CLINICAL DEPRESSION AND DAILY PHYSICAL ACTIVITY LEVEL

BY

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Abstract

Depression has been linked to a number of health problems, including cardiovascular disease (CVD). One link may be depression-related decreases in physical activity. This study was designed to examine physical activity in clinically depressed and never-depressed individuals. Depressed participants were predicted to be less physically active and to exhibit more restlessness than nondepressed individuals. Participants were 20 clinically depressed and 37 never-depressed undergraduates (mean age = 19.7, SD = 2.30). Participants were screened for depressive symptomatology and diagnostic status was confirmed by structured clinical interview. Participants wore an actigraph for 48 hours. A 2 (depressed vs. nondepressed) x 2 (male vs. female) between person ANCOVA was used to test the hypothesis. Depressed individuals were found to significantly exhibit restless behavior; however, they were not significantly less active. Despite the findings, treatment for depression should incorporate physical activity to promote health and reduce CVD risk. Additionally, treatment should encourage relaxation therapies.

Clinical Depression and Daily Activity Level

Approximately 10% of the population suffers from clinical depression at any point in time (Robins & Regier, 1990), with women being twice as likely as men to be depressed (Blehar & Oren, 1999). Furthermore, over the course of a lifetime, approximately 17% of adults will suffer at least one depressive episode (APA, 2000). Depression is the leading cause of disability in the United States (U.S.), with associated annual costs greater than $30 billion (NIMH, 1999). Depressive disorders are considered to be a significant public health concern in that they meet the following criteria: they occur frequently in the population, they cause significant distress and debilitation, effective treatments/interventions exist, and their prevalence is increasing (Sartorius, 2001). Depression is also a public health concern due to its comorbidity with other debilitating illnesses like cardiovascular disease (CVD) (e.g., Musselman, Evans, & Nemeroff, 1998).

Along with depressed and anhedonic mood (APA, 2000), people with depression have been found to have a lower quality of life (Ravindran et al., 2002), more interpersonal and occupational difficulties (Goeth & Fischer, 1995), more physical pain (Trivedi, 2006), and more health problems in general (Murray & Lopez, 1997). The sequelae of depression often include symptoms of anxiety, alcohol abuse, and insomnia (Fawcett, 1993). Additionally, it is suspected that the mortality rate of clinically depressed individuals is quite high due to both direct (i.e., suicidality) (e.g., Fawcett, 1993; Sartorius, 2001) and indirect causes, like death from CVD (e.g., Musselman et al., 1998).

Depression is a complex disorder, with dysfunction at biological, psychological, social, and behavioral levels of analysis. A number of biological theories have been proposed connecting depressive symptoms and CVD. Most of these theories include dysregulation of the hypothalamic-pituitary adrenocortical (HPA) axis (Bornstein, Schuppenies, Wong, & Licinio, 2006; Musselman et al., 1998), which causes hypercortisolemia and an overabundance of pro-inflammatory cytokines. These molecular level changes can impact all levels of an individual's functioning, both physical (cardiovascular problems) and emotional (depressive symptomatology).

Considering a biopsychosocial approach to illness, the mind and body are not separate entities. Consequently, biological mechanisms interact with thoughts, emotions, and behaviors. The cognitive model states that clinical depression is characterized by negative views of the self, the environment, and the future (Beck, 1976), and the presence of dysfunctional schemas (Stein & Young, 1992). More specifically, the cognitive vulnerability theory of depression (Ingram, Miranda, & Segal, 1998) takes specific environmental and interpersonal antecedents into account. Maladaptive early attachments, negative self-schemas that influence information processing, and the kindling of these factors all have an effect on depressive symptomatology across the lifespan (Ingram et al., 1998). It is implicit that these cognitive states operate concurrently and cyclically with neurophysiological aspects. For example, helpless, hopeless, and hypervigilant thinking may be related to the activities of the serotonin system (Weissenburger & Rush, 1996) and the activation of

stress-hormones (Charlton & Ferrier, 1989). Moreover, these cognitive and psychophysiological factors may lead to changes in behavior, most notably suicide. Cognitive factors including hopelessness and dysfunctional attitudes have been tied to suicidal ideation/attempts, as have increased blood serum cortisol levels and lower levels of dopamine and serotonin (Weissenburger & Rush, 1996). Similar causal chains may also help to explain the link between depression and other health risks, most notably CVD.

Depression and CVD

A number of studies have shown a link between depression and CVD. Upwards of 20% of the U.S. population is afflicted with CVD (i.e., hypertension, coronary heart disease, stroke, rheumatic heart disease) and this is the single leading cause of death in the U.S. (Morbidity Mortality Weekly Report, 1998). Depression is not only a risk factor for developing CVD (e.g., Ferketick, Schwartzbaum, Frid, & Moeschberger, 2000), but also increases the CVD-related mortality risk (Wulsin, Vaillant, & Wells, 1999). Many recent studies examining the relationship between depression and CVD have found that patients exhibiting depressive symptoms have substantially increased cardiovascular morbidity and mortality (Musselman et al., 1998). For instance, in a prospective analysis of individuals (who were initially free of CVD) participating in a national health study, those who initially reported elevated levels of depressed symptoms were at an increased risk of experiencing a cardiac event over the next four years (Rowan, Haas, Campbell, MacLean, & Davidson, 2005).

