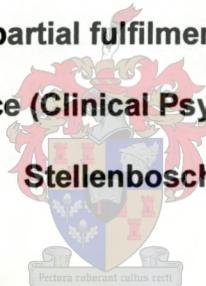


**A COGNITIVE CONCEPTUALIZATION OF DEPRESSION
IN ADULTS WITH DIABETES MELLITUS**

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degree of Master of Science (Clinical Psychology) at the University of
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DECLARATION

I, the undersigned, declare that the work contained in this assignment is my own original work and has not previously, in its entirety or in part, been submitted at any university for a degree.

B.S. Drake

OPSOMMING

Individue wat met diabetes mellitus gediagnoseer word, toon 'n verhoogde risiko om depressie te ontwikkel. Volgens die literatuur word depressie in diabetes mellitus geassosieer met 'n swakker lewensgehelte, swakker nakoming van behandeling, swakker aanpassing by diabetes, swakker glisemie-kontrole, en 'n verhoogde risiko om diabetes verwante komplikasies te ontwikkel. Hoewel die rol van bepaalde psigososiale veranderlikes in die ontstaan en instandhouding van depressie reeds ondersoek is, is min nog gedoen oor 'n konseptualisering van die assosiasie tussen diabetes en depressie. Hierdie werkstuk handel oor 'n konseptualisering van die verband tussen diabetes en depressie, gebaseer op Beck (1967, 1979) se kognitiewe model van depressie. Die konseptualisering dien as 'n raamwerk om hierdie verband te verstaan en toekomstige navorsing hieroor te rig.

ABSTRACT

Individuals diagnosed with diabetes mellitus are at an increased risk for developing depression. According to the literature, depression in diabetes mellitus has been associated with a poorer quality of life, poorer regimen adherence, poorer adjustment to diabetes, poorer glycaemic control, and an increased risk of developing diabetes related complications. While the role of certain psychosocial determinants in the onset and maintenance of depression has been investigated, mental health professionals and researchers have neglected the task of conceptualizing the relationship between depression and diabetes from a psychological perspective. This assignment presents a psychological conceptualization of the relationship between diabetes and depression, using Beck's (1967, 1979) cognitive model of depression as a framework. This conceptualization may serve as a means of theoretically understanding the relationship between these two conditions and as a framework in directing future research on this relationship.

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Dedicated in loving memory of the late Kirtesh Dullabh.

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1. Diabetes and depression

Diabetes Mellitus is a chronic metabolic disorder, characterized primarily by elevated blood glucose levels. Type 1 diabetes, comprising roughly 10% of all cases, results from an insufficient supply of endogenous insulin, whereas type 2 diabetes, which comprises the remaining 90% of all cases diagnosed, is characterized by insulin deficiency and/or insulin resistance (American Diabetes Association, 2003xb). It is now generally accepted that glycaemic control is most easily achieved through a combination of pharmacotherapy, dietary modifications and regular physical activity. Both type 1 and type 2 diabetes require consistent, intensive management if the numerous microvascular and macrovascular complications associated with poor glycaemic control, are to be prevented or controlled (American Diabetes Association, 2003xa; 2003xb). In addition to the numerous physical comorbid conditions associated with diabetes mellitus, an increased prevalence for a variety of psychiatric disorders among diabetics has also been noted (Rubin & Peyrot, 2001).

The literature suggests that diabetics are at an increased risk for developing depression (Gavard, Lustman, & Clouse, 1993). A review by Gavard et al. (1993) suggests prevalence rates ranging from 8.5% to 27.3%, with a mean of 14% derived from controlled studies. In addition, Garvard et al. (1993) estimated that at any one time, 32.4% of adult diabetics may present with clinically significant depressive symptoms. This association is noteworthy as a result of the

