May 2012

Type II Diabetes and Depression in Adults from One Primary Care Practice

Christine Ernst

Follow this and additional works at: http://digitalcommons.ohsu.edu/etd

Recommended Citation
Ernst, Christine, "Type II Diabetes and Depression in Adults from One Primary Care Practice" (2012). Scholar Archive. 902.
http://digitalcommons.ohsu.edu/etd/902

This Dissertation is brought to you for free and open access by OHSU Digital Commons. It has been accepted for inclusion in Scholar Archive by an authorized administrator of OHSU Digital Commons. For more information, please contact champieu@ohsu.edu.
Clinical Inquiry Project:

Type II Diabetes & Depression

Christine Ernst

Oregon Health & Science University
Student Name: Christine Ernst
Degree: Doctor of Nursing Practice

Title of Clinical Inquiry Project:
Type II Diabetes and Depression in Adults from One Primary Care Practice

APPROVED:

Committee Chair: Cheryl Wright, PhD, FNP
(name and credentials)
Signature:

Committee Member: Deborah Messecar, PhD, GCNS
(name and credentials)
Signature:

Committee Member:
(name and credentials)
Signature:

Christine A. Tanner, R.N., Ph.D, FAAN,
Interim Dean, School of Nursing
Signature:

Date: 6/1/12

Submit completed original form to the Graduate Program office.

Revised 12/2011
Clinical Inquiry Project: Type II Diabetes & Depression

Introduction

The Clinical Problem

Type II diabetes mellitus (T2DM), also known as adult-onset diabetes mellitus or non-insulin dependent diabetes mellitus, is a serious chronic illness that, when poorly controlled, can have significant lifelong effects. According to the CDC (2011a), an estimated 25.8 million people in the United States have diabetes. T2DM, the most common form, affects approximately 90-95% of those that have the disease (CDC, 2011a).

Diabetes is a rapidly growing problem. Thirty years ago, type I diabetes (T1DM) and T2DM combined affected about 2.5% of the United States population (CDC, 2010). Today it impacts over 8% of the total population and almost 27% of those aged 65 and older (CDC, 2011a). Diabetes is a risk factor for micro- and macrovascular disease, which leads to complications such as cardiovascular and cerebrovascular disease as well as retinopathy, nephropathy, neuropathy, increased incidence of infection, increased risk for disability and increased mortality. It contributes to about $174 billion worth of total direct and indirect health care costs and is the seventh leading cause of death (CDC, 2011a).

Among adults with T2DM, depression often occurs as a comorbid condition (Anderson, Freedland, Clouse, & Lustman, 2001; Nouwen A. Winkley K. Twisk J. Lloyd CE. Peyrot M. Ismail K. Pouwer F. European Depression in Diabetes (EDID) Research Consortium, 2010). Studies suggest that patients with T2DM who are depressed are at higher risk for increased rates of complications, have increased costs of health care, poorer quality of life and increased mortality (Kalsekar et al., 2006; W. J. Katon et al., 2005; Lin et al., 2010; Verma et al., 2010; L.
H. Williams et al., 2010). Although depression among diabetics is a known risk for poor T2DM-related outcomes, it often goes undetected or untreated (Lustman & Harper, 1987).

Current research supports the association between depression and diabetes (Ali, Stone, Peters, Davies, & Khunti, 2006; Anderson et al., 2001; Nouwen et al., 2010); however, the relationship between the two remains unclear. Some theories hold that depression may cause T2DM due to poor hormonal regulation, impaired glucose transport, and insulin resistance (Musselman, Betan, Larsen, & Phillips, 2003). While still supporting a biochemical link, conversely, other theories implicate the physiologic process of hyperglycemia and insulin resistance (Fujinami et al., 2008; Krabbe et al., 2007; Pickup, 2004) as a cause of depression (Castren & Rantamaki, 2010). Alternative theories conclude that the burden of the disease and its complications are the primary determinants for developing depression (Adriaanse et al., 2005; de Groot, Anderson, Freedland, Clouse, & Lustman, 2001). Still others support a bidirectional relationship between diabetes and depression, determining that diabetes can be both a cause and consequence of depression (Golden et al., 2008; Pan et al., 2010). Despite uncertain causality, studies have shown that screening and detecting depression among diabetics may lead to improved health outcomes (Khazaie et al., 2011; M. M. Williams et al., 2007).

At the state level, 219,000 adult Oregonians had T1DM and T2DM in 2009 (CDC, 2011b). Similar to the national trend, Oregon’s incidence and prevalence of diabetes has grown significantly over time. The age-adjusted percentage of adult Oregonians with all-type diabetes rose from 3.9% in 1994 to 7.1% in 2009, an 82% increase (CDC, 2011b). Preventing and treating diabetes and obesity is a priority for the Oregon Health Authority, formerly known as the Oregon Department of Human Services (DHS), Public Health Division (Oregon DHS, 2008).
Current guidelines for the diagnosis and management of diabetes recognize the importance of screening for depression. The American Diabetes Association (2011) recommends periodic assessment of psychosocial status based on expert opinion and presents Level C evidence supporting psychosocial assessment in the presence of poor self-management. Additionally, the National Quality Measures Clearinghouse lists several quality improvement measures monitoring regular screening for depression among adult diabetics (NQMC, n.d.a; NQMC, n.d.b). Despite these recommendations, patients may not be routinely screened for depression in the clinic setting.

As a DNP student, my objective was to translate research into practice and improve quality of care. At the time of this project, there was no established screening protocol for patients at an Oregon City primary care clinic. In line with the latest evidence-based guidelines, I assumed a leadership role and conducted screenings for depression among adults with T2DM in the practice.

The purpose of this clinical inquiry project was to determine the prevalence of depression among adults with T2DM in an Oregon City clinic and to identify associations between glycemic control and level of depressive symptoms. Clinical inquiry questions included:

1. What was the percentage of adult type II diabetics that have depression as measured by a score greater than nine on the Patient Health Questionnaire (PHQ-9)?

2. Controlling for demographic and clinical data, was there a correlation between poor glycemic control and increased depressive symptoms?

Synthesis of Evidence

Introduction
A link between diabetes and depression has been theorized for centuries. A British physician named Dr. Thomas Willis is said to have observed a relationship between diabetes and depression in the late 17th century as he surmised that diabetes was the outcome of “sadness or long sorrow” (Willis, 1971). Anderson et al. (2001) confirmed this hypothesis over 300 years later with a meta-analysis of 42 studies. The results of the meta-analysis indicated a prevalence of clinically relevant depressive symptoms among all-types of diabetics to be 31% (chi-squared =159.8, P<0.0001) and an odds ratio of 2.9 (95% confidence interval (CI) 2.3-3.7, chi-squared = 84.3, P<0.0001) for depression among type 2 diabetics over non-diabetics (Anderson et al., 2001).

While the results are slightly varied, others have validated these findings. Ali and colleagues (Ali et al., 2006) conducted a systematic review of the literature using 10 controlled studies, which determined the overall prevalence of depression was 17.6% in patients with T2DM and the odds ratio for depression was 1.77 (95% CI=1.5-2.0) in type 2 diabetics compared to those without diabetes. Further, Nouwen et al. (2010) found a pooled relative risk of 1.24 (95% CI=1.09-1.40) or 24% increased risk for developing depression in type 2 diabetics without depression at baseline.

