Health (Steer, Beck, Riskind, & Brown, 1986). The BDI measures one underlying syndrome of depression in different patient populations (Clark, Cavanaugh, & Gibbons, 1983). The BDI consists of 21 items. Six refer to vegetative symptoms and the remainder refer to mood and cognitive

symptoms. Each item consists of a graded series of statements ranging from 0-3; the total score range is from 0-63. Scores above 13 indicate depression. Beck & Beamesderfer (1974) suggest that it is not useful to estimate the test-retest stability of the BDI, because the patient's depression scores change over to the passage of time.

Secondly, the Zung Self-Rating Depression Scale: It has a concordance rate of 0.89 (Gabris, & Peters, 1985), an internal validity of 0.88, a significant discriminant validity, and a predictive validity of 0.77. The scale consists of 20 items which represent affect (2 items), somatic concomittants (8), and psychological concomitants (10). The subject must rate each item on a four-point scale in terms of frequency. The total score is derived by dividing the sum of the raw scores by the maximum possible score of 80, and multiplying it by 100. Scores of below 50 lie in the normal range; 50-59 indicate a mild depression; 60-69 a moderate depression; and, 70-100 a severe depression.

Thirdly, the PRIME-MD: This is a new procedure for diagnosing mental disorders in primary care. The overall accuracy rate for the agreement between PRIME-MD diagnoses and those of independent health professionals was 88% (Spitzer et al., 1994).

Self-report rating scales have been selected because they are the method of choice in studies which compare depression treatments (Greenberg, Bornstein, Greenberg, & Fisher, 1992). When compared with data derived from interviewer rated scales such as the Hamilton Rating Scale for Depression (HRSD), patient ratings provide more accurate descriptions of how the patient reacted to the treatment. It is suspected that clinician ratings are more biased because the interviewers have a vested interest in finding the experimental treatments to be superior to the control conditions. Among other advantages of the self-report scales are that they are short, do not tire out the participants, and are useful

tools for quickly identifying depression. There are no inconsistencies and variations across interviews by the clinician, and no problems of interrater reliability. These are popular questionnaires, making it possible to compare the results of this study with those of other studies conducted in this field.

Design and Procedure. This was a between-groups design. During a routine check-up in a medical office, or participation in a college introductory psychology course, the participants were informed that they might participate in a study about depression and its physiological correlates. They were asked to sign an informed consent form (Appendix A) to fill out Beck's Depression Inventory, Zung's Self-Rating Depression Scale, and PRIME-MD. Those scoring in the moderately to severely depressed range were assigned to the depressed group. Temperature, GSR, and EMG were measured with a computerized biofeedback system. Measurements were recorded manually every five minutes. Blood pressure and pulse were measured once at the session beginning, with an AND-UA-701-digital blood pressure meter. This was a between-groups design.

Results

The difference between the depressed and the non-depressed group was analyzed with a (2 X 5) MANOVA on the five outcome variables which were blood pressure, temperature, heart rate, muscle tension, and galvanic skin response. There was no difference between the physiological measurements of depressed and non-depressed persons $F(5,10) = 0.879$, $p =$ (0.529). The independent variables were EMG, GSR, Temp., P., and BP. The descriptive statistics collected from that sample are shown in Table 3 and Figures 3-7 illustrate the fact that

no significant difference between the various measurements of the depressed and non-depressed women could be found.

A covariance matrix of the five physiological variables of the depressed women was not significantly different from a covariance matrix of the same variables of the non-depressed women $X^2(15) = 15.590, p = 0.410$. This suggests that the homeostasis of the autonomic

Table 3

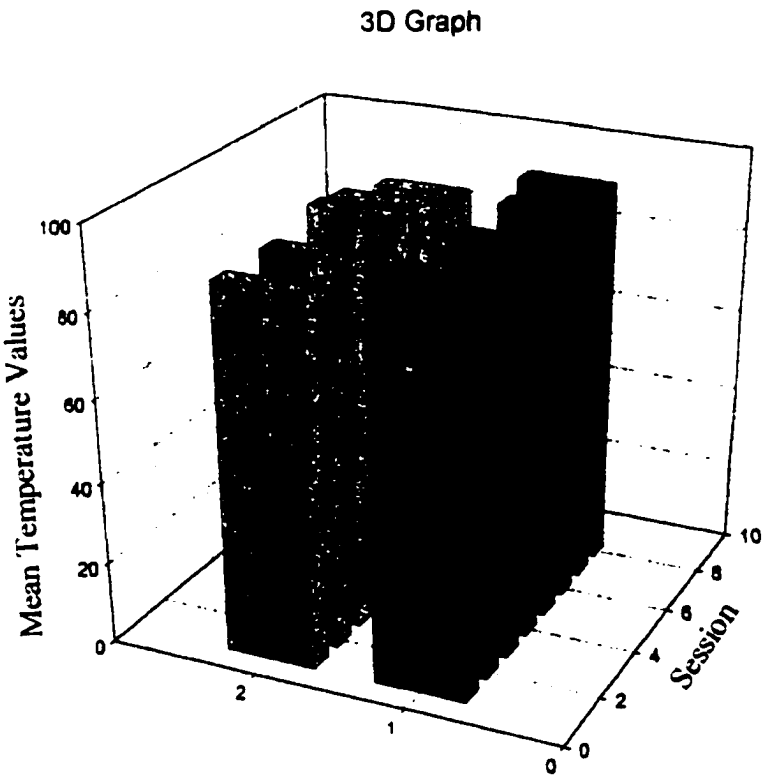
Descriptive Statistics (Means, Standard Deviations) of Data collected in the Observational Study

Physiological	*Depressed*		*Non-Depressed*	
	Mean	*SD*	*Mean*	*SD*
Mean of Temperature	85.10	7.25	87.76	7.61
Logarithm of Blood Pressure	2.04	0.04	2.05	0.03
Logarithm of Pulse	1.89	0.07	1.86	0.06
Square of EMG	2.83	0.68	2.52	0.41
Square of GSR	2.09	1.04	2.23	.88

nervous system of the depressed women was not different from that of the non-depressed women. The independent variable was depressed versus non-depressed participants, and the dependent variables were again the various responses of the autonomic nervous system. A one way ANOVA supported the assumption that there is a difference between the amount of physical complaints between depressed and non-depressed women $F(1,14) = 14.6, p = 0.002$. The independent variable was again depressed versus

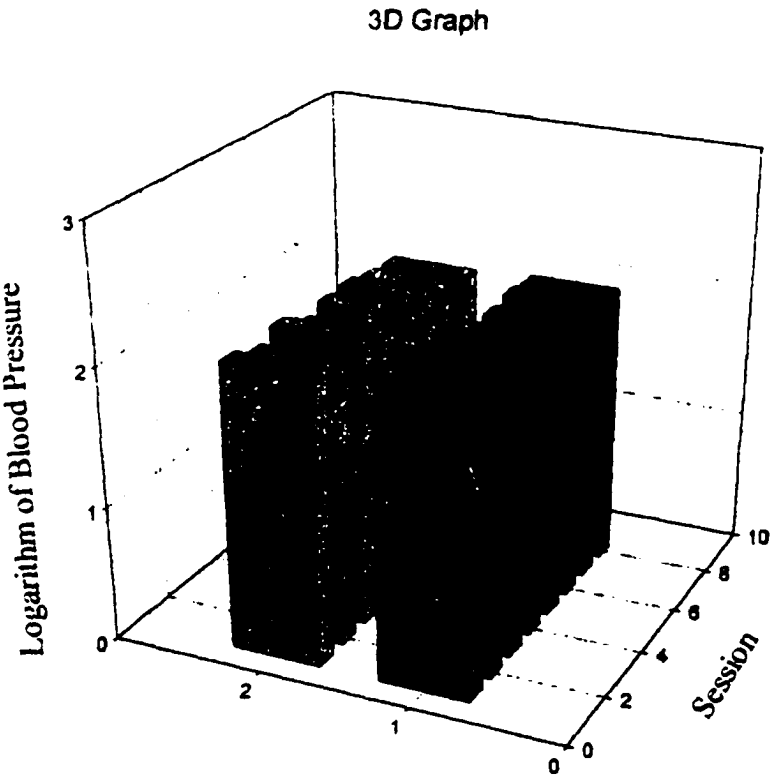
non-depressed participants, and the dependent variable was physical complaints. Figure 8 illustrates the significant difference.