potentially pervasive impact of depression or depressive symptoms on quality of life, diabetes management or regimen adherence (McGill et al., 1992), adjustment (Lernmark, Persson, Fishert, & Rydelius, 1999), and glycaemic control (Lustman, Griffith, & Clouse, 1997; Lustman, Griffith, Clouse, & Cryer, 1986; Lustman et al., 1997; Lustman, Griffith, Freedland, & Clouse, 1997). Furthermore, depression has also been associated with an increased risk of developing diabetes-associated complications such as cardiovascular disease (Lloyd, Wilson, & Forrest, 1997) and retinopathy (Kovacs, Mukerji, Drash, & Lyengar, 1995). These associations are also confirmed by numerous studies that have demonstrated the hypoglycaemic effect associated with the treatment and remission of depression among diabetics (Lustman, Griffith, Freedland, Kissel, & Clouse, 1998; O'Kane, Wiles, & Wales, 1994; Potter Van Loon et al., 1992).

Although depressive symptomatology may at times be overlooked by physicians (Van der Does et al., 1996), the potentially adverse consequences of depression in relation to glycaemic control and its correlates have not been ignored, and both psychotherapy (Lustman et al., 1998) and pharmacotherapy (Goodnick, Henry, & Buki, 1995; O'Kane et al., 1994; Potter Van Loon et al., 1992) interventions have been researched and utilized in the treatment of depression among diabetics. Talbot and Nouwen's (2000) extensive review of the relationship between depression and diabetes in adults provides a summary of those investigations which have attempted to illicit the etiological link between depression and diabetes, by focusing either on the biochemical changes

associated with, or the psychosocial demands imposed by, the illness and/or its treatment. Talbot and Nouwen (2000) concluded that "the cause of major depressive disorder (MDD) in individuals with diabetes is not causally independent of diabetes", but "rather represents a complex phenomenon resulting from interactions between genetic, biologic and psychosocial factors, which may account for the recurrence and longer duration of MDD" (p. 1560).

A number of studies have focused on the environmental variables or stressors associated with the onset of depression in diabetics, yielding in most cases, results that both support and contradict these associations. This may suggest that while a variety of stressors may act as determinants of depression among diabetics, the presence of a precipitating stressor alone is not always sufficient to ensure the onset of depressive symptoms or MDD. This phenomenon is similar to that found within the general population, and may be attributed to two possible explanations. The first being that the onset of depression may be conceptualized (Beck, 1967; Talbot & Nouwen, 2000) as the result of the interaction of a variety of genetic, biological and psychosocial factors, and that the presence of a stressor can therefore not be expected to always be followed by the onset of a depressive episode. A second explanation, and one that will be highlighted extensively throughout this assignment, is that the majority of investigations examining the environmental or psychosocial determinants of depression in diabetes have rendered contradictory results, possibly as a result of their almost exclusive focus on variables, which would ordinarily be conceptualized as

precipitating psychosocial determinants of depression. The role of predisposing psychological factors, which are significantly more difficult to assess, has received little attention thus far. A more comprehensive understanding of the relationship between depression and diabetes would thus require a focus on both potential precipitating psychosocial factors as well as an understanding of the impact of predisposing variables. The reported prevalence rates of depression among diabetics (Gavard et al., 1993), together with the contradictory findings reported by researchers interested in the psychosocial determinants of depression among diabetics, are sufficient to highlight the need for a psychological conceptualization of depression among diabetics.

Only through the provision of a psychological conceptualization of depression in diabetes would we be able to theoretically speculate about an explanation for the increased prevalence of depressive disorders within this population. The conclusion drawn by Talbot and Nouwen (2000, p. 1558) that "analysis using the APA guidelines does not support the notion of a mood disorder caused by diabetes" (i.e. a general medical condition), further highlights the need for a psychological conceptualization of this relationship.

The objectives of this assignment are therefore (1) to provide a theoretical conceptualization of the association of depression and diabetes, (2) to use this conceptualization as a means of further understanding the increased prevalence, higher recurrence rate and longer duration of depressive episodes experienced

by diabetics, and (3) to provide a theoretical framework from which future research may be derived.