Despite numerous studies supporting a correlation between diabetes and depression, the causal mechanism remains unclear. Some theories have shown that depression can lead T2DM. Depression, a state of neurohormonal imbalance, can alter glucose transport and increase inflammation, contributing to insulin resistance and islet cell dysfunction, which can lead to the development of T2DM (Musselman et al., 2003). Alternatively, T2DM is also theorized to cause depression. Hyperglycemia and insulin resistance may physiologically alter inflammatory processes and neurotrophic function (Fujinami et al., 2008; Krabbe et al., 2007; Pickup, 2004),
which may impact neuronal networks, leading to depression (Castren & Rantamaki, 2010). Additionally, T2DM is also believed to cause depression through the psychosocial burden of chronic disease as well as the threat or existence of complications from the disease (de Groot et al., 2001; Pouwer et al., 2005; Talbot & Nouwen, 2000). Further, several studies have shown evidence of a bidirectional relationship, suggesting that depression can result from diabetes and vice versa (Golden et al., 2008; Mezuk, Eaton, Albrecht, & Golden, 2008; Pan et al., 2010).

Because diabetes mellitus is a disease characterized by hyperglycemia and there is strong evidence linking diabetes to depression, it is logical to speculate a relationship between hyperglycemia and depression. This review of literature will examine studies whose objective was to identify a relationship between glycemic control and depressive symptoms with the hypothesis that poor glycemic control correlates with increased depression.

**Critical synthesis**

A Medline and PsycINFO search revealed 42 articles regarding glycemic control and depression in type 2 diabetics. Of those 42, 36 supported evidence of a relationship between glycemic control and depression, while six did not. Despite the majority, findings are mixed. As with diabetes and depression, there is not a clear causal relationship between glycemic control and depression.

Many studies found evidence of a relationship between glycemic control and depression. Most of these were cross-sectional studies that found significant correlations between worsening glycemic control (Hb A1c) and increased depressive symptoms (Daly et al., 2009; Grandinetti et al., 2000; Katon et al., 2004; Kivimaki et al., 2009; Papelbaum et al., 2010; Trief et al., 2006; Wagner, Abbott, Heapy & Yong, 2009). Three cross-sectional studies found a correlation between glycemic control and depression in men, but not women (Cherrington, Wallston, &

Asghar, Hussain, Ali, Khan & Magnusson (2007) and Timonen et al. (2005) found significant relationships between glycemic control and depression, but used measures other than Hb A1c to determine glycemic control. Asghar et al. (2005) found a significant association between fasting blood glucose and mean depressive score (F-value 854.2, p<0.01). Timonen et al. (2005) found a negative correlation between depression scores and insulin sensitivity in patients with impaired glucose tolerance (r=-0.24, p=0.029).

Egede & Osborn (2010) and Fisher et al. (2010) both found evidence to suggest that depression and glycemic control were related, but not directly. Egede & Osborn (2010) reported that depression affects self-care behaviors, including diabetes knowledge, personal motivation, and social support, which is marginally associated with glycemic control (r=-0.20, p=0.08). Similarly, Fisher et al. (2010) concluded that there was no cross-sectional, longitudinal or time-concordant significance with major depressive disorder and glycemic control; however, there was a cross-sectional and time-concordant relationship between Hb A1c and diabetes distress (b=0.026, p=0.006 and b=0.023, p=0.001, respectively). Diabetes distress is measured using a self-report scale like other depression screening tools, but its questions focus on psychosocial disease burden rather than strictly depressive symptoms.

Further, Stankovic, Jasovic-Gasic & Zamaklar (2011) determined depression was predicted by psychological and disease-specific variables. The Problem Areas in Diabetes (PAID) questionnaire total score, which measures diabetes distress, significantly predicted depression (OR=1.084, 95% CI=1.038-1.133, p=0.000). The total number of life events, using
the Scale of Life events tool (OR=4.528, 95% CI=1.929-10.630, p=0.001) as well as presence of neuropathy (OR=8.699, 95% CI=1.115-67.885, p=0.039) also significantly predicted depression. The somatic sub-score for the Beck Depression Inventory in depressed type 2 diabetics correlated with Hb A1c level (r=0.343, p=0.020) in this study.

One longitudinal study, Chiu et al. (2010), determined that higher baseline depressive symptoms were associated with poor glycemic control in a 5-year follow-up study (b direct=0.10, t=2.0, p<0.05). Similarly, Richardson, Egede, Mueller, Echols & Gebregziabher (2008) conducted a study among 11,525 veterans that found a significant longitudinal relationship between depression and glycemic control. The mean difference in Hb A1c values between depressed and non-depressed was 0.13 (95% CI=0.03-0.22, p=0.008).

Several other studies attempted to explore a causal pathway. Among those who studied whether depression was associated with an increased risk for poor glycemic control were Zuberi, Syed, & Bhatti (2011), Dirmaier et al. (2010), and Gross et al. (2005). Zuberi et al. (2011) reported greater than 5-fold risk for poor glycemic control in depressed patients, while Dirmaier et al. (2010) conducted a longitudinal study reporting twice the risk for poor glycemic control in those with baseline depression at 12 months (Adjusted OR=2.01, 95% CI=1.10-3.69). Gross et al. (2005) determined that risk for poor glycemic control increased with increased depressive symptoms. Mild to moderate depression had 1.51 times the risk (95% CI=0.67-3.42) and moderate to severe depression 3.27 times the risk (95% CI=1.23-8.64) of having poor glycemic control (Gross et al., 2005).

Gale et al. (2010), Calhoun et al. (2010), Poongothai et al. (2010), Maraldi et al. (2007), and Grandinetti et al. (2000) all found that poor glycemic control led to increased risk for depression. Calhoun et al. (2010) determined among 2,832 participants that every one-unit
increase in Hb A1c increased depression odds by 22% (OR=1.22, 95% CI=1.05-1.42). A separate study found that worsening glycemic control, excluding patients with known diabetes, led to incrementally increased risk for depression, lending evidence toward the theory that hyperglycemia has a pathophysiologic effect on neurohormonal function (Poongothai et al., 2010). In contrast, Gale et al. (2010) found that in 4,293 veterans, those with diagnosed T2DM had nearly four times the risk for major depression than patients with normal fasting glucose (OR=3.82, 95% CI=1.68-8.70), while those with undiagnosed T2DM had less than twice the risk as those with normal fasting glucose (OR=1.80, 95% CI=1.01-3.22). The Gale et al. (2010) study favors the psychological burden of disease hypothesis.

Aikens, Perkins, Lipton & Piette (2009) observed both pathways over six months. They concluded that depressive symptoms did not likely predict poor glycemic control because their results were not significant after controlling for baseline glycemic values (p=0.361). Glycemic control generally did not predict depression either (p=0.558); however, glycemic control predicted depression in patients on insulin (beta=0.31, p=0.002) but not in patients on oral hypoglycemics only (beta=-0.10, p=0.210).