The relationship between depression and physiological change (actual or perceived) is not clear. The results from the previous study indicate that the responses of the autonomic nervous system of depressed women do not differ from those of non-depressed women, nor does the homeostasis of these responses differ. Nevertheless, depressed women report more physiological



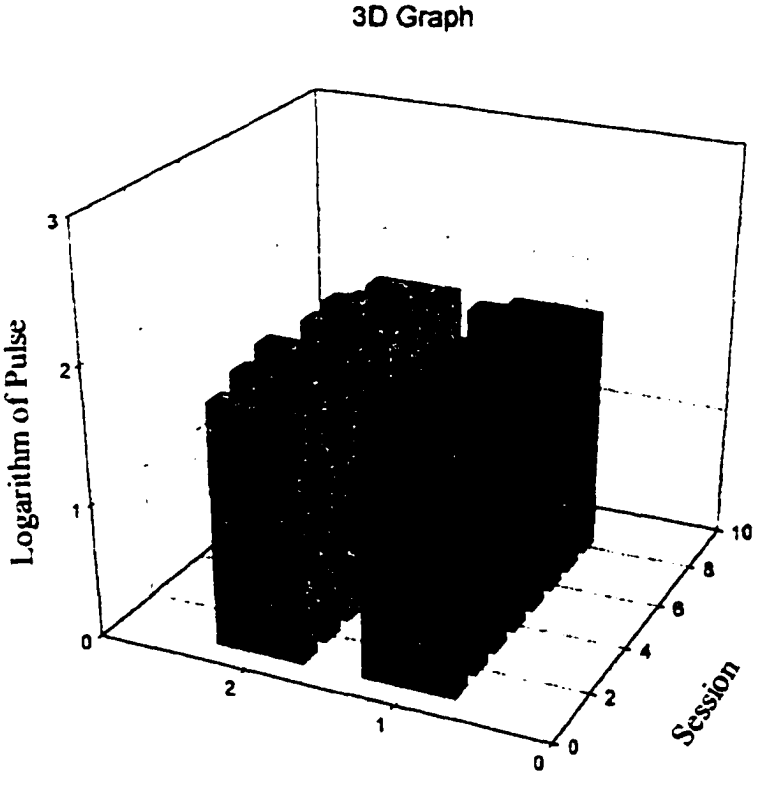
Gray columns represent the mean of the temperature values of the non-depressed women. Black columns represent the mean of the temperature values of the depressed women.

Figure 3. The Mean Temperature Values of Depressed and Non-Depressed Women.



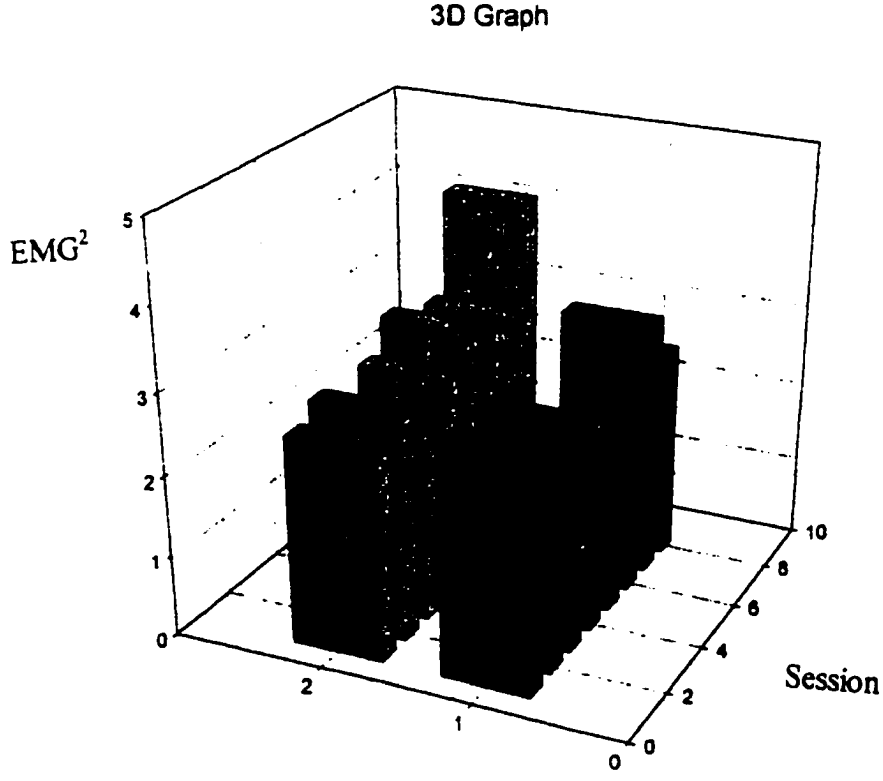
Gray columns represent the logarithm of the blood pressure of non-depressed women. Black columns represent the logarithm of the blood pressure of depressed women.

Figure 4. The Logarithm of the Blood Pressure of Depressed and Non-Depressed Women



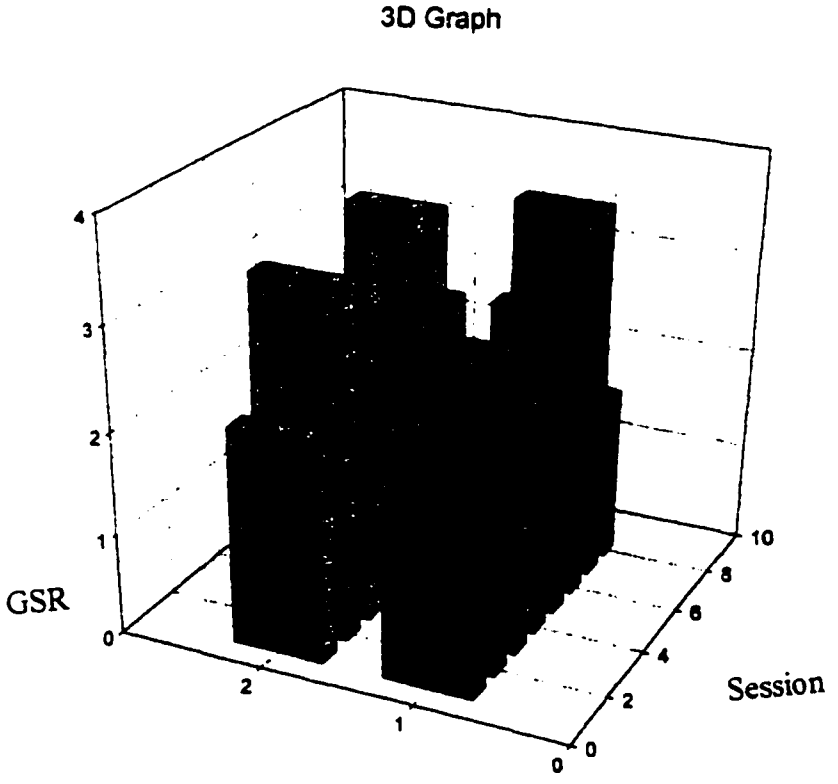
Gray columns represent the logarithm of the pulse of depressed women.
Black columns represent the logarithm of the pulse of non-depressed women.

Figure 5. The Logarithm of the Pulse of Depressed and Non-Depressed women.



Gray columns represent the squared muscle tension of non-depressed women.
Black columns represent the squared muscle tension of depressed women.

Figure 6. The Square Muscle Tension of Depressed and Non-Depressed Women.



Gray columns represent the squared gsr of non-depressed women.
Black columns represent the squared gsr of depressed women.

Figure 7. The Squared GSR of Depressed and Non-Depressed Women.