The association between depression and CVD is clearly one of great importance, and a greater understanding of the relationship is crucial. However, results of meta-analyses assessing this association have been unable to determine causality or possible mechanisms of action. Furthermore, the directionality of the relationship is not as well-understood as the biomedical and behavioral medicine community would deem adequate. One meta-analysis showed that depressive symptoms had a strong positive relationship with cardiac and total mortality in patients with CVD (Barth, Schumacher, & Herrmann-Lingen, 2004). This study determined that the risk of mortality was at least two times higher in the short- and medium-term for CVD patients suffering from clinical depression. The researchers concluded that depression should be considered a significant risk factor in patients with CVD (akin to smoking and a high-fat diet). Another review (Wulsin et al., 1999) established a sizeable effect of depression on mortality in some populations; however, this review stated that it was impossible to determine the underlying mechanism or the directionality of the relationship. Noting these studies as examples, it is clear that there is a need for more well-controlled studies designed to examine the mechanisms linking depression to CVD.

Mechanisms possibly linking depression to CVD

Depression could be linked with CVD via a number of biopsychosocial pathways. One common biological factor linked to both depression and CVD is obesity. Obesity is a well-known risk factor for CVD (Roberts, Deleger, Strawbridge, & Kaplan, 2003). A recent study found that clinically depressed adults had higher

body mass index (BMI) than age/gender matched nondepressed individuals (Dragan & Akhtar-Danesh, 2007). Moreover, this relationship was gender dependent in the sense that depressed men had a higher BMI than nondepressed men, but the relationship between depression and higher BMI was not statistically significant in women. Examinations of the relationship between depression and CVD biopsychosocially must also take into account micro-level changes that occur in both disorders. Hyperactivation of the HPA axis, which often occurs in depression and in overweight individuals, results in elevated blood levels of corticosteroids and has been demonstrated to induce hypertension and damaging effects on vascular endothelial cells (Musselman et al., 1998). Thus, the link between depression and CVD might be mediated via obesity or the behaviors and physiological reactions that lead to and promote obesity.

Activity Level

At a behavioral level, changes in activity levels may link depression to obesity and, ultimately, CVD. In recent years, there has been a great deal of focus on the link between physical activity and depression. Prior to a discussion of this relationship, it is important to note that the diagnostic criteria of depression identify changes in physical activity (i.e., psychomotor agitation/retardation) as a symptom of clinical depression (APA, 2000). Thus, changes in motor activity may be inherent in some individuals experiencing depression.

Epidemiological studies typically show an inverse relationship between depressive symptomatology and activity levels (e.g., Phillips, Kiernan, & King,

2003). For example, a large-scale, longitudinal study found that individuals low in physical activity were two times more likely to show worsening levels of depression symptoms than their highly active counterparts (Farmer et al., 1988). More specifically, participants at time one with moderate to high levels of depressive symptoms (as measured by the Center for Epidemiological Studies Depression Scale) and little/no physical activity, were much more likely to report depressive symptoms at follow-up than those with moderate to high baseline levels of depressive symptoms and much/moderate physical activity. Another longitudinal study using the Alameda County data determined that low activity levels predicted development of depressive symptoms nine years later; however, this relationship was non-significant after adjusting for covariates (e.g., physical health and socioeconomic status) (Camacho et al., 1991). Not surprisingly, these studies seem to indicate that there is a bi-directional relationship between depression and activity levels.

Although epidemiological studies are important, most rely on self-report data, which are vulnerable to self-presentation bias, and many operationalize depression based on symptom reports rather than diagnostic interviews. Furthermore, most of these studies do not take into account the full range of physical activity. Even though it is typically thought that at least moderate aerobic exercise is necessary to promote fitness and prevent CVD, recent research has shown that even low level gross motor activity (e.g., fidgeting and activities of daily living) may have significant metabolic implications (Levine, Schleusner, & Jensen, 2000). In other words, if general activity

level significantly influences metabolic processes, then perhaps low levels of gross motor activity could have a cumulative effect on mood as well.

Theoretical Background

As previously noted, the relationship between depression and physical activity may have less to do with formal exercise regimens than it does with other forms of physical activity. Physical activity is comprised of many different behaviors and physiological components; however, the end result of physical activity is energy expenditure. There are three principle components of total daily energy expenditure (TDEE): basal metabolic rate, the thermic effect of food, and activity thermogenesis (Levine, 2004). Activity thermogenesis comprises both exercise-related activity thermogenesis and non-exercise activity thermogenesis (NEAT) (Levine et al., 2000). NEAT (the energy expended due to such daily activities as working, sitting, shopping, fidgeting, etc.) accounts for ~15% of TDEE in extremely sedentary individuals, and up to ~50% of TDEE in very active individuals (Levine, 2003). Therefore, NEAT often accounts for a large portion of an individual's energy expenditure. Exercise and non-exercise activity can be measured using actigraphy, a non-invasive technique that has been validated for use in a variety of populations (e.g., Patterson et al., 1993).