Four randomized controlled trials (Bogner & de Vries, 2010; Echeverry, Duran, Bonds, Lee, & Davidson, 2009; Georgiades et al., 2007; J. W. Williams Jr et al., 2004) indicated there may be a relationship between glycemic control and depression if an intervention resulted in similar effects on both variables. All the studies targeted depression with their intervention, and were, therefore, looking for a corresponding decrease in Hb A1c. Bogner & deVries (2010) and Echeverry et al. (2009) both found Hb A1c and depressive symptoms significantly lowered in the intervention group. Georgiades et al. (2007) and Williams et al. (2004), however, did not find
similar changes in Hb A1c and depressive symptoms. Despite significantly lower depressive symptoms, Hb A1c values did not decrease accordingly.

There are several gaps in the literature. First, despite numerous studies, the relationship between glycemic control and depression is still unclear as in some studies there is a strong correlation, but in others there does not seem to be a relationship. Second, there is a lack of randomized, controlled studies to suggest how interventions, either on depression or on glycemic control can affect change in the other condition. To date, most studies have been observational studies, albeit some with large sample sizes or control groups (case-control and cohort studies); however, these studies are simply descriptive and lack clinical application or significance. Most authors call for effective treatment of diabetes and depression, but without being able to name a specific regimen other than standard, well-accepted treatments for each condition. Therefore, a lack of consensus remains on the cause, treatment, and direction of the relationship between depression and T2DM. Future research directions include more bench science to examine effects of glycolization of nerve cells, in addition to more randomized control trials to assess the effects of specific interventions for lowering depression symptoms and Hb A1c to determine if there is, indeed, a causal relationship.

Other sources

Several clinical practice guidelines have acknowledged the strong correlation between diabetes and depression. The American Diabetes Association (ADA) (2011) and American Association of Clinical Endocrinologists (AACE) (2011) both recommend routine screening for comorbid depression in diabetic patients, as well as treatment for depression where necessary. The ADA (2011) suggests screening for psychosocial problems especially when “self-management is poor,” implying that uncontrolled hyperglycemia may be a sign of depression (p.
S5). Additionally, the AACE (2011) guideline states, “Untreated comorbid depression can have serious clinical implications for patients with DM because depression contributes to poor self-care, less treatment-related adherence, and poor glycemic control,” with the caveat that, “Continuing use of antidepressant medication is associated with an increased relative risk of T2DM,” (p. 38).

Further, the Joslin Diabetes Center (2007) recommends screening for depression in the older adult in both T1DM and T2DM. Depression is recognized as a condition that is part of the geriatric syndrome, along with cognitive dysfunction and functional disabilities. The Joslin (2007) guidelines state, “Depression in older adults with diabetes is associated with poor glycemic control, decreased adherence, increased functional disability and mortality,” (Recommendations section, para. 4). Thus, screening should be performed when “conditions are suspected or when an older adult fails to achieve the treatment target,” (Joslin Diabetes Center, 2007, Recommendations section, para. 4).

Summary

Although causality remains unclear, there is strong evidence of a correlation between T2DM and depression. Additionally, the literature supports a relationship between poor glycemic control and depression. Despite current guideline recommendations, there is no routine screening for depression among type 2 diabetics in an Oregon City family clinic. The objectives of this project are to identify the prevalence of depression in a sample of type 2 diabetics and determine if a relationship exists between glycemic control and depression. Untreated depression can lead to increased adverse outcomes; therefore, it is important to target and treat depression to prevent complications and death.
The prevalence of diabetes is growing exponentially. Knowing that T2DM will be an increasing proportion of the population in primary care clinics, it is paramount to be proactive about management of these patients. Depression in the Oregon City clinic is likely undertreated because there are no screening protocols in place. This practice improvement project is in line with current literature recommendations and seeks to establish a need for routine screening in T2DM patients.

**Methods**

**Clinical Inquiry Design**

For this clinical inquiry project, I used a descriptive, cross-sectional design and partial correlational analysis. The purpose of the study was to determine the prevalence of depression in a sample of T2DM patients at an Oregon City clinic and further analyze the data for correlation between glycemic control and depression severity. The clinical site was not routinely screening for depression; thus, this project was first assessing the presence and severity of the problem. In choosing a cross-sectional design, there was an inability to determine causality in the relationship as well as a potential loss of statistical significance because of the timeframe from which the data is captured; however, given the time constraints of this project and lack of prior data from the clinic on this topic, a cross-sectional design was appropriate as an initial approach. Additionally, there was a possibility for a prevalence-incidence bias because patients with T2DM and depression have a higher mortality than T2DM without depression.

A partial correlational analysis was also used to assess for a relationship between glycemic control and depressive symptoms. To conduct testing, I made valid assumptions that the data would be normally distributed and that the variables were both random and linearly related. If the data was not normally distributed, I considered utilizing Spearman’s Rho.
Setting

The project was conducted in a privately owned family practice in Oregon City, Oregon. There were six providers employed by the clinic: two medical doctors, two doctors of osteopathy, and two family nurse practitioners. All the providers diagnosed and treated T2DM patients. On a given day, there were five providers working, each seeing several adult T2DM patients per day. As a privately owned practice, salary was based on productivity. It was paramount that the screening practice be instituted with minimal impact on visit time. An advantage was the screening tool used in this study was one that was already familiar to the clinic, but was not necessarily used with regularity to screen for depression in diabetics. This made for an easier transition to use post-data collection.

An additional driving force was a desire for better diabetic outcomes. With the advent of electronic health records and accountable care organizations, patient health outcomes may guide reimbursement from the government and private health insurance companies. Evidence indicates that depression exacerbates diabetic outcomes, thus, there is an incentive to screen and treat depression among T2DM patients to help improve glycemic control and prevent costly adverse sequelae.

A potential restraining force for implementing new screening practices may have been a concern over management of a positive screening. Once a positive result was recorded, it placed an increased burden on the screening provider to follow through with an intervention. The presence of Major Depression may have required a referral to a mental health provider. There was a shortage of mental health providers in the area, so the patient may not have been able to receive therapy until several weeks after the initial finding. Therefore, it became the screening provider’s responsibility to ensure proper care for the depressed patient.
Sample

The sample for this study was patients from one Oregon City family practice. Inclusion criteria included: T2DM patients, as identified by the International Classification of Diseases, 9th revision, as 250.X0 or 250.X2 on their problem list who had a glycosylated hemoglobin (Hb A1c) drawn within the last 6 months, 18 years old or older, and had no major psychiatric illness other than a possible diagnosis of anxiety or depression. Exclusion criteria included: any patient with type I diabetes mellitus, children under 18 years, any patient on medications known to exacerbate glycemic control such as atypical antipsychotic agents and any unforeseen situations at the discretion of the researcher. Estimated sample size was 100. To obtain a confidence interval of 90%, a sample size of 214 would have been necessary, based on the following formula:

\[ n = \frac{N \times r}{(N-1)E^2 + x} \]

Where \( n \) = sample size, \( E \) = margin of error, \( N \) = population size, \( r \) = fraction of responses interested in, and \( Z(c/100) \) is critical value of confidence level \( c \). Because the goal of this study was not necessarily to provide generalizable population data and it was a pilot study, a smaller sample size (\( n=100 \)) was employed. The margin of error was 7.81% using the formula:

\[ E = \sqrt{\frac{N - n}{n}x/n(N-1)} \]

with symbols as above. If I was unable to recruit sample size goal, I would have adjusted the number of dependent variables analyzed to compensate for the smaller sample size and maintain statistical rigor.