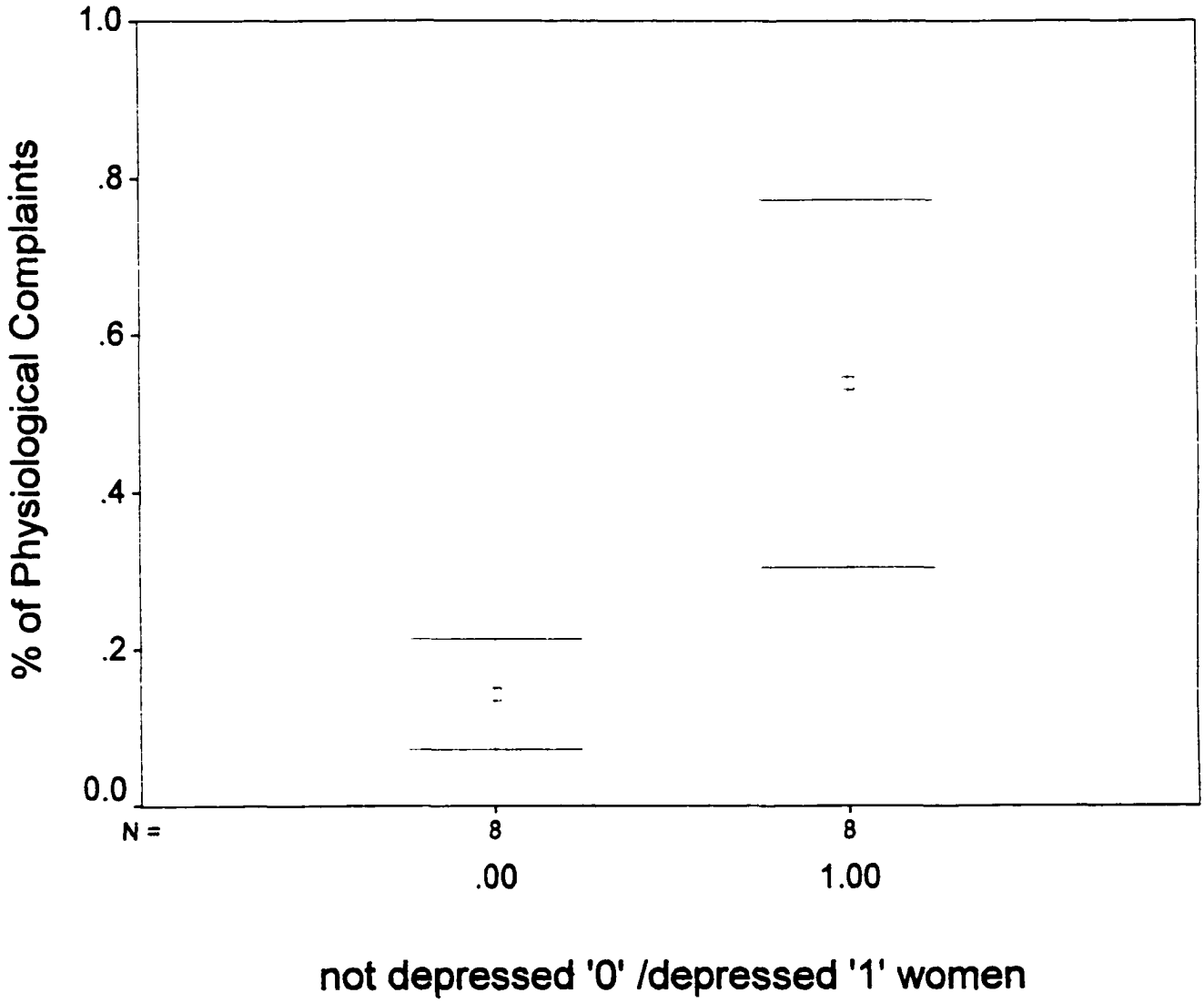


Figure 8. Physiological Complaints of Depressed and Non-Depressed Women

problems. The physiological measurements taken in the experiment do not mirror the women's reports, possibly because the measurements were not sustained for a long enough period of time. Perhaps longer periods of observation with a 24 hour monitor would reveal a difference between the responses of the autonomic nervous system in depressed and non-depressed women. Variables would be collected in a real life setting, with daily stresses impinging on the women, instead of in an artificial laboratory setting. Also, a difference in circadian rhythm could have been detected for the various variables.

Another possibility is that the responses of the depressed and non-depressed women do not in fact differ, rather only the perception, interpretation, and the way the women experience events differ. It is unclear if depression causes physiological changes or vice versa, or if a third unknown factor (for example style of processing and interpreting events) causes both depression and abnormal physiology.

It was in order to learn whether there is a causal relationship between abnormal physiological responses and depression, eight women modified the aforementioned physiological variables, while their depression was under observation. The reasoning behind this attempt was that individuals under stress tend to show abnormal physiological responses. For example, increased sympathetic outflow to vessels supplying muscle or skin will result in vasoconstriction, which results in a decrease in skin temperature (Basmajian, 1993). Since biofeedback allows individuals to modify their physiology, it may counteract the influences of excessive stress, which has been implicated in the etiology of depression (Resident Services Committee, 1988). The efficacy of biofeedback in the treatment of depression was examined

because prior research indicated that it might be helpful (Newton-John, 1995 & Peniston et al., 1986).

An Experiment to Investigate how Biofeedback Affects MDD

It was examined how EMG biofeedback assisted relaxation affects depression, physiological variables, and the homeostasis of the responses of the autonomic nervous system.

Method

Participants. A new set of 17 depressed women, 18-48 years old, was selected from the three aforementioned questionnaires. Nine were randomly assigned to an eight-week biofeedback treatment group; the remaining eight participated in a pseudo-treatment group. All participants were classified as suffering from MDD by the PRIME-MD questionnaire. The overall correlation between the other two questionnaires, the Beck's Depression Inventory and the Zung's Self-Report Depression Scale, was $r = 0.72$, $p < .05$.

One woman in the control group was excluded because she started a weight loss medication during the eight weeks of therapy, which significantly increased her pulse rate and may have changed other physiological variables as well.

Materials

The EMG biofeedback was conducted with a J&J Physiological Data System I-410 BCS. The women's temperature, GSR, and EMG were measured with the same computerized system. Heart rate and blood pressure were assessed with an AND-UA-701-digital blood pressure meter. The depression scores were obtained with the same self-report questionnaires used as in the first experiment.

Design and Procedure

During a routine check-up in a private medical practice, or in the classroom at a college introductory psychology course, young women were informed that they might be selected as participants in a study about biofeedback, depression and its physiological correlates. They were asked to fill out Beck's Depression Inventory, Zung's Self-Rating Depression Scale, and the PRIME-MD. Seventeen women scoring in the moderately to severely depressed range were randomly assigned to either an eight-week biofeedback treatment or a pseudo-treatment group. The women gave permission to have their physiological measurements taken in an informed consent form (Appendix A). Participants completed self-report questionnaires before the experiment, after four weeks of treatment, and at the conclusion of eight weeks of treatment. All physiological variables were evaluated in the same fashion used in the first experiment. Women volunteering for the eight-week biofeedback-treatment were subjected to the following treatment procedures:

- participants were seated in a comfortable chairs or recliners in a dimly lit room to allow for relaxed body posture and atmosphere,

- participants' physiological measurements were taken;
- participants were informed of how biofeedback works and how it might help them with their depression;
- electrodes were attached to participants' frontalis muscle; women were asked to wrinkle their foreheads to demonstrate computer response to incoming signals;
- volunteers were given standardized relaxation instructions;
- visual feedback of muscle tension in the frontalis muscle was displayed on a computer screen; auditory feedback was sounded whenever the tension reached a set limit, which varied with the level set for each individual;
- Figure 5 shows what the participating women viewed on the computer screen;
- participants were asked to keep tension below a set limit; and
- participants were instructed to interrupt or end the session at any time and inform the biofeedback therapist of any discomfort.

The pseudo-treatment group was also seated in the same room in the same chairs, and their physiological measurements were taken. Electrodes were attached to the frontalis muscle and these volunteers were told that they were participating in an experiment for a helpful treatment of depression. This group viewed the same computer screen graph as the biofeedback therapy group (Figure 5). However, the range of the display was set so low that it obscured the actual feedback results. Participants were also told that they could interrupt or end the session at any moment, to inform the researcher of any discomfort. The questionnaires were filled out in the same sequence as the biofeedback therapy group, at the beginning, after four weeks, and after

eight weeks of treatment. Physiological measurements were taken at each session. This was a randomized and combined between-within-subject design.