A study using actigraphy to predict depressive symptomatology in a sample of non-psychiatric adults (mean age = 50.00, SD = 23.97, men = 41 %, women = 59%), found that lower daytime activity level was the best predictor of observed depressed mood (Mendlowicz et al., 1999). Although interesting, it is difficult to exclude third-

variable causes for this relationship. Biopsychosocially, there are numerous variables that interact with one another. For instance, activity differences in older depressed people may be caused by confounding health problems including CVD and obesity. To our knowledge, there has not been a study of this nature that compares actively depressed and never-depressed young adults. It would be important to determine whether differences in activity level are present in younger people, prior to onset of the types of physical health problems that might lead to depression and also impair physical activity.

Depression may also have other, counterintuitive, relationships with activity level. High comorbidity of anxiety and/or psychomotor agitation may mean that although depressed individuals are less active *overall*, they may be more restless and thus exhibit fewer periods of idleness (i.e., brief periods of inactivity). Times of stillness (e.g., relaxation) have been found to be integral to both mental and physical health (Ernst, Rand, & Stevinson, 1998).

Summary

Depression is posited to be a biopsychosocial disorder with disruption occurring at the biochemical (with neurochemicals like serotonin and hormones like cortisol), psychological (cognitive disturbances), and sociological levels (influencing various behaviors like diet and exercise). Inactivity may also be viewed as a biopsychosocial phenomenon. Activity levels may be influenced by individuals' thought processes and mood which ultimately have an effect on their behavior. This preliminary study is not positing directionality; it is simply emphasizing the

complicated and multilevel relationships between depressive symptoms and physical activity. In sum, this model posits a biopsychosocial theory of depression and physical activity and their possible subsequent effects on physical health.

Purpose of the Study

The present study is a preliminary investigation examining the cross-sectional relationship between depression and daily levels of physical activity. This study was designed to examine an index of gross motor physical activity and periods of inactivity in clinically depressed and never-depressed young adults, using an objective measure of physical activity. It was predicted that currently clinically depressed young adults would be less physically active than nondepressed young adults. Conversely, participants exhibiting psychomotor agitation would have fewer discreet periods of inactivity/rest.

Methods

Participants

Participants were 20 clinically depressed (75.0% female) and 37 never-depressed (43.2% female) university undergraduates, mean age 19.7 years (SD = 2.30). The majority were Caucasian (87.7%). Participants' average BMI was 23.9 (SD = 4.46). BMI was calculated by the following formula: weight in kilograms / height in meters^2. See Table 1 for descriptive characteristics of depressed and nondepressed participants.

Apparatus

Actigraph

Physical activity was measured using a portable electronic actigraph (Actiwatch-Score). Actiwatches are small, sturdy, wrist-worn data loggers. These instruments record a digitally integrated calculation of an individual's physical activity. Actiwatches are equipped with a highly sensitive, 360 degree accelerometer that records all motor movement. The actigraph has been shown to provide a relatively inexpensive and reliable measurement of gross motor activity (Teicher, 1995). Actigraph data have been found to accurately differentiate between physical and sedentary activities and are highly correlated with oxygen uptake, heart rate, and participant diaries of activity (Patterson et al., 1993). These relationships are substantiated with both laboratory and field findings.

In the present study, overall physical activity was operationalized by the 'Total Activity Score' as determined by the actigraph algorithm. Periods of rest were operationalized as 'Immobility Phases of 1-minute, Percentage' as calculated by the actigraph algorithm. The total activity index (see Figure 1A) is determined by summing all activity counts between the start (wake) and end (bed) times. To increase reliable estimation of wake and bed times, study participants noted in a diary at what time they woke-up and what time they went to bed. These markers were used to identify wakeful periods, including total activity index and percentage of brief periods of immobility. The percentage of brief immobility phases of one-minute (see Figure 1B) is the number of one-minute increments in which there is no motion in relation to

the individual's total number of immobile phases. This index is determined by dividing the immobility phases of one-minute by the total number of immobile phases and multiplying by 100. Practically speaking, this index can be viewed as an indicator of restlessness. Lower values equal more restlessness in an individual, and higher values equate to more restful behavior (Bruck, Kennedy, Cooper, & Apel, 2005).

Measures

Beck Depression Inventory (BDI)

The BDI (Beck et al., 1961) is a self-administered, 21-item self-report measure used to assess depressive symptomatology (see Appendix A). Participants indicate to what degree certain depression characteristics apply to them (e.g., "I do not feel sad; I feel sad much of the time; I am sad all the time; I am so sad or unhappy that I can't stand it"). Internal consistency ranges from .73 to .92 with a mean of .86 (Beck, Steer, & Garbin, 1988). The BDI has a high internal consistency [α = .86 (psychiatric population), α = .81 (non-psychiatric population)] (Beck et al., 1988). Test-retest reliabilities range from .48 to .86, and are dependant upon the interval between re-testing and the nature of the population (Groth-Marnat, 1990).

Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-IV)

The SCID-IV (First, Spitzer, Gibbon et al., 1997) is a semi-structured interview used to make DSM-IV diagnoses and is designed to be administered by a clinician or trained professional. The interview takes into account all DSM-IV criteria for specific disorders and, through a series of questions and eliminations, accurate clinical diagnoses can be made.

Inventory to Diagnose Depression, Lifetime version (IDDL)

The IDDL (Zimmerman & Coryell, 1987) is a self-administered, 22-item self-report measure used to diagnose a lifetime history of major depression (see Appendix B). The IDDL has good internal consistency (α = .92), split-half reliability (Spearman-Brown coefficient = 0.90), and significant item-total correlations. Using the Diagnostic Interview Schedule as a criterion measure, the sensitivity of the IDDL is 74% and its specificity is 93%.

Procedure

Participants were selected from a pool of undergraduates enrolled in Introduction to Psychology at the University of Kansas. Almost 4,500 students completed the online prescreen and 2,080 were eligible to participate. Of these, 133 agreed to complete a screening packet in the laboratory. This packet included the BDI. Participants with BDI scores greater than 12 (likely to be depressed) and those with scores ranging from 1-5 (likely not to be depressed) were invited for further screening. Participants selected using these pre-screening criteria were contacted and invited to participate in a larger study.

Participants were then assessed using the SCID-IV and the IDDL. Participants were considered eligible for the study if they were either currently depressed or had never been depressed. Sixty-one of these participants were not eligible for further study due to possible confounding factors like a history of depression or the presence of manic symptoms (bipolar disorder). Nine participants withdrew, leaving 62 participants who completed the study. These qualified participants were fitted with a

portable electronic actigraph and were requested to return to the lab in 48 hours for further evaluation. Of these, five were excluded from analysis due to insufficient activity data. Participants were weighed and measured in the laboratory in order to determine BMI. The final sample included 20 clinically depressed individuals and 37 never-depressed individuals.

Data Analysis

All analyses followed a similar format. A series of 2 (depressed vs. nondepressed) x 2 (male vs. female) between-person analyses of covariance (ANCOVA) were used to test all study hypotheses. Prior to analyses, a correlation matrix was used to identify possible demographic covariates of activity level. Thus, all analyses covaried for lifetime depression status and the use of stimulant medications (methylphenidate and dextroamphetamine). See Table 2 for correlation matrix outcomes.

Results

A 2 (depressed vs. never-depressed) x 2 (male vs. female) between-person ANCOVA, covarying for depression history and use of stimulant medication, was used to test the hypothesis that clinical depression was associated with reduced activity levels. The results were inconsistent with the hypothesis that currently depressed young adults (M = 380350, SD = 150207) were less active than nondepressed young adults (M = 356835, SD = 139642), $F(1, 57) = 0.435$, $p = .512$, partial $Eta^2 = .008$. There were no main effects of depression status or gender. Nor

was there a gender x depression interaction. Thus, there were no discernable effects of depression (or gender) on overall activity level.

A 2 (depressed vs. never-depressed) x 2 (male vs. female) between-person ANCOVA, covarying for depression history and use of stimulant medication, was used to test the hypothesis that depressed individuals would spend less time immobile. Depressed participants had significantly fewer periods of rest (percent of time in which there is no motion in one-minute increments) (M = 18.43, SD = 4.28; Never depressed M = 19.61, SD = .4.57) than never-depressed individuals [$F(1,57) = 8.40$, $p < .006$, partial $Eta^2 = .072$] (see Figure 2). In addition, females (M = 20.46, SE = .790) spent more time briefly immobile than males (M = 17.69, SE = .865), $F(1,57) = 12.44$, $p < .001$, partial $Eta^2 = .196$ (see Figure 2). There was a marginally significant interaction between gender and depression status, $F(1,57) = 3.96$, $p = .052$, partial $Eta^2 = .072$.

Discussion

Results of this study indicated that depressed individuals were significantly more likely to exhibit restless behavior; however, they were not significantly less active than their nondepressed counterparts. Although previous research has found low activity levels in depressed individuals, none to our knowledge have used objective measures of activity in non-hospitalized, young, depressed individuals. Furthermore, there have been no studies to our knowledge that have attempted to separate moderate daily activity from restlessness.

It is noteworthy that the gender x depression interaction was marginally significant. Examination of the means clearly shows that men who are depressed tend to have more restlessness than nondepressed men or women (both depressed and nondepressed). It is possible that with more depressed males in the sample that this interaction would have been statistically significant. If true, this may have implications for the differences between women and men in both depression and CVD; including the higher prevalence of depression among women (Blehar & Oren, 1999) and CVD being the leading cause of death among women (e.g., Musselman et al., 1998).