Patients were pre-selected for recruitment the day before their appointment, based on previous diagnosis of T2DM in their problem list and Hb A1c result from the last 6 months. Upon arrival to the clinic for their scheduled appointment, a medical assistant queried their
interest in participating in the study. If the patient declined, the process ceased there. If the patient agreed, I consented the subject for the study.

**Description of Intervention**

After obtaining consent, seen in Appendix A, the participants were brought to an exam room and administered the clinic’s depression screening tool. The screening tool with additional demographic survey is found in Appendix B. The participant was left alone to complete the questionnaire. Once finished, I collected the tool and communicated the results to the patient’s primary care provider prior to the visit. The results revealed the presence or absence of depressive symptoms as well as severity of symptoms. If the patient scored greater than nine, the providers used their discretion whether to treat with medical therapy and/or refer for counseling. If the participant indicated that he/she had considered harming themselves or others, immediate referral to an inpatient psychiatric unit was made.

**Measures**

There were two primary outcomes that were measured during this study: depression score and glycemic control. Depression was measured using the Patient Health Questionnaire (PHQ-9) screening tool. The PHQ-9 is a reliable and valid self-administered questionnaire for screening depression and assessing depression severity across multiple settings (Kroenke, Spitzer, & Williams, 2001; Martin, Rief, Klaiberg, & Braehler, 2006). A score greater than nine was considered positive for depressive symptoms. It was selected as the screening tool for this study because in addition to being a valid and commonly used depression screening tool, it was available and already in use at the study site. It was more likely for the intervention to change practice if the tool was accessible and familiar to the providers at the clinic.
The other outcome, glycemic control, was the lab value for glycosylated hemoglobin or hemoglobin A1c (Hb A1c). It is an appropriate measurement to assess diabetes management and control because it allows the health care provider to assess an estimated average blood glucose over the previous 3-4 months. The lifespan of a red blood cell, which contains hemoglobin (Hb), is approximately 120 days. During that time, Hb is exposed to glucose in the bloodstream. The glucose molecules react with Hb forming glycosylated Hb, which is a permanent change. A rise in glycosylated Hb reflects the cell’s exposure to glucose during its lifecycle. There is a predictable rise in Hb A1c based on increases in average serum glucose, making it a reliable measure (Dawson, 2010). A Hb A1c value greater than 7.0% is considered uncontrolled by the American Diabetes Association (2011).

Data Collection Procedures

Participant data, including gender, age, body mass index, and Hb A1c level was collected from MedInformatix, the electronic medical record utilized by the clinic. All Hb A1c blood samples were collected on-site by the medical assistants. Lab samples remained in-house and were run on a point-of-care machine, A1C Now by Bayer, that reported the Hb A1c values. From the screening tool, I collected the depression score. I also queried for race/ethnicity and educational level via questionnaire, which I attached to the screening tool. Data was deidentified and entered into a Microsoft Excel spreadsheet for later importation to SPSS for analysis.

Analytic Methods

There were two objectives. The first was to determine the prevalence of depression in a sample of adult type 2 diabetics. This was measured using the following formula:

\[
\text{prevalence} = \frac{\sum_{i=1}^{n} x}{n}, \text{ where } x=1 \text{ if depressed and } n=\text{sample.}
\]
For the second objective, I analyzed if a relationship existed between glycemic control and depression along with other patient demographics using a partial correlation. The following formula was used: \( p_{corr} = \frac{t}{\sqrt{t^2 + n + k}} \), where \( t \) was the t statistic, \( n \) was the number of observations and \( k \) was the number of independent variables (Green, 2008).

The cost to conduct the study was minimal. I collected existing patient data from the clinic’s electronic health record. The laboratory data was previously entered, thus the data collection occurred at no extra cost to the participant or the clinic. The PHQ-9 screening tool was free to use. Potential costs may have accrued for the participant if the screening was positive for depressive symptoms, as medical treatment or referral to mental health providers may have been required. Alternatively, treatment may have curtailed later costs for the participant in the form of co-payments for medication or therapy. Depression as a comorbid condition to T2DM can increase health care utilization and health care costs (Kalsekar et al., 2006). Better glycemic control through treatment of depression may decrease overall costs of care (Wagner et al., 2001).

Data was presented using tables and figures. Demographic data was displayed in table format in two columns depicting differences between depressed versus non-depressed patients. Graphs were used to delineate the relationship between glycemic control and depression severity.

After collection of data from subjects, I deidentified the data. The subject’s name was not used. Instead the subject was assigned a number (ex. subject number one was assigned 101). The master list was kept in a locked file cabinet in a locked clinic office. Signed consent forms were kept in the Oregon Health & Science University’s School of Nursing research department, room 236, in a locked cabinet in a locked closet. Data was kept on a personal computer but was
deidentified once entered, and the computer was password protected. This data was not repositoried.

**Protection of Human Subjects**

I received approval from Oregon Health & Science University’s Institutional Review Board (IRB) prior to data collection. The clinic, not having its own IRB, granted approval through reciprocity. Respect for participants was maintained throughout the course of this study. Concern for coercion existed because the medical assistants initially approached the patients for the study. I ensured that the patient understood there was no reward or punishment for either joining or refusing participation when initially approached or during the consent process. There was also concern that beneficence was upheld with results from the screening tool. It was the duty of the clinician to provide services based on patients’ clinical presentations. The depression screening score was entered into the medical chart. The clinicians acknowledged this objective data and treated it according to best medical evidence.

**Plan for dissemination**

I presented a poster at the WIN conference in Portland in April of 2012. I also presented the same poster during Research Week at Oregon Health & Science University’s School of Nursing in May of 2012. I will also seek publication in the *American Journal of Nursing* under their “Original Research and Quality Improvement” column or the *Journal of Nursing Diabetes*.

**Timeline**

I received IRB approval in the fall of 2011. Data collection occurred in December and January of 2012. Data analysis occurred in February of 2012. Writing and synthesis of data occurred in March and April of 2012, followed by final presentation at the end of May 2012.

**Results**
Sample

Approval for this study was obtained from Oregon Health & Science University’s Institutional Review Board (IRB). One hundred subjects were approached to participate in the study in Oregon City. Of these, 96 agreed to participate.

Table 1 depicts the demographic and other important characteristics of the sample. The average age of subjects was 65 years old (standard deviation (SD) = 12.76). Slightly more than half of participants were female (n=54, 56.3%). Educational status was mixed. Most subjects’ highest degree was a high school diploma (n=52, 54.2%), followed by 2-year college diploma (n=20, 20.8%), 4-year college diploma (n=12, 12.5%), did not finish high school (n=9, 9.4%), and graduate level college degree (n=3, 3.1%). Ninety-three percent (n=89) of the subjects were Caucasian. Body mass index ranged from 20.1 to 49.7 kg/m^2, with a mean of 33.4 kg/m^2 (SD = 6.96). A chart of body mass index frequencies can be seen in Figure 1. The mean Hb A1c was 7.1 (SD = 1.25). A histogram of Hb A1c values can be seen in Figure 2.