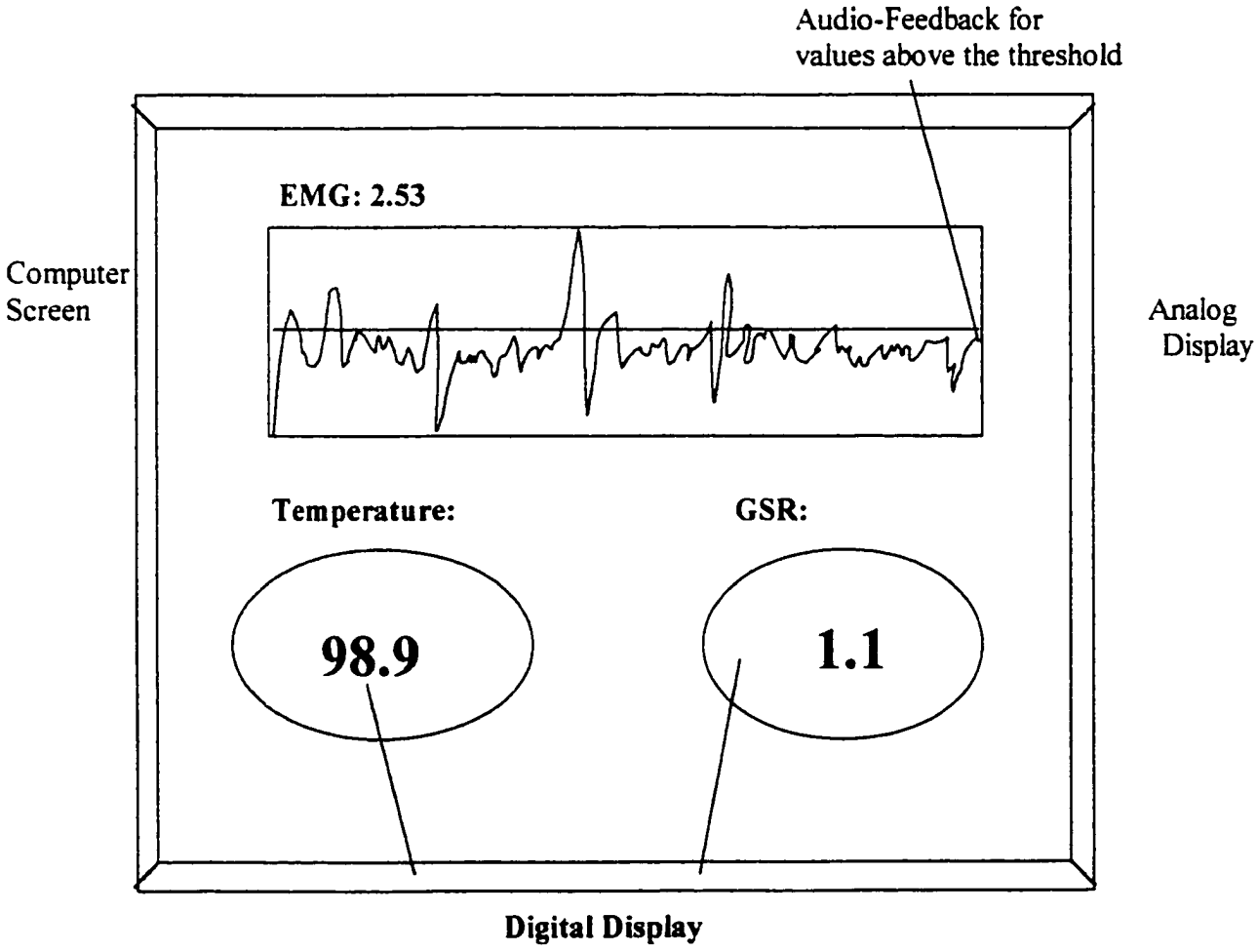


Figure 9. Visual representation on the computer screen visible to all participants

Results

The collected data were analyzed with a (2 X 2 X 3) MANOVA on the biofeedback treated and non-treated, on the two self-report questionnaires (Zung's and Beck's depression inventories), and on the three times of measurement (at the first time, after the fourth, and after the time). The depression scores of participants who received eight weeks of biofeedback therapy improved significantly in comparison with participants who did not receive the therapy $F(2,14) = 495.1$, $p = 0.000$. These patients reported less depression as early as the fourth week of treatment, but only the depression scores measured by the Zung scale differed significantly $t(8) = 2.69$, $p = 0.028$. The independent variable was eight weeks of biofeedback training, or four weeks of biofeedback versus eight weeks (four weeks) of control group. The dependent variables were the scores on the BDI and Zung questionnaires and the above mentioned physiological variables. The results are summarized in Table 4 and illustrated in Figure 10 below:

The difference in the EMG scores of biofeedback treated and those in the control group, and the EMG scores of women, who did and did not improve over the eight weeks of treatment was analyzed with a (2 X 2 X 8) ANOVA.

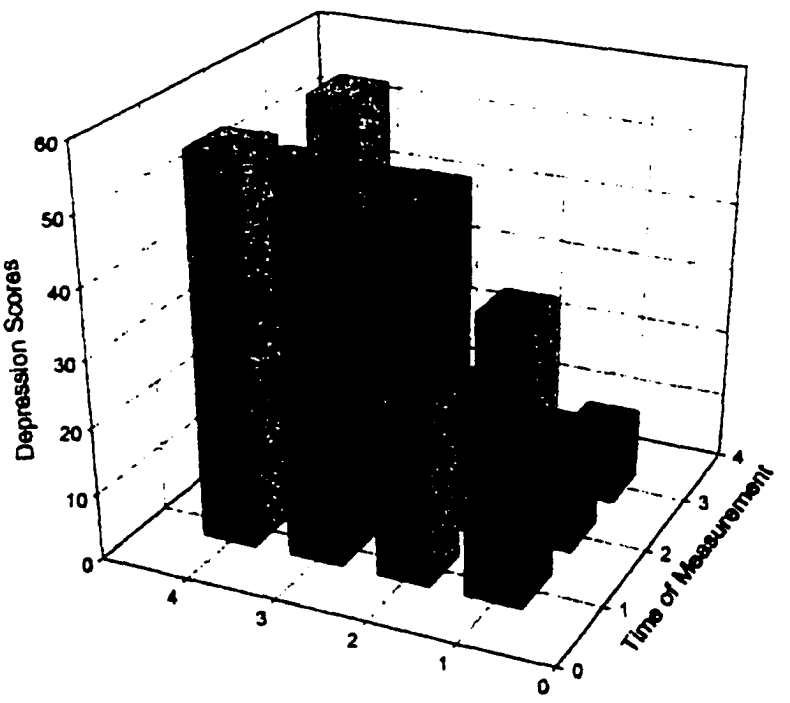
Biofeedback training significantly lowered the depressed participants' EMG $F(7,9) = 4.054$, $p = .028$. After only four weeks of training, the tension values were significantly lower than those of the control group $F(1,15) = 12.829$, $p = 0.003$. The independent variable was eight weeks (four weeks) of biofeedback training versus control group. The dependent variable

was the mean weekly EMG-scores. The EMG means of the treated and control group are graphed in Figure 11 and the descriptive statistics are shown in Table 5.

Table 4

Descriptive Statistics of the BDI and Zung Scores obtained from the Treated and Untreated Group in the Experimental Study

Time of Measurement	*Biofeedback Treated*		*Control Group*	
	Mean	*SD*	*Mean*	*SD*
	Beck's Depression Inventory			
1	23.6	6.8	26.9	7.4
4	16.4	7.3	20.5	6.0
8	10.3	8.0	25.9	11.9
	Zung's Questionnaire			
1	53.0	5.3	56.6	8.4
4	48.3	8.3	49.0	7.1
8	42.2	10.5	54.5	7.2



First column (black): Mean BDI scores of the treated group
Second column (gray): Mean BDI scores of the control group
Third column (dark gray): Mean Zung scores of the treated group
Fourth column (gray): Mean Zung scores of the control group

Figure 10. Means of the Depression Scores of the treated and Control Group obtained with the BDI and Zung Questionnaires