Investigation of both activity and restlessness was embedded within a biopsychosocial model of depression in which activity levels are considered outcomes of the cognitive and emotional changes associated with depression. In this model, depressive cognitions may lead to behavioral outcomes including reduced social activities and active recreation. Reduced activity contributes to obesity, which is also related to depression and CVD. Conversely, anxious and vigilant thoughts could increase restlessness. It is possible that short periods of rest promote relaxation, and future research should evaluate the optimal ratio of rest to relaxation as it relates to cardiovascular health. Relaxation therapies such as meditation and yoga indicate the importance of periods of rest for both the mind and body since their goal is that of stillness and rest. A review of the value of relaxation interventions concluded that relaxation is a promising treatment modality for improving emotional well-being (Ernst et al., 1998). Additionally, in a recent systematic review of the literature, yoga

was determined to significantly decrease specific risk factors for CVD, including reducing blood pressure, improving lipid profiles, and reducing sympathetic activation, to name a few (Innes, Bourguignon, & Gill Taylor, 2005). This further supports the notion that rest/relaxation not only promotes mental and emotional well-being, but that it also affects outcomes of physical health and well-being.

When considering explicit attempts at motor activity (i.e., exercise), most data suggest that there is a positive relationship between exercise and emotional well-being. A large number of studies demonstrate an inverse relationship between depression symptoms and physical activity (e.g., Phillips et al., 2003). However, in a recent review, researchers concluded that exercise was an adequate, albeit short-term, intervention for depressive symptoms, but that this judgment was tentative due to the poor quality of available studies (Lawlor & Hopker, 2001). The idea of NEAT and its contribution to cumulative physical activity must not be underestimated when conceptualizing exercise interventions. Exercise related activity and nonexercise activity (i.e., fidgeting and activities of daily living) both account for activity thermogenesis. It is important to remember that exercise's role is relatively negligible. NEAT is the *predominant* component of activity thermogenesis, accounting for up to 50% of TDEE (Levine, 2004). Encouraging people to increase motor activity in general, particularly in small yet frequent ways, may prove to be just as or more effective in combating negative mood as prescribing regular aerobic exercise. This notion certainly warrants further exploration.

It must also be noted at this point that the link between inactivity periods (restlessness) and CVD is speculative. Future research should examine whether periods of inactivity correlate with biological mediators that have a known relationship with CVD, such as blood pressure and heart rate variability (Nemeroff et al., 1998). It is possible that restlessness could be related to blood pressure. However, it is more likely that it could be linked to heart rate or heart rate variability, because movement and, conversely, stillness have known relationships to heart rate (Kramer, Beatty, Plowey, & Waldrop, 2002).

Limitations

Limitations of this study include a small sample size and a single assessment of only one day's physical activity. The interaction was particularly underpowered due to the fact that the sample of depressed males was relatively small (25.0%). Additionally, because anxiety was not overtly assessed, it is impossible to determine the effects that anxiety spectrum symptomatology may have had on the results. Thus, the results could be confounded due to the high incidence of comorbid anxiety disorders in Major Depression. Over half of individuals suffering from depression also present with a comorbid anxiety disorder (Kessler, Nelson, McGonagle, et al., 1996); therefore, anxiety should be assessed in any study pertaining to depression due to its inevitable confounding presence.

Although our study did not find differences in total activity between depressed and nondepressed individuals, physical activity was only assessed in young adults. Most longitudinal, epidemiological studies have primarily looked at middle-aged to

older adults. Also, contrary to other studies looking at obesity and depression, we found no significant relationship with BMI and depression status and our sample had a healthy weight range overall (mean BMI = 23.9; healthy BMI range = 20-25). It is possible that depression-related changes in activity level and obesity do not emerge this early in life.

Directions for Future Research

Future research should examine the relationships among depression, anxiety, activity levels, and periods of rest. Furthermore, the temporal relationship between depression and activity should be assessed using longitudinal methodology. It would also be important to evaluate the relationship between activity levels and depressive symptoms as a within-subjects effect. Even if there are no discernable differences in activity level between depressed and nondepressed individuals, changes in activity level may be related to changes in symptom severity, within the individual, and/or over time. Also warranting examination are what activity levels precede the onset of depression, whether levels of activity stay constant throughout depressive episodes, and whether gross motor activity significantly shifts upon remission of depressive episodes. Additionally, looking at cardiovascular reactivity may provide further insight into the relationship between depression, physical activity, and CVD.

Despite this study's findings, treatment for depression should routinely incorporate physical activity, not only as a possible intervention to treat depression, but also to reduce risk for cardiovascular problems. Additionally, treatment may not only need to focus specifically on the construct of activity (e.g., aerobic exercise).

The role of *in*activity or rest (e.g., relaxation therapy) is also important based on its known health promoting benefits. Meditation, yoga, and massage therapy could all aid in improving the physical and mental health of individuals with depression.

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Table 1. Descriptive characteristics.