Findings

The first research question inquired about the prevalence of depression among the T2DM population at the Oregon City clinic. The PHQ-9 questionnaire scoring ranges from zero to 27. A score greater than nine was considered a positive screening for depression. Twenty-three participants recorded a score of 10 or higher, resulting in a prevalence of 24%. Table 2 represents the demographic and clinical characteristics of those who screened positive for depression versus those who did not.

Females had a higher prevalence of depression (31.5%) than males (14.3%). Two of the seven (28.6%) non-white participants were depressed. Both were Hispanic. Twenty-three percent of the Caucasian subjects were depressed. One in three people with 4-year college
degrees or higher had depressive symptoms. Twenty-nine percent of people whose highest
degree was a high school diploma had depression, followed by 15% of people with 2-year
degrees. None of the subjects who did not finish high school registered as having depression.
People 65 years old and older had a lower prevalence of depression (20.8%) than those less than
age 65 (27.1%). More than one-fourth of subjects with BMIs greater than or equal to 30 had
depression. Those less than 30 had a prevalence rate of 16.7%. Participants with good glycemic
control (Hb A1c <7%) had a higher rate of depression (26.9%) than those with poor control
(20.5%).

The second research question examined if there was a relationship between glycemic
control and level of depressive symptoms, controlling for other demographic and clinical factors.
A partial correlation was used. After controlling for age, gender, education, race, and BMI, Hb
A1c and depression did not have a significant correlation (r=.091, p=.392).

Of the 96 original participants, 43 had Hb A1c values that were considered current,
defined as within the last 30 days. After controlling for age, gender, education, race, and BMI,
current Hb A1c and depression were found to be moderately correlated (r=.31), but not
statistically significant (p=.06). A plot of the data can be seen in Figure 3.

Discussion

Interpretation

The results of this study indicated that approximately one in four type II diabetics had at
least mild depressive symptoms, while the prevalence of depression in the general population is
about one in twenty (Pratt & Brody, 2008). While the study did not necessarily identify
depression as a new diagnosis in patients, it did identify which patients were being adequately
treated for their depression and which were not, if they were being treated at all.
In post-hoc analysis, I excluded Hb A1c values that were collected more than 30 days prior to the depression screening because it may have affected the validity of the results. Permitting the use of values from up to six months ago may not reflect the patient’s current state. Therefore, a sub-set of data (n=43) were analyzed, which only included participants whose Hb A1c had been drawn within the last month. The results were not statistically significant (p=.06), even though a correlation of r=.31 would be considered moderately robust. However, my study was underpowered because of my small sample size. It is important to note that the correlation in the group that included both the old and the current Hb A1c values was far smaller (r=.09). Had it been possible to obtain current Hb A1c values on all of the patients, it is likely that a significant association would have been detected between depressive symptoms and poorer glycemic control. These results are consistent with prior studies that have reported a relationship between glycemic control and depression severity (Daly et al., 2009; Fortmann, Gallo, Walker, & Philis-Tsimikas, 2010; Grandinetti et al., 2000; Stankovic, Jasovic-Gasic, & Zamaklar, 2011; Trief et al., 2006).

**Context**

Prevalence rates of depression among type II diabetics vary (Ali, Stone, Peters, Davies, & Khunti, 2006; Anderson, Freedland, Clouse, & Lustman, 2001). Inconsistencies in findings have been attributed to demographic variables and methodologies for assessing depression (Anderson et al., 2001). As in nondiabetics, women have a higher rate of depression among diabetics than men (Ali et al., 2006; Anderson et al., 2001). Racial/ethnic sub-groups have shown a variation in depression prevalence (Li, Ford, Strine, & Mokdad, 2008). Other studies have shown that depression is associated with obesity (Onyike, Crum, Lee, Lyketsos, & Eaton, 2003). Self-report questionnaires also consistently result in increased prevalence rates over interview-based...
estimates (Anderson et al., 2001). This is likely because clinical interviews may not detect milder forms of depression, while self-report questionnaires capture major depressive disorder as well as a broader spectrum of other depressive disorders, including dysthymic disorder or minor depression.

Similar findings were delineated in this study. Women had higher rates; however, the sample was not large or diverse enough to determine if there was a significant variation between ethnicities’ rates of depression. People with higher body mass indexes on average had a greater prevalence of depression.

Prevalence of depression from this study was within the range of estimates from other similar studies. Anderson and colleagues (Anderson et al., 2001) determined an aggregate estimate of depression at 32.9% for type II diabetics using self-report scales. Another meta-analysis found a depression rate of 17.6% in patients with T2DM (Ali et al., 2006). In yet another study, data from telephone screenings in the US using a self-report questionnaire revealed depression prevalence estimates between 17.3% and 24.0% for type II diabetics who did not use insulin and who used insulin, respectively (Li et al., 2008). Despite similar findings, it is possible that rates of depression were slightly elevated in this study because of seasonality. Screenings were conducted in the winter months in a northern latitude location, which may have inflated some scores (Harmatz et al., 2000; Oyane, Bjelland, Pallesen, Holsten, & Bjorvatn, 2008).

In prior research, results are mixed for the relationship between glycemic control and depressive symptoms with some studies finding a significant relationship and others not. Five studies found a positive correlation between poor Hb A1c values and increased level of depression (Daly et al., 2009; Fortmann et al., 2010; Grandinetti et al., 2000; Stankovic et al.,

The recruitment strategy for this study was successful in that type II diabetic patients who presented for common complaints, not just follow up diabetic visits, could be screened for depression. However, this decreased the likelihood that their Hb A1c was current, as their providers often did not order Hb A1c’s during these episodic visits. Because I was unable to obtain current Hb A1c data on all subjects, this created a limitation in terms of having a large sample with current Hb A1c values to address the practice improvement questions. I chose to reduce the sample size to 43 to limit the confounding factor of older Hb A1c values. This, however, decreased the power of my study, leaving us unable to determine if a larger sample would have shown a statistically significant association.

Despite a lack of statistical significance, these findings are clinically significant. The prevalence rate supports the need for routine depression screening. Furthermore, a recent study indicated that simultaneous management of diabetes and depression in a primary care setting could improve control of both conditions (Bogner, Morales, deVries, & Cappola, 2012). Therefore, it is important to consider concurrent treatment of depression during regular diabetic check ups.

Financial Considerations

The results of this study reinforce the need for routine screening and treatment of depression in type II diabetics. A large portion of the diabetic population suffers from depressive symptoms. Additionally, it appears that there may be a small positive correlation between
glycemic control and depression severity. No financial data was collected for this study.

Nevertheless, a business case can be made.