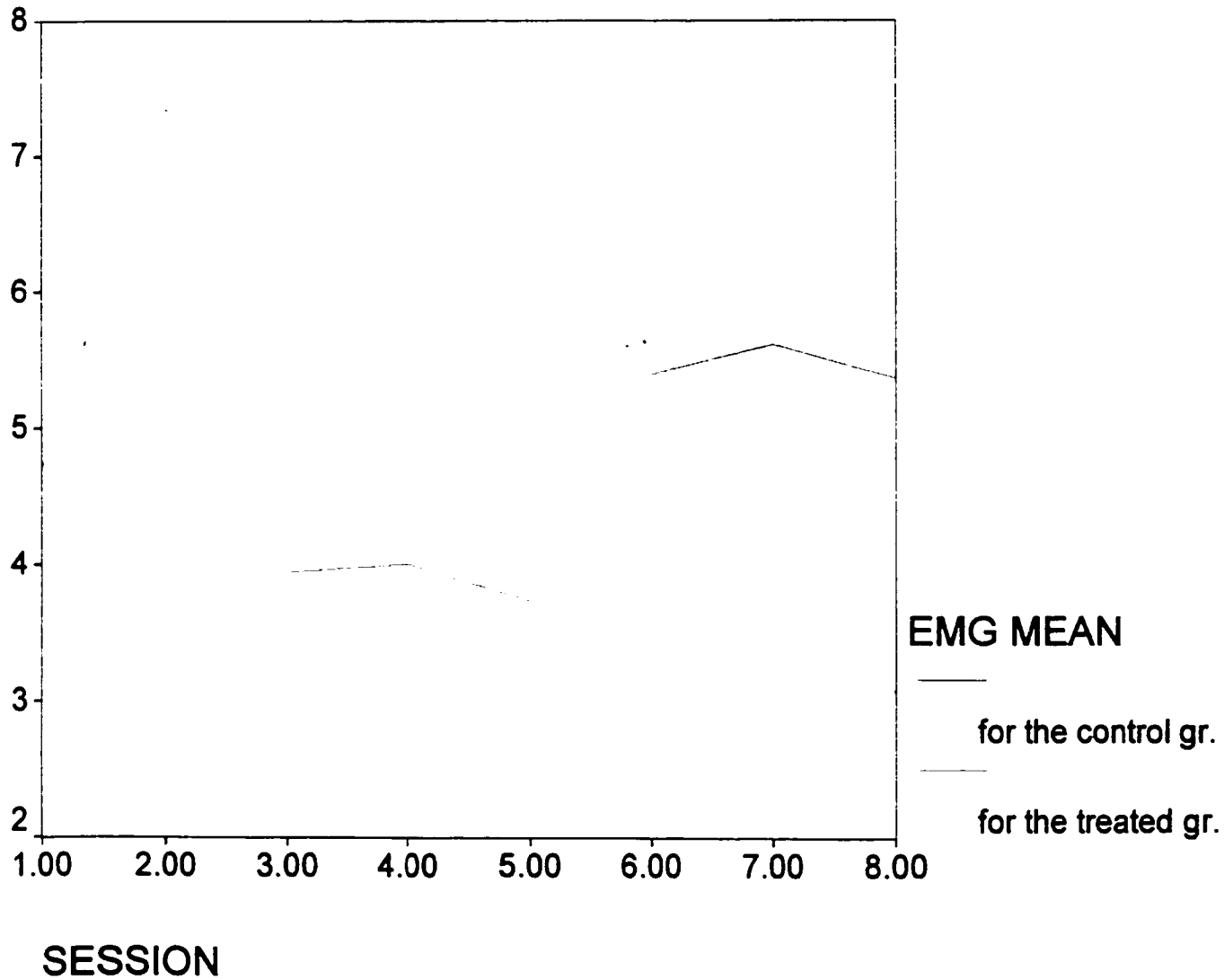


Figure 11. Means of EMG for each successive session for the EMG biofeedback and for the control group

Table 5

Descriptive Statistics of the Logarithm of EMG Means Measured from Biofeedback Treated Women and from Women in the Control Group During Eight Weeks of Treatment

Week	*Biofeedback treated Group (n =9)*		*Control Group (n=8)*	
	Mean	*SD*	*Mean*	*SD*
1	.7541	.1393	.6257	.2124
2	.6619	.2060	.8003	.2654
3	.5632	.1838	.7335	.2567
4	.5779	.1465	.7114	.1651
5	.5678	.0718	.7778	.1824
6	.5033	.1708	.6983	.1806
7	.6136	.1805	.7139	.1842
8	.4735	.1155	.6976	.1791

- The EMG values of all women in the experimental group improved significantly after four weeks of treatment. Their depression scores also improved after only four weeks. However, only the scores as measured by the Zung scale were significantly improved; the scores as measured by the BDI were significantly improved after eight weeks.

- The EMG scores of people who improved with treatment did not differ significantly from those who improved spontaneously. The independent variable was improved participants in therapy versus spontaneously improved participants. The dependent variable was EMG scores.
- Improved participants in the control group did not have a different EMG from those who did not improve. The independent variable was improved participants in the control group versus non-improved participants in the control group. The dependent variable was the EMG scores.

Furthermore, the overall difference between in the physiological measurements between the women in the biofeedback treated and in the control group was analyzed with a (2 X 5) MANOVA. There was no overall difference between the physiological variables of treated and untreated women $F(35.497) = 1.273$, $p = 0.14$. The independent variables were treatment versus no-treatment and the dependent variables were the physiological variables (EMG, GSR, Temp., BP, and P). A covariance matrix of the five variables measuring the responses of the autonomic nervous system (GSR, EMG, Temp., P, and BP) of the treated women was calculated and compared with a covariance matrix of the untreated women. The results demonstrated that the homeostasis of the responses of the autonomic nervous system of treated women did not differ from the homeostasis of untreated women. The evaluations indicated that biofeedback can influence the depressed participants' depression scores and their physiology. However, the treatment had no effect on the homeostasis of the responses of the autonomic nervous system. According, to Reda (1986), depressed patients need more than eight weeks to acquire the skills necessary to change their physiology. He treated patients in 30-60 sessions for 15-30 weeks

instead of in eight weekly sessions. After this period, the correlations of the depressed patient responses started to resemble those of non-depressed patients. Had the study run for 30 weeks, one might have observed a change in the homeostasis. The fact that the patients showed significantly lower EMG values after only four weeks of treatment, that is, before both depression scores improved significantly, implies that physiological changes were followed by emotional and cognitive adjustment.

Discussion

This investigation examined whether biofeedback is an effective treatment for major depressive disorder.

Several models have been implicated (Schwartz, 1995). The following Figures (12a and 12b) illustrate various theories for the operation of biofeedback:

The first model assumes that biofeedback changes the functioning of a target physiological system to alleviate the disease. For example, when tense muscles cause a backache, relaxing the involved muscles alleviates the pain. This model cannot explain how biofeedback treats primary depression, because there is no target physiological system, yet it could be used to explain why secondary depression improves, when biofeedback is used.

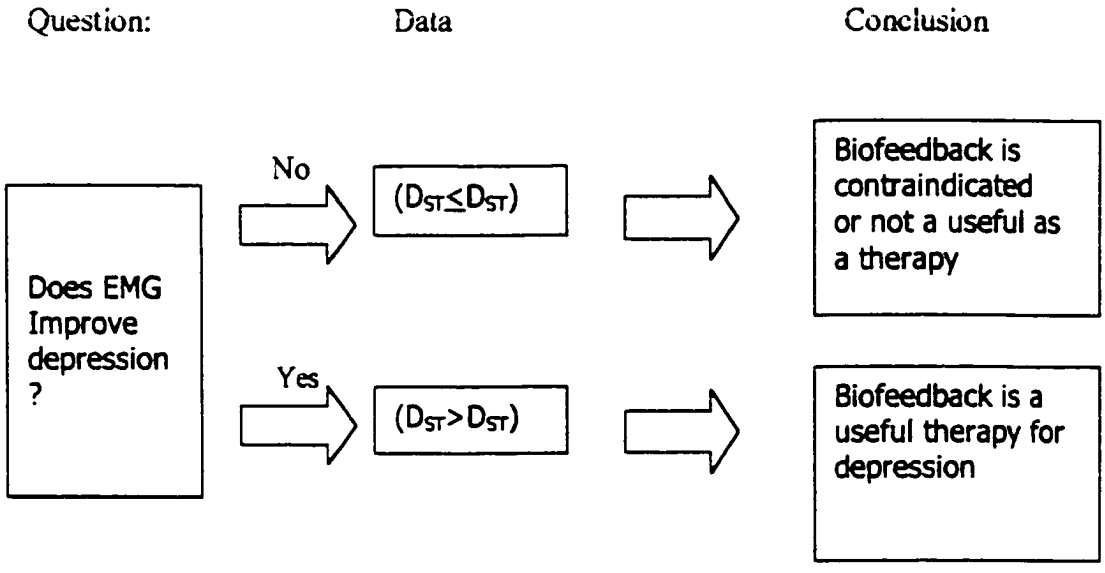


Figure 12. Decision Chart for the Effectiveness of Biofeedback and Depression

The following Figure 13 consists of part a and part b. Part a describes refuted explanations like the placebo effect, part b discusses how biofeedback might reduce depression. At part a, the first column is the question, in the second column are the refuted theories, and in the third column are the explanations. The second part of Figure 13 consists of pages 62 and 63. The first column contains the question, the second the possible four solutions, the third looks at the data, and the last describes the conclusions.