	Total Sample (N=57)	Clinically Depressed (n=20)	Never Depressed (n=37)
Age	M=19.7, SD=2.30	M=19.7, SD=2.35	M=19.7, SD=2.31
Gender	54.4% Female	75.0% Female	43.2% Female
BMI	M=23.9, SD=4.46	M=22.8, SD=3.43	M=24.6, SD=4.85
Ethnicity	87.7% Caucasian	80.0% Caucasian	91.9% Caucasian
BDI	M=10.9, SD=12.18	M=22.7, SD=12.56	M=3.9, SD=3.37
IDDL	M=30.1, SD=17.72	M=46.3, SD=15.99	M=21.4, SD=11.45
Use of Stimulant Medication	7.0%	15.0%	2.7%
SSRI	8.8%	15.0%	5.4%
SNRI	1.8%	5.0%	0.0%
NDRI	1.8%	5.0%	0.0%
Mood Stabilizer	3.5%	5.0%	2.7%

Table 2. Correlation matrix.

	Gender	Depression Status	Lifetime Depression Status	Use of Stimulant Medication	Total Activity Level	Immobility Percent
Gender	1	-.304*	-.241	-.114	.003	-.306*
Depression Status		1	.675**	.230	.079	-.128
Lifetime Depression Status			1	.471**	-.073	.049
Use of Stimulant Medication				1	-.019	-.077
Total Activity Level					1	-.007
Immobility Percent						1

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).

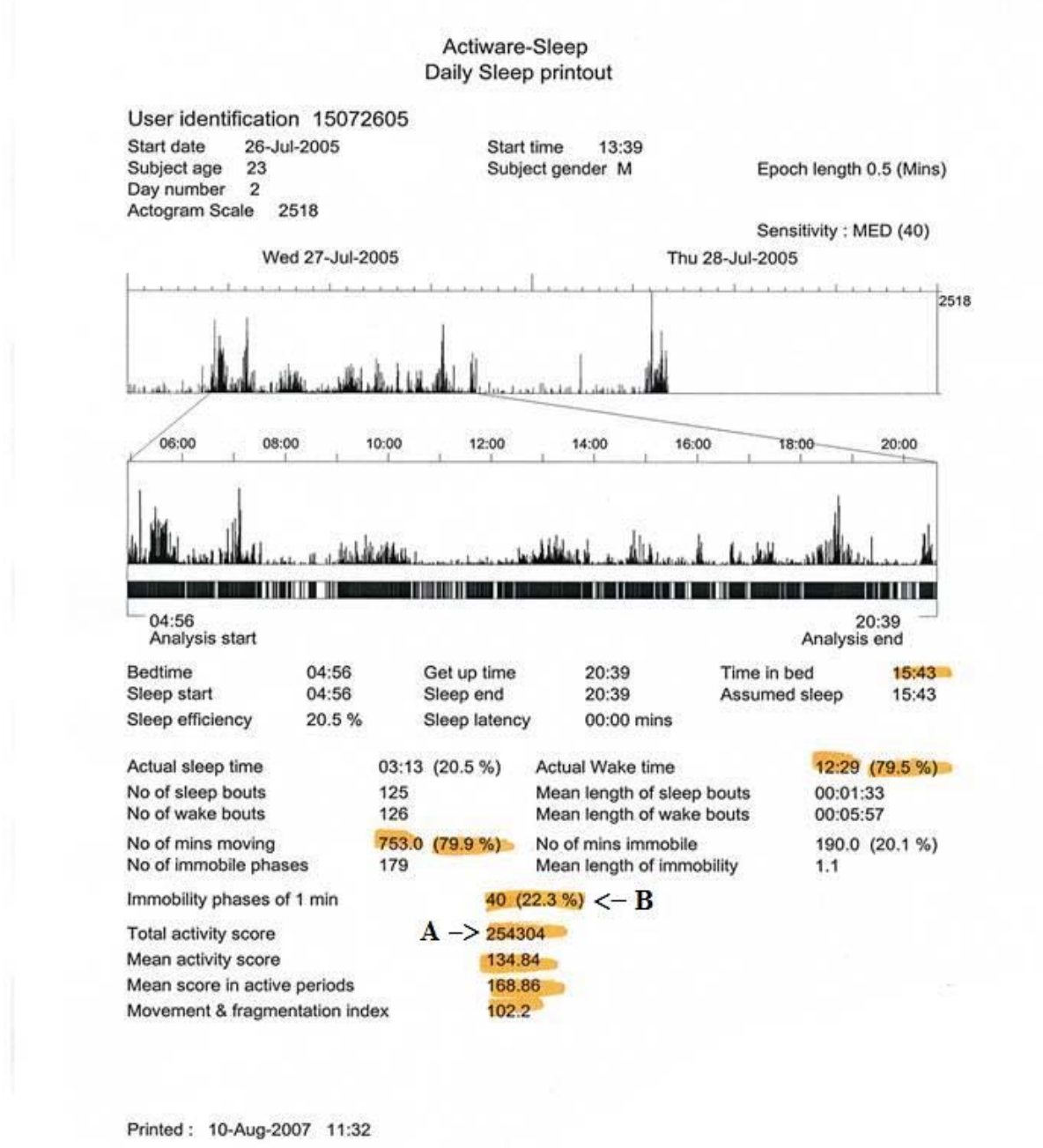

Figure 1: Example of Actigraph printout.