From a micro level, the cost of conducting routine depression screenings is minimal. The clinic would have to pay the printing costs of copying the screening tool, but it is otherwise free to administer. It could be distributed to patients in the waiting room prior to their appointment to avoid using visit time filling it out. The benefit of having objective data for measuring depression is invaluable for tracking outcomes of depression treatment. The cost to patients varies. Laboratory fees for Hb A1c values are not additional costs to the patient as they are part of routine, evidence-based care for diabetics. A positive depression screening could result in additional expense for treatment of the depression; however, treatment of the depression may prove more cost-effective than not treating the disorder and remaining at increased risk for expensive, adverse outcomes.

On a macro level, evidence has shown that type II diabetic patients with depression have higher rates of complications (Lin et al., 2010; Williams et al., 2010) and increased health care costs (Kalsekar et al., 2006). Diabetic complications arise in patients with poor glycemic control. Over time, continued hyperglycemia increases the need not only for more diabetic medications, but also for medications to treat complications, including therapy to treat peripheral neuropathy, and costly procedures, such as amputations or hemodialysis. Identifying and treating at-risk patients for depression could potentially lower costs of health care for society by preventing complications (Wagner et al., 2001).

Measuring financial impact of routine depression screenings would be difficult given the number of variables affecting outcomes and lack of a controlled setting. One future intervention, though, could follow a small sample of patients who screened positive for depression over time
to monitor the changes in their depression severity and compare that with the number of new complications related to their diabetes. Costs of medications or procedures could be estimated to determine if lowering depression proved cost-effective.

**Situation Analysis**

The idea was conceived in the summer of 2011 to create a quality improvement project involving the care of type II diabetic patients in one family practice. I originally wanted to determine the screening rate for depression in these patients, but after conferring with the clinic providers, it became apparent that this practice was not routinely done. Thus, I designed this project, conducting depression screenings on a sample of adult type II diabetic patients to establish the need for depression screenings and secondarily, to examine the potential relationship between glycemic control and depressive symptom severity.

Overall, the project was successful. It was proposed, approved by the IRB, and conducted in its entirety at the proposed clinical site in the allotted amount of time. The results of the study provided useful clinical information to providers regarding presence and severity of current depression, as well as an opportunity to ensure that diabetic patients met current diabetic guidelines by having a Hb A1c drawn within the last 6 months (ADA, 2011).

Methods for examining correlation could be improved. Inclusion criteria should have been limited to only those whose Hb A1c value reflected current glycemic status, such as within the last 30 days, but 6 month criteria may have been sufficient. Narrowing inclusion criteria would have lengthened the timeframe needed to conduct the study to capture sample of 100 patients, though. For the purpose of this project, the primary goal was to screen as many type II diabetic patients as able to establish evidence for the need for screening, which was ultimately carried out in this project.
To complete this practice improvement project, great leadership and collaboration skills were needed. I conducted many communications between my academic chair and the practitioner from the study site to coordinate an appropriate topic, method and design for the project. I had to communicate effectively with clinic providers and staff in order to receive approval to perform the study and then to ensure the needs of the project were met. I frequently reassessed provider and staff satisfaction to ensure that their flow was not interrupted. At the conclusion of the study, I demonstrated leadership by presenting the results to the providers and proposing change in the practice.

**Outcomes**

The main purpose of this project was to determine the prevalence of depression among type II diabetic patients in this clinic and reinforce the need for screening. The results indicate that nearly a quarter of the type II diabetic patients at the Oregon City clinic have at least mild depressive symptoms. This is a substantial percentage of the population whose depression is not adequately being treated and represents strong evidence for regular screenings for depression in these patients.

Because of this project, approximately one-tenth of the clinic’s population of type II diabetics was screened for depression. Their results were recorded as part of their medical record for future comparison. All providers were educated regarding the merits of screening and given copies of the depression screening tool to use for follow-up visits. There are not specific clinical policies or guidelines in place at the clinic for care of diabetic patients, but the providers will consider using the screening tool in their patients during regular diabetic visits.

**Limitations**
This was a pilot study, so the following limitations were a problem. The controlling factors chosen for the correlation analysis were not comprehensive. Other factors that could have contributed to the presence of current depression were histories of past depression or family history of depression, but data on these variables were not collected. People that were currently being treated for depression were included in the screening as well. I chose not to collect this historical data because of its sensitive nature. Patients may have been less likely to participate if mental health information was recorded from their chart. Additionally, collecting data regarding diabetic management, specifically whether the patient was using insulin or not, as well as the presence of any diabetic complications, would have been other possible factors to control for as they may indicate poorer control or greater burden of the disease on the patient.

There are limitations with using a self-administered questionnaire. It requires that the subject have the comprehension or ability to fill it out independently. In order to not systematically exclude these patients from participation, I would read the survey and circle the answers to the subject’s responses if the subject was unable to see the page or write legibly. My presence in these instances may have impacted the answers given on the survey. I otherwise left the room while the participants filled out the questionnaire. Based on the interview and consenting process that preceded giving the patients the questionnaire, no one that filled it out lacked the mental capacity to comprehend the questions.

As mentioned earlier, over half of participants’ Hb A1c’s were not collected within 30 days of the depression screening. A better-designed study would have the Hb A1c’s drawn on the same day that the depression screening was administered for consistency. Given the nature of this project and lack of funding for laboratory testing, though, I had to use existing data, which may have affected the correlational findings because the sample size was too small.
The study was a convenience sample, which limits the statistical strength of the project. Patients were non-randomly asked to participate based on them presenting for their appointment. Therefore, this may not be a representative sample because the patients coming in for appointments may have a higher acuity requiring more frequent routine care, or may represent a sample of patients that tend to be motivated about their health.

The small sample size and lack of diversity of gender and race/ethnicity also affected the strength and ability to extrapolate the results of this study outside of this clinic. The project was conducted over a short period (8 weeks), which is a relatively small snapshot in time. Finally, as stated above, data collection in the winter months may have exaggerated the depression prevalence because of seasonal depression or worsening of depression in the darker, colder months (Harmatz et al., 2000; Oyane et al., 2008).

Conclusion

This clinical inquiry project demonstrated the importance of screening the diabetic patient for depression. A major proportion of these patients suffered from depression, as evidenced by the results of this study. Through self-administered questionnaires, which have proved to be an accurate and efficient method of screening (Kroenke, Spitzer, & Williams, 2001), providers were given an objective measure to document and use as a gauge for treatment success.

Additionally, although not statistically significant, a relationship between glycemic control and depression symptom severity seems likely. This adds to the body of literature already present regarding this correlation. Providers, should therefore, consider incorporating depression care along with their routine diabetic care to hopefully decrease the risk of poor outcomes that are likely if depression remains untreated (Wagner et al., 2001).
Future studies should be expanded to include larger sample sizes and across multiple site locations. Data collection should be captured over a longer period of time. Overall, the structure of this project offers a guide to other practitioners who want to establish a need for routine depression screening and treatment in type II diabetic patients.
References


Centers for Disease Control and Prevention Division of Diabetes Translation. (October, 2010).


Georgiades, A., Zucker, N., Friedman, K. E., Mosunic, C. J., Applegate, K., Lane, J. D., . . .  