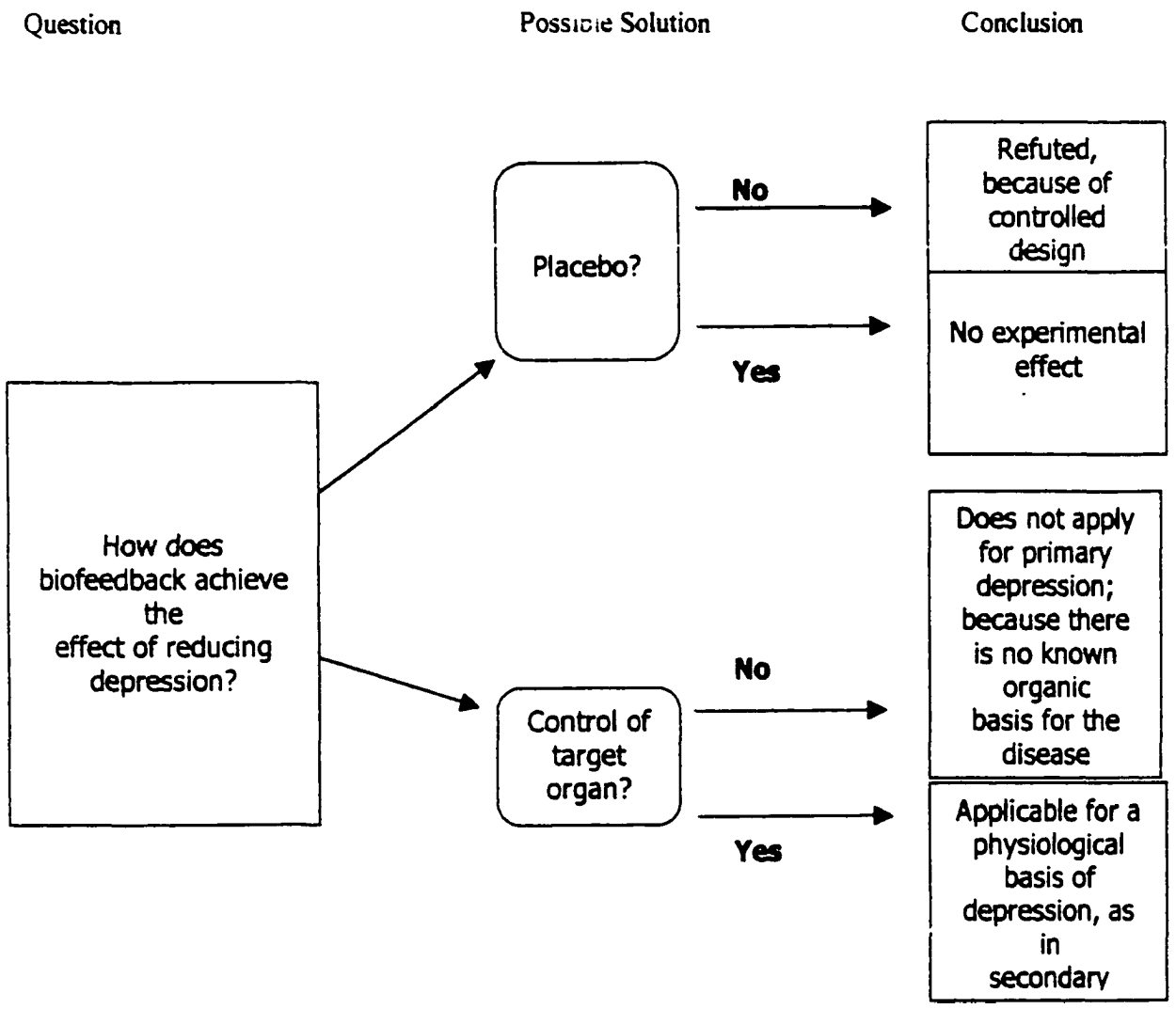
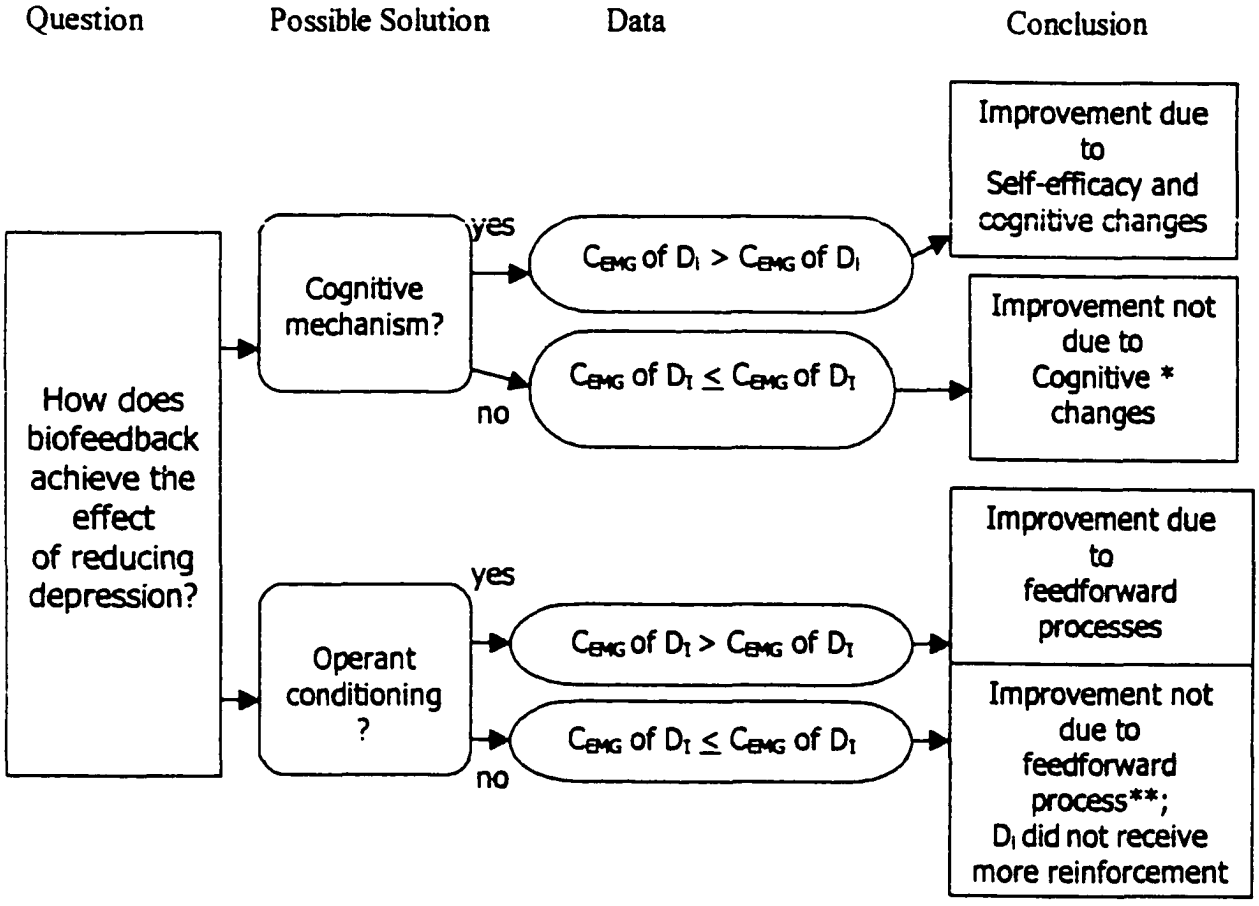