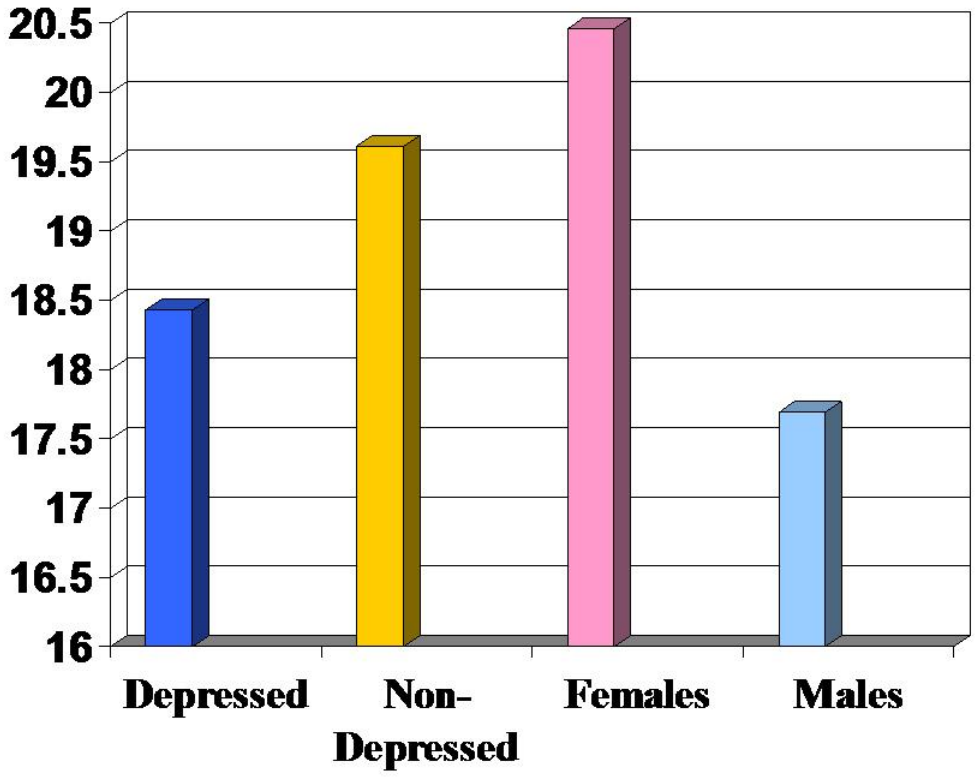

Figure 2: Percent of time immobile. Lower scores are related to higher levels of restlessness.

Appendix A

BDI – Survey Instrument Removed

Appendix A

BDI – Survey Instrument Removed

Appendix A

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Appendix A

BDI – Survey Instrument Removed

Appendix B

IDDL

Try to remember THE WEEK IN YOUR LIFE YOU FELT THE MOST DEPRESSED.

What was the approximate starting and ending date of the episode you have in mind?
began: _____ ended: _____

Circle the number of the one statement that best describes how you felt. Remember to also circle whether you felt that way for MORE or LESS than two weeks.

1. 0 I did not feel sad or depressed.
1 I occasionally felt sad or down.
2 I felt sad most of the time, but I was able to snap out of it.
3 I felt sad all the time, and I couldn't snap out of it.
4 I was so sad or unhappy that I couldn't stand it.
This lasted MORE/LESS than two weeks (circle one)

2. 0 My energy level was normal.
1 My energy level was a little lower than normal.
2 I got tired more easily and had less energy than is usual.
3 I got tired from doing almost anything.
4 I felt tired or exhausted almost all the time.
This lasted MORE/LESS than two weeks (circle one)

3. 0 I was not feeling more restless and fidgety than usual.
1 I felt a little more restless or fidgety than usual.
2 I was very fidgety, and I had some difficultly sitting still in a chair.
3 I was extremely fidgety, and I paced a little bit almost everyday.
4 I paced more than an hour per day, and I couldn't sit still.
This lasted MORE/LESS than two weeks (circle one)

4. 0 I did not talk or move more slowly than usual.
1 I talked a little slower than usual.
2 I spoke slower than usual, and it took me longer to respond to questions, but I could still carry on a normal conversation.
3 Normal conversations were difficult for me because it was hard to start talking.
4 I felt extremely slowed down physically, like I was stuck in mud.
This lasted MORE/LESS than two weeks (circle one)

5. 0 I did not lose interest in my usual activities.
1 I was a little less interested in 1 or 2 of my usual activities.
2 I was less interested in several of my usual activities.
3 I lost most of my interest in almost all of my usual activities.
4 I lost interest in all of my usual activities.
This lasted MORE/LESS than two weeks (circle one)

6. 0 I got as much pleasure out of my usual activities as usual.
1 I got a little less pleasure from 1 or 2 of my usual activities.
2 I got less pleasure from several of my usual activities.
3 I got almost no pleasure from several of my usual activities.
4 I got no pleasure from any of the activities which I usually enjoy.
This lasted MORE/LESS than two weeks (circle one)