Harmatz, M. G., Well, A. D., Overtree, C. E., Kawamura, K. Y., Rosal, M., & Ockene, I. S.  


TITLE: Clinical Inquiry Project: Type II Diabetes and Depression in Adults from One Primary Care Practice

PRINCIPAL INVESTIGATOR: Cheryl Wright, PhD, FNP-C (503) 494-3828

CO-INVESTIGATORS: Christine Ernst, RN, MN, FNP-BC (585) 739-1755

This form contains important information about the study in which you are being invited to participate. Please read the form carefully, ask questions of the investigators or others who are obtaining your consent to participate in the study, and take time to think about your participation. You may want to discuss the study with your family or friends before agreeing to be in the study.

What is the purpose of this study?

The purpose of this study is to:
(a) determine how many people have both type II diabetes and depressive symptoms and
(b) if there is a relationship between average glucose values and level of depressive symptoms.

What is required to participate in this study?

To qualify for this study, you must meet the following criteria:

1. Be diagnosed with Type II Diabetes Mellitus
2. Have a blood test (Hemoglobin A1c) value in your medical record from the last 6 months
3. Be 18 years old or older
What can I expect as a study participant?

We will ask you to fill out a questionnaire in an exam room. This study will take approximately 5-10 minutes to complete. Once you are finished, that completes your task for the study.

We will also be reviewing your medical record for this study.

If you have any questions regarding this study now or in the future, contact Cheryl Wright (503) 494-3828

What effect will this study have on my care?

There will be no effect on standard care. Being in this study will not affect any care that you might receive at OHSU.

How will my privacy be protected?

We will protect your privacy in the following ways:

1. Your name or other protected information will not be used. Instead, we will identify you by a number (ex. 101)
2. Only the researchers, Cheryl Wright & Christine Ernst, will be able to access your information.
3. The deidentified patient data will be kept on a password protected computer. The questionnaires will be kept in a locked file cabinet in a locked office at the OHSU School of Nursing.

The specific health information we will collect from you will be limited to your responses to questions in a questionnaire, patient demographic data, and hemoglobin A1c lab values. The purposes of our use and disclosure of this health information are described in the Purpose section of this Consent & Authorization Form.

The persons who are authorized to use and/or disclose your health information are all of the investigators who are listed on page one of this consent form and the OHSU Institutional Review Board.

The persons who are authorized to receive this information are the Office for Human Research Protections as required for their research oversight and public health reporting in connection with this research study.

This authorization will expire and we will no longer keep protected health information that we collect from you in this study after study is completed.
What are the possible risks of participating in this study?

Although we have made every effort to protect your identity, there is a minimal risk of loss of confidentiality.

What are the possible benefits of participating in the study?

You may or may not personally benefit from being in this study. However, by serving as a subject, you may help us learn how to benefit patients in the future.

Costs

There will be no cost to you or your insurance company to participate in this study. You will not be paid for participating.

Liability

If you believe you have been injured or harmed while participating in this research, contact Cheryl Wright (503) 418-2248.

You have not waived your legal rights by signing this form. Any claim you make against the Oregon Health & Science University may be limited by the Oregon Tort Claims Act (ORS 30.260 through 30.300). If you have further questions on this subject, please call the OHSU Research Integrity Office at (503) 494-7887.

What are my rights as a participant?

If you have any questions regarding your rights as a research subject, you may contact the OHSU Research Integrity Office at (503) 494-7887.

You do not have to join this or any research study. If you do join, and later change your mind, you may quit at any time. If you refuse to join or withdraw early from the study, there will be no penalty or loss of any benefits to which you are otherwise entitled.

You have the right to revoke this authorization and can withdraw your permission for us to use your information for this research by sending a written request to the Principal Investigator listed on page one of this form. If you do send a letter to the Principal Investigator, the use and disclosure of your protected health information will stop as of the date he/she receives your request. However, the Principal Investigator is allowed to use information collected before the date of the letter or collected in good
faith before your letter arrives. Revoking this authorization will not affect your health care or your relationship with OHSU. Your information will be de-identified. Please be advised that once this occurs, it will be impossible to remove your information from the study because the researcher will have no way to link the information to a specific subject.

The information about you that is used or disclosed in this study may be re-disclosed and no longer protected under federal law. However, federal or state law may restrict re-disclosure of HIV/AIDS information, mental health information, genetic information and drug/alcohol diagnosis, treatment, or referral information. OHSU tries to protect against re-disclosure without your permission by being very careful in releasing your information.

If the researchers publish the results of this research, they will do so in a way that does not identify you unless you allow this in writing.

Your health care provider may be one of the investigators of this research study, and as an investigator is interested in both your clinical welfare and in the conduct of this study. Before entering this study or at any time during the research, you may ask for a second opinion about your care from another doctor who is in no way involved in this project. You do not have to be in any research study offered by your physician.

You may be removed from the study if the investigator stops the study or you do not follow instructions.

By answering the questionnaire you may find out that you have a positive screen for depression. If you feel uncomfortable proceeding with the study, please let the investigator know and you will be withdrawn and your data will not be used.

If you choose to withdraw, there will be no consequence to you or your care.

To participate in this study, you must read and sign this consent and authorization form. If you withdraw your authorization for us to use and disclose your information as described above, you will be withdrawn from the study.

We will give you a copy of this form.
**SIGNATURES:**

Your signature below indicates that you have read this entire form and that you agree to be in this study.

**OREGON HEALTH & SCIENCE UNIVERSITY**
**INSTITUTIONAL REVIEW BOARD**
**PHONE NUMBER (503) 494-7887**
**CONSENT/AUTHORIZATION FORM APPROVAL DATE**

**Nov. 3, 2011**

**Do not sign this form after the**
Expiration date of: **11/02/2012**

_______________________________  _______________________
Signature of Subject                Date
(or Parent/Guardian if subject is under 18)

_______________________________
Printed Name of Subject
(or Parent/Guardian if subject is under 18)

_______________________________  _______________________
Signature of Person Obtaining Consent                Date

_______________________________
Printed Name of Person Obtaining Consent
Appendix B

OHSU School of Nursing
IRB # 7937

Demographic Survey

Instructions: Please circle the answer that best describes you.

1. What is your gender?
   a. Male
   b. Female
   c. Other

2. What is your highest level of education?
   a. Did not finish high school
   b. High School
   c. 2-year college degree
   d. 4-year college degree
   e. Graduate school degree
   f. Post-graduate degree

3. What is your race/ethnicity?
   a. Hispanic
   b. White, Non-Hispanic
   c. African American
   d. Asian/Pacific Islander
   e. Other
### PATIENT HEALTH QUESTIONNAIRE-9 (PHQ-9)

Over the **last 2 weeks**, how often have you been bothered by any of the following problems?