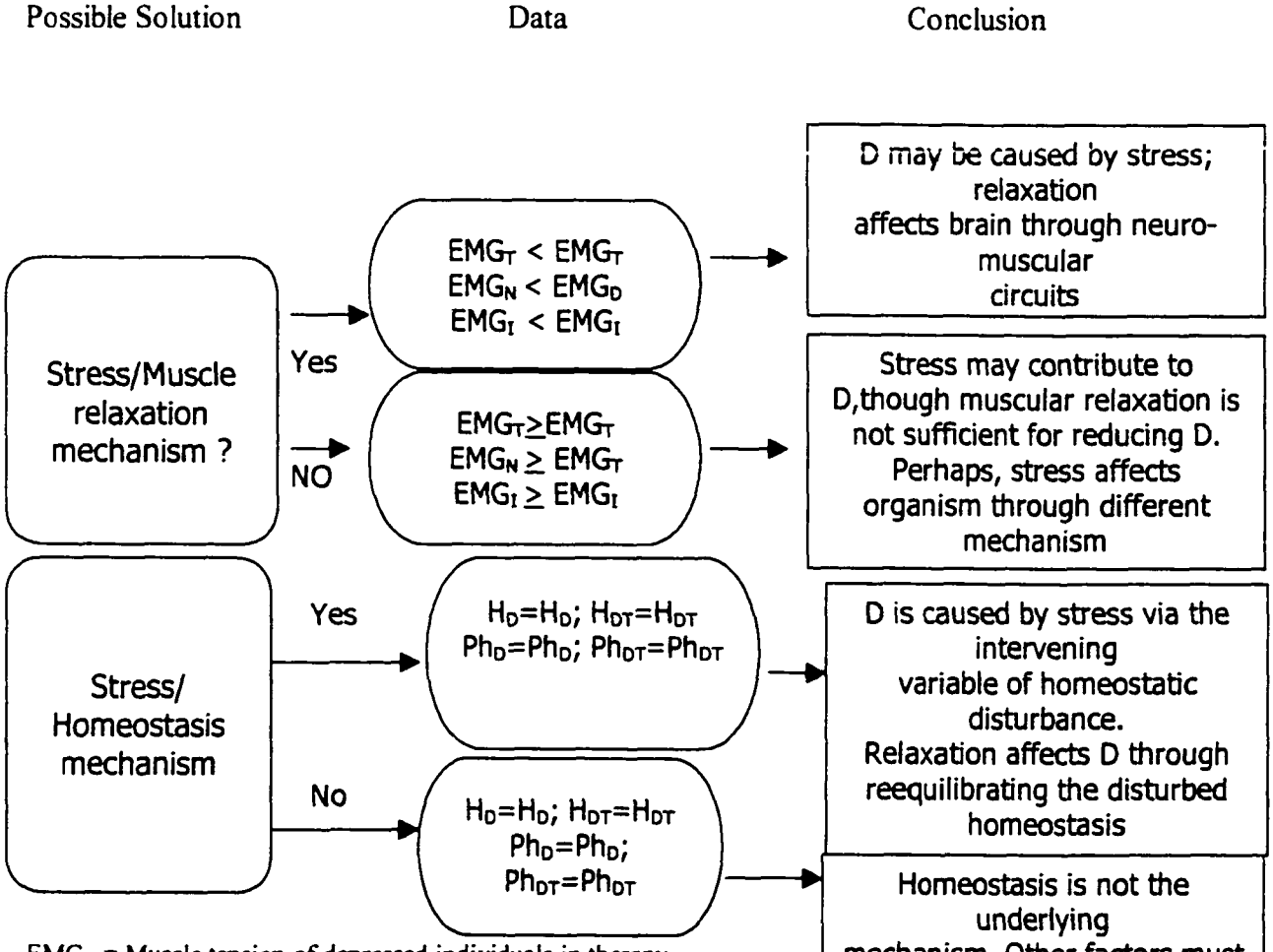
Figure 13a. Refuted Models for the 'Modus Operandi' of Biofeedback in the Treatment of Primary Depression



Note: C_{EMG} = control of muscle tension; D_I = depression scores of individuals, who improved
D_I = depression scores of individuals, who did not improve

* The cognitive changes that are thought to cause symptom changes are: experiences of mastery, positive expectations, reduced sense of helplessness, sense of empowerment over their destiny. However, future research will have to demonstrate, that this is really what depressed participants are thinking. There may be a discrepancy between what is objectively happening during biofeedback therapy and what depressed subjects may be subjectively perceiving. For example, Beck (1981), pointed out that some depressed persons tend to attribute failures to their inadequacy and successes to poor chance. The above flowchart assumes that depressed participants are objectively perceiving their success and interpreting it correctly.

** feedforward proc The feedback signal is a reinforcer for a response that a person can execute. The reinforcing feedb ill cause the response to be emitted more and more until the target physiological system . proved.



EMG_T = Muscle tension of depressed individuals in therapy
EMG_T = Muscle tension of depressed individuals, who are not in therapy
EMG_N = Muscle tension of non-depressed individuals
EMG_D = Muscle tension of depressed individuals
EMG_I = Muscle tension of depressed individuals, who improved
EMG_I = Muscle tension of depressed individuals, who did not improve
H_D = Homeostasis of depressed individuals
H_D = Homeostasis of non-depressed individuals
H_{DT} = Homeostasis of depressed individuals in therapy
H_{DT} = Homeostasis of depressed individuals not in therapy
Ph_D = Physiological variables of depressed individuals
Ph_D = Physiological variables of non-depressed individuals
Ph_{DT} = Physiological variables of depressed individuals in therapy
Ph_{DT} = Physiological variables of depressed individuals not in therapy

Figure 13b. Models for the 'Modus Operandi' of Biofeedback in the Treatment of Primary Depression

A second model states that placebo, or non-specific factors, account for the beneficial effect of biofeedback. Interestingly, the majority of symptomatic improvements in cognitive behavior therapy occur prior to the formal introduction of cognitive restructuring techniques after only four weeks of treatment. This suggests that other likely nonspecific treatment factors play a large role in mediating clinical improvement (Ilardi & Craighead, 1994). One candidate for a non-specific factor, which is present in most therapies and may be responsible for the improvement of depression, is "the treatment rationale" (Ilardi & Craighead, 1994). It leads to an "amelioration of feelings of hopelessness," which goes hand in hand with the receding of all other symptoms of depression. If non-specific factors are really responsible for the improvement of depression after the first few sessions, then the same effect should be evident in other therapies, including the treatment of depression with biofeedback.

The present experiment had a control group. If non-specific factors like reduced feelings of hopelessness and hope of recovery are the central mediational processes underlying the clinical improvement of depression, then one should observe the same amount of improvement in the experimental and in the control group, since both were offered the same treatment rationale at the outset of the experiment. However, this is not the case. The control group controlled for extraneous placebo variables and non-specific factors. Another factor that keeps the study controlled is patient ratings. Effect sizes reported by clinicians based on clinical interviews were significantly larger than those based on patient ratings (Greenberg et al., 1992). Patient ratings reveal that the treatment was having an effect. The placebo effect can thus be dismissed here.

A third model combines self-efficacy and cognitive changes and is widely accepted. A change in beliefs and in expectations results in symptom changes. The experience of being able to control symptoms of arousal increases perceived self-efficacy, self-confidence, and well being (Langosch, 1994). Positive expectations, perceived success, experiences of mastery, reduced anxiety, and a reduced sense of helplessness are necessary elements of change. Participants may have perceived that they were successfully controlling their muscle tension, giving them a sense of empowerment that may have eased their anxiety and sense of helplessness. As the body came under the control of the mind, the emotional upset and depression lessened. This study finds support for this model, since participants who were able to control their muscle tension, and received feedback that they were so doing, also showed improved depression scores later on. If the knowledge of being in control is what makes biofeedback work, one must exercise care in selecting patients for this treatment. Active attempts at mastery are most effective when events are actually controllable. When events are beyond an individual's personal control, problems may be exacerbated by persistent efforts to control (Shapiro, D. H., Schwartz, C. E., & Astin, J. A., 1996).

However, the perception that one is gaining control may not be all there is to biofeedback. A recent study tested whether perceived self-efficacy, rather than self-regulation ability, influenced the efficacy of biofeedback treatment in migraine sufferers. Self-efficacy here is defined as the extent to which a person believes that she can perform the behavior necessary for the desired outcome. The study found that perceived self-efficacy had no influence on therapy outcome. That is, bogus feedback that told participants that they

demonstrated either superior or inferior hand-warming skills produced significantly different perceived self-efficacy, but had no effect on headache activity (French, D. J., Gauthier, J. G., Roberge, C., Bouchard, S., & Nouwen, A., 1997). Nevertheless, one should not quickly dismiss the cognitive aspect of biofeedback treatment. It should be further investigated if perceived self-efficacy has a direct influence on the improvement of depression, since French's study did not consider the depression of the migraine sufferers.

A fourth model states that biofeedback may operate through muscle relaxation, which can be learned through operant conditioning. This model explains that biofeedback improves depression by lowering the tension level in treated persons through feedforward processes in which a feedback signal becomes a reinforcer for a response that a person can execute. This reinforcing feedback will cause the response to be emitted again and again until the tension level is lowered. The learned relaxation response then affects the brain through neuro-muscular circuits: the muscles are being trained to be calm and quiet in the face of stress, while the emotions are also being trained to be calm and quiet. The model is postulated on the view that mind and body are linked, and that the central nervous system (CNS) is undergoing training during biofeedback.

At first, one perceives the events as outside oneself; one then experiences an emotional and mental response to these events, the brain then responds and fourthly, one experiences a physiological response which is perceived via biofeedback; one then produces an emotional response to the perception of the physiological response, which is again picked up by the brain, which again leads to a renewed physiological response etc., etc., etc..The outcome is that a

response to a sensory perception becomes more and more controlled (Green & Green, 1986). This model implies that depressed women are more tense than non-depressed women, that women whose depressions improve are less tense than before the amelioration, and thirdly, that they should become less tense whether the depression disappeared spontaneously or as a result of biofeedback.

The statistical analysis of the data collected in this experiment shows that depressed women in general are not more tense than non-depressed women, nor are the tension levels of improved (including the spontaneously improved) women significantly different from those who did not improve. However, the tension levels of treated women are significantly lower than those of non-treated women; that is, only when the alleviation of the depression was brought about through EMG-biofeedback were the tension levels significantly lowered. Otherwise, in women who improved spontaneously, the tension levels did not change significantly. Thus depression may have improved as suggested by the third and fourth models. Relaxation alone does not seem to be the only mechanism responsible for lifting depression, since some women recovered spontaneously without a change in their tension levels.