7. 0 My interest in sex was normal.
1 I was only slightly less interested in sex than usual.
2 There was a noticeable decrease in any interest in sex.
3 I was much less interested in sex then.
4 I lost all interest in sex.
This lasted MORE/LESS than two weeks (circle one)

8. 0 I did not feel guilty.
1 I occasionally felt a little guilty.
2 I often felt guilty.
3 I felt quite guilty most of the time.
4 I felt extremely guilty most of the time.
This lasted MORE/LESS than two weeks (circle one)

9. 0 I did not feel like a failure.
1 My opinion of myself was occasionally a little low.
2 I felt I was inferior to most people.
3 I felt like a failure.
4 I felt I was a totally worthless person.
This lasted MORE/LESS than two weeks (circle one)

10. 0 I didn't have any thoughts of death or suicide.
1 I occasionally thought life was not worth living.
2 I frequently thought of dying in passive ways (such as going to sleep and not waking up) or that I'd be better off dead.
3 I had frequently thoughts of killing myself.
4 I tried to kill myself.
This lasted MORE/LESS than two weeks (circle one)

11. 0 I could concentrate as well as usual.
1 My ability to concentrate was lightly worse than usual.
2 My attention span was not as good as usual and I had difficulty collecting my thoughts; but this didn't cause any problems.
3 My ability to read or hold a conversation was not as good as usual.
4 I could not read, watch TV, or have a conversation without great difficulty.
This lasted MORE/LESS than two weeks (circle one)

12. 0 I made decisions as well as usual.
1 Decision making was slightly more difficult than usual.
2 It was harder and took longer to make decisions, but I did make them.
3 I was unable to make some decisions.
4 I couldn't make any decisions at all.
This lasted MORE/LESS than two weeks (circle one)

13. 0 My appetite was not less than normal.
1 My appetite was slightly worse than usual.
2 My appetite was clearly not as good as usual, but I still ate.
3 My appetite was much worse.
4 I had no appetite at all, and I had to force myself to eat even a little.
This lasted MORE/LESS than two weeks (circle one)

14. 0 I didn't lose any weight.
1 I lost less than 5 pounds.
2 I lost between 5-10 pounds.
3 I lost between 11-25 pounds.
4 I lost more than 25 pounds.
This lasted MORE/LESS than two weeks (circle one)

15. 0 My appetite was not greater than normal.
1 My appetite was slightly greater than usual.
2 My appetite was clearly greater than usual.
3 My appetite was much greater than usual.
4 I felt hungry all the time.
This lasted MORE/LESS than two weeks (circle one)

16. 0 I didn't gain any weight.
1 I gained less than 5 pounds.
2 I gained between 5-10 pounds.
3 I gained between 11-25 pounds.
4 I gained more than 25 pounds.
This lasted MORE/LESS than two weeks (circle one)

17. 0 I was not sleeping less than usual.
1 I occasionally had light difficulty sleeping.
2 I clearly didn't sleep as well as usual.
3 I slept about half my normal amount of time.
4 I slept less than 2 hours per night.
This lasted MORE/LESS than two weeks (circle one)

18. 0 I was not sleeping more than normal.
1 I occasionally slept more than usual.
2 I frequently slept at least 1 hour more than usual.
3 I frequently slept at least 2 hours more than usual.
4 I frequently slept at least 3 hours more than usual.
This lasted MORE/LESS than two weeks (circle one)

19. 0 I did not feel anxious, nervous, or tense.
1 I occasionally felt a little anxious.
2 I often felt anxious.
3 I felt anxious most of the time.
4 I felt terrified and near panic.
This lasted MORE/LESS than two weeks (circle one)

20. 0 I did not feel discouraged about the future.
1 I occasionally felt a little discouraged about the future.
2 I often felt discouraged about the future.
3 I felt very discouraged about the future most of the time.
4 I felt that the future was hopeless and that things would never improve.
This lasted MORE/LESS than two weeks (circle one)

21. 0 I did not feel irritated or annoyed.
1 I occasionally got a little more irritated than usual.
2 I got irritated or annoyed by things that usually didn't bother me.
3 I felt irritated or annoyed almost all the time.
4 I felt so depressed that I didn't get irritated at all by things that would normally bother me.
This lasted MORE/LESS than two weeks (circle one)

22. 0 I was not worried about my physical health.
1 I was occasionally concerned about bodily aches and pains.
2 I was worried about my physical health.
3 I was very worried about my physical health.
4 I was so worried about my physical health that I could not think about anything else.
This lasted MORE/LESS than two weeks (circle one)

23. 0 This bout of depression is the only one I have ever had.
1 I have had an additional period of depression similar to the one I already described.
2 I have had two more periods of depression similar to the one I already described.
3 I have had three more periods of depression similar to the one I already described.
4 I have had four or more periods of depression similar to the one I already described.

24. 0 I did not get any treatment for how I felt.
1 I got psychotherapy, but did not take anti-depressant medication.
2 I took anti-depressant medication, but did not get psychotherapy.
3 I got psychotherapy and took anti-depressant medication(s).
4 I was admitted to a psychiatric hospital for treatment.