*Use "→" to indicate your answer*

<table>
<thead>
<tr>
<th>Problem</th>
<th>Not at all</th>
<th>Several days</th>
<th>More than half the days</th>
<th>Nearly every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Little interest or pleasure in doing things</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. Feeling down, depressed, or hopeless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. Trouble falling or staying asleep, or sleeping too much</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. Feeling tired or having little energy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. Poor appetite or overeating</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. Feeling bad about yourself — or that you are a failure or have let yourself or your family down</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. Trouble concentrating on things, such as reading the newspaper or watching television</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. Moving or speaking so slowly that other people could have noticed? Or the opposite — being so fidgety or restless that you have been moving around a lot more than usual</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. Thoughts that you would be better off dead or of hurting yourself in some way</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

For office coding: $0 + ____ + ____ + ____$

**= Total Score: ____

If you checked off any problems, how difficult have these problems made it for you to do your work, take care of things at home, or get along with other people?

<table>
<thead>
<tr>
<th>Difficulty Level</th>
<th>Not difficult at all</th>
<th>Somewhat difficult</th>
<th>Very difficult</th>
<th>Extremely difficult</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
</tbody>
</table>

Developed by Drs. Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke and colleagues, with an educational grant from Pfizer Inc. No permission required to reproduce, translate, display or distribute.
### Table 1. Demographic Frequencies

<table>
<thead>
<tr>
<th>Demographic Variable</th>
<th>Number of participants (n=96)</th>
<th>Percentage of participants (100%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (64.9 ± 12.76)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18 – 40</td>
<td>2</td>
<td>2.1</td>
</tr>
<tr>
<td>41 – 60</td>
<td>31</td>
<td>32.3</td>
</tr>
<tr>
<td>61 – 80</td>
<td>48</td>
<td>50.0</td>
</tr>
<tr>
<td>81 - 100</td>
<td>15</td>
<td>15.6</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>42</td>
<td>43.8</td>
</tr>
<tr>
<td>Female</td>
<td>54</td>
<td>56.3</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Did not finish HS</td>
<td>9</td>
<td>9.4</td>
</tr>
<tr>
<td>High School</td>
<td>52</td>
<td>54.2</td>
</tr>
<tr>
<td>2-year degree</td>
<td>20</td>
<td>20.8</td>
</tr>
<tr>
<td>4-year degree</td>
<td>12</td>
<td>12.5</td>
</tr>
<tr>
<td>Graduate degree</td>
<td>3</td>
<td>3.1</td>
</tr>
<tr>
<td><strong>Race/Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>89</td>
<td>92.7</td>
</tr>
<tr>
<td>Other (Black, Hispanic, Asian, Pacific-Islander, Other)</td>
<td>7</td>
<td>7.3</td>
</tr>
</tbody>
</table>
Table 2. Demographic and clinical characteristics of those who screened positive for depression versus those who did not (N=96)

<table>
<thead>
<tr>
<th></th>
<th>Depressed</th>
<th>Not depressed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N</strong></td>
<td>23 (24%)</td>
<td>73 (76%)</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;65 yo</td>
<td>13 (27.1%)</td>
<td>35 (72.9%)</td>
</tr>
<tr>
<td>&gt;65 yo</td>
<td>10 (20.8%)</td>
<td>38 (79.2%)</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>17 (31.5%)</td>
<td>37 (68.5%)</td>
</tr>
<tr>
<td>Male</td>
<td>6 (14.3%)</td>
<td>36 (85.7%)</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Did not finish HS</td>
<td>0 (0%)</td>
<td>9 (100%)</td>
</tr>
<tr>
<td>High School</td>
<td>15 (28.8%)</td>
<td>37 (71.2%)</td>
</tr>
<tr>
<td>2-year degree</td>
<td>3 (15%)</td>
<td>17 (85%)</td>
</tr>
<tr>
<td>4-year degree</td>
<td>4 (33.3%)</td>
<td>8 (66.7%)</td>
</tr>
<tr>
<td>Graduate degree</td>
<td>1 (33.3%)</td>
<td>2 (66.7%)</td>
</tr>
<tr>
<td><strong>Race/Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>21 (23.6%)</td>
<td>68 (76.4%)</td>
</tr>
<tr>
<td>Other (Black, Hispanic, Asian/Pacific-Islander, Other)</td>
<td>2 (28.6%)</td>
<td>5 (71.4%)</td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;30 kg/m2</td>
<td>6 (16.7%)</td>
<td>30 (83.3%)</td>
</tr>
<tr>
<td>≥30 kg/m2</td>
<td>17 (28.3%)</td>
<td>43 (71.7%)</td>
</tr>
<tr>
<td><strong>Hb A1c</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percentile</td>
<td>Cases (Percentage)</td>
<td>Total (Percentage)</td>
</tr>
<tr>
<td>------------</td>
<td>-------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>&lt;7%</td>
<td>14 (26.9%)</td>
<td>38 (73.1%)</td>
</tr>
<tr>
<td>≥7%</td>
<td>9 (20.5%)</td>
<td>35 (79.5%)</td>
</tr>
</tbody>
</table>
Figure 1. **Histogram of BMI values**

- Mean = 33.42
- Std. Dev. = 6.962
- N = 96
Figure 2. *Histogram of Hb A1c values*

Mean = 7.1
Std. Dev. = 1.254
N = 96
Figure 3. Scatter plot of depression score with Hb A1c
Executive Summary
Christine Ernst, MN, FNP-BC
Doctor of Nursing Practice Candidate
OHSU School of Nursing
Clinical Inquiry Project: Type II Diabetes and Depression

Purpose: (1) To determine the prevalence of depression among adult type II diabetics and (2) assess if a relationship exists between glycemic control and depressive symptoms.

Background: Type II diabetes mellitus is a serious chronic illness that, when poorly controlled, can have significant lifelong effects. Depression often occurs as a comorbid condition. Despite uncertain causality, studies have shown that screening and detecting depression among diabetics may lead to improved health outcomes.

Methods: A convenience sample of 96 adult type II diabetics from one primary care clinic were screened for depression using the Patient Health Questionnaire (PHQ-9). Scores greater than nine were considered positive screenings to determine prevalence. A partial correlation was used to assess a relationship between hemoglobin A1c (Hb A1c) scores in the last 6 months with PHQ-9 scores, controlling for demographic and patient data.

Outcomes: Prevalence of depression in adult type II diabetics was 24%. Controlling for age, gender, education, race/ethnicity, and body mass index, Hb A1c and depression did not have a significant correlation ($r=.091$, $p=.392$). In post-hoc analysis, when using only participants with current Hb A1c values (within the last month, $n=43$), results showed a stronger correlation, but were not statistically significant ($r=.31$, $p=.06$).

Conclusion: In this sample, approximately 1 in 4 adults with T2DM had at least mild depressive symptoms. There is some evidence of a relationship between glycemic control and severity of depressive symptoms. This pilot study has limited power and generalizability outside the clinic because of its small, non-random sample from one site. This study demonstrates that routine depression screening may be beneficial to detect current depression in type II diabetics, and may be correlated with poor glycemic control.

Study’s Strengths: The study was able to demonstrate some evidence in favor of routine screening of current depression for type II diabetic patients and was completed with minimal cost to the clinic and with little impact to the clinic staff or patients’ visit time. It adds to the body of literature supporting a possible relationship between worsening glycemic control and level of depression. Finally, it depicts the capabilities of a doctorally-prepared nurse practitioner to carry out a quality improvement project within a clinic setting.