Now that the question of if and how biofeedback alleviates depression has been discussed, the remaining step is how to integrate relaxation, or biofeedback therapy into the treatment of depression.

CHOICE OF TREATMENT—THE INTEGRATIVE MODEL

According to a meta-analysis (Dobson, 1989), cognitive therapy seems to be very effective, although it does have its limitations in the treatment of depression. The question arises whether all therapies are equally effective in the treatment of patients with different severity levels of depression. The following therapies were compared: psychotherapy, relaxation training, behaviour therapy, and pharmacotherapy.

A secondary analysis, in which treatment by severity interactions were investigated, revealed that no treatment was differentially effective in improving more severely depressed patients. Between treatment groups there was little difference across severity levels in the proportions of recovered patients. Regardless of the type of treatment, most patients displayed either a recovery or nonremission outcome profile, with a few instances of remission followed by a recurrence of depression. None of the treatments were associated with a higher incidence of relapse; but severe episodes of depression and greater neuroticism were associated with nonremission regardless of the type of therapy. In summary, all therapies were effective for most people even for severely depressed patients. However, neurotic patients did not do well, no matter the type of therapy they experienced (Mc Lean & Taylor, 1992, 1993). It thus remains to be investigated why all therapies were effective even though each treatment rationale is based on very different theories. There may be two reasons: first, perhaps certain symptoms respond best to certain therapies, or to a combination of existing therapies (Karasu, 1990). Thus the best treatment for a patient may require a combination of different successive or simultaneous treatment strategies. Second, the optimum effect of different strategies is

achieved when they are introduced at different points in time. For example, at the outset drugs could be the method of choice, because they affect symptom formation and affective distress, take effect sooner, and last for shorter periods; severe suicidal patients may be recommended for electroconvulsive therapy (ECT). Relaxation, biofeedback, and acupuncture therapies may then be introduced to prepare the patient for psychotherapy. Hence, psychotherapy could be used in conjunction with drugs at the beginning of treatment or later on. Since it influences interpersonal relations and social adjustment, results may not be seen until a few weeks into treatment.

This combination of different types of therapy may produce an enhanced method of treatment and reduce the number of patients who show symptom relapse. This notion is supported by the findings illustrated in the figure below:

Each type of therapy targets one particular aspect of the patient that is considered pivotal for his or her remission. For example, in cognitive-behavioral therapy, the aspect of cognition is considered, while during pharmacotherapy, the patient's psychobiology is treated. A more powerful method of therapy should emerge if the different approaches are combined into one integrative model that considers different aspects of the disease. This may lead to a more effective way to treat depression.

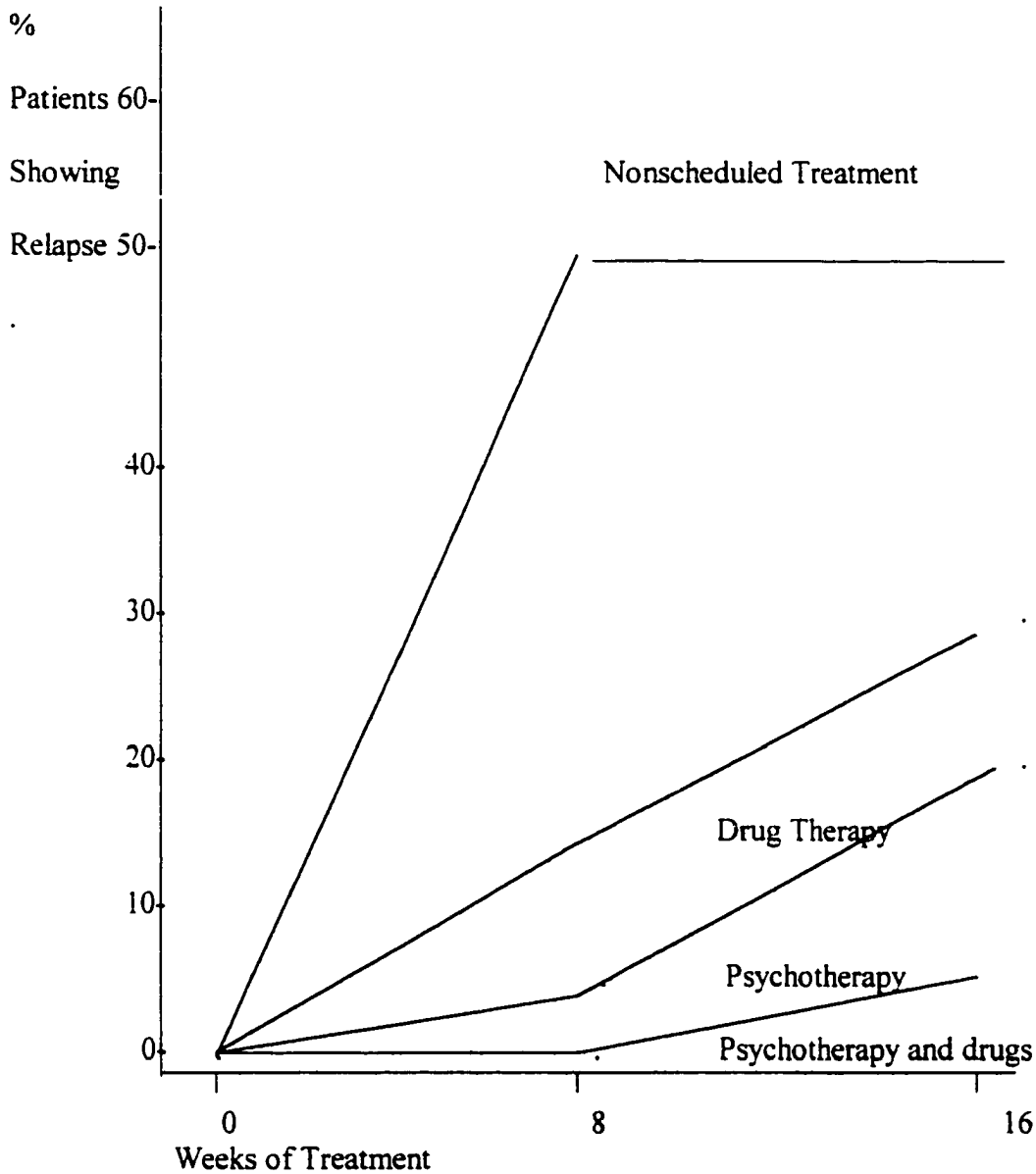


Figure 14. Effectiveness of Various Treatments for Depression; Depression Symptom Relapse

APPENDIX A

Consent Form

Agreement to Participate

Please read the following material about this study. Signing this form indicates that you have read this form and you have chosen to participate in a free and informed manner.

What kind of study is this?

This is a psychological study to observe the physiological correlates of depression. You are being asked to participate in this study because you may be suffering from depression. This study will show if your physiological variables, such as your temperature, heart rate, blood pressure, GSR, and EMG are altered during depression.

What will I have to do, if I decide to participate?

If you agree to participate, you will fill out two psychological questionnaires, and your temperature, heart rate, blood pressure, GSR, and EMG will be measured. If your psychological evaluation indicates that you are indeed depressed you may volunteer for a second study. You will undergo the same procedures as in the first study, that includes the filling out of questionnaires, and having physiological variables measured. You will be invited to come to the behavior modification laboratory twice a week for eight weeks and spend some time in a quiet room sitting comfortably for about 30 minutes. While relaxing you will observe and listen to a computer.

What am I going to get out of this if I agree to participate in the study?

There is no monetary compensation for your participation in these studies, though you will be acknowledged for helping further the knowledge in the science of psychology. If you participate in the first study you will get partial credit toward your course requirement. Since the second study is not connected to any course work there will be no extra credit for participation in it.

Will there be any additional costs to me if I participate?

There will be no monetary costs to you if you participate.

Will my name be given to anyone or will any information identify me by name?

All information regarding your participation will be kept confidential.

Is there anything that could happen to me during the study/ies?

There are no known negative side effects to the procedures performed in this study.

Can I change my mind even after I agree to be in the study/ies?

You are free to withdraw from the study at any time. Your decision not to be in this study will not influence your grade and will not result in any penalty.

I will answer any questions you may have now or at a later point in time.

In giving my consent by signing this form, I agree that these studies have been explained to me, and my questions have been answered, I understand, that this form will be filed, and that I will receive a copy of this form upon request.

Signature: _____

Name printed: _____

Thank you for your participation

Sincerely,

Heli Apelbaum

(Heli Apelba m)

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