Controlled studies have demonstrated the efficacy of cognitive therapy in the treatment of major depressive disorder (see Dobson (1989) for a meta-analysis of the efficacy of cognitive therapy for depression). In addition, the theoretical principles of Beck's (1967, 1983) cognitive model of depression, on which cognitive therapy is based, have also been validated by both cross-sectional (Olinger, Kuiper, & Shaw, 1987; Wise & Barnes, 1986) and longitudinal investigations (Abela & D'Alessandro, 2002; Joiner, Metalsky, Lew, & Klocek, 1999; Kwon & Oei, 1992). While the efficacy of cognitive behavioural therapy (CBT) for the treatment of depression among diabetics has been demonstrated (Lustman et al., 1998), mental health professionals and researchers have neglected the task of conceptualizing the relationship between depression and diabetes from the perspective of cognitive theory. An overview of Beck's cognitive theory of depression (Beck, 1967) will thus be presented prior to conceptualizing depression in diabetes from this theoretical perspective.

2. Beck's cognitive model of depression

The empirically supported cognitive therapy approach towards treating depression is based on the underlying theoretical rationale that affect and behaviour are largely determined by the meanings that individuals draw from

their experiences within their environment, and that affective and behavioural responses are not merely a direct result of the onset of some antecedent event. The answer, in terms of cognitive theory, to understanding the onset of depressive symptoms associated with a particular event lies in understanding the intrapersonal processes, which account for the way in which meanings are extrapolated from experience. As a result, while an individual's emotional and behavioural responses to a particular stressor are of central importance to the diagnosis of depression, it is the cognitions with which they present that are seen as central to the formulation and treatment of the depressed patient. Beck (1984, p. 1113) has however acknowledged that "while cognitive phenomena may be seen as an integral part of depression, they are hardly capable of causing themselves", and should rather be seen as an important link in the development and maintenance of the observable signs and symptoms of depression, rather than as the sole etiological variable responsible for their onset. Beck (1983) conceptualized depression as the final common pathway of many converging variables. Any combination of predisposing and precipitating factors may contribute to the onset of depressive symptoms and the relative contributions of predisposing versus precipitating factors may also vary from case to case. This theoretical assumption would account for the way in which research, aimed at investigating the relationship between antecedent events or environmental stressors and the onset of depression, is often represented by studies both in support of and against such associations. This may be due to an overemphasis by researchers on precipitating variables with a subsequent lack of emphasis on

predisposing psychological factors. The following section will summarize the cognitive model of depression and will provide an understanding of the role of predisposing phenomena in the conceptualization of depression.

The cognitive model (Beck, 1967; Beck, Rush, Shaw & Emery, 1979) of depression makes use of three associated conceptual phenomena as a means of explaining the psychological substrate of depression: (1) the cognitive triad, (2) schemas, and (3) cognitive errors or faulty information-processing.

2.1. The cognitive triad

The cognitive triad (Beck, 1967; Beck et al., 1979) refers to the negatively orientated misperceptions and misinterpretations that depressed individuals present with, which results in negatively orientated thinking patterns about (1) the self, (2) one's situation or the world, and (3) the future. Depressed individuals view themselves "as defective, inadequate, diseased, or deprived" (Beck, 1967; Beck et al., 1979), and therefore attribute their unpleasant or undesirable experiences to "an innate psychological, moral or physical defect" (Beck, 1967; Beck et al., 1979), which is then further interpreted as evidence that they are undesirable, unlovable, and worthless. Such patients often underestimate their potential and/ or overestimate their deficiencies as a result, and ultimately believe that they lack the attributes essential to the attainment of happiness. The depressed person also sees the world as excessively demanding, unfair or

dismissive of him or her. Interactions with the world are often misinterpreted as representations of defeat or deprivation. As a result, the depressed person will often interpret events within his or her daily life in a biased manner, tailoring his perceptions to fit already preformed negative conclusions (Beck et al., 1979). The third component of the cognitive triad consists of a negative view of the future. Unremitting hardship, frustration, deprivation and failure are expected. The cognitive model therefore views the ongoing affective, motivational and behavioural symptoms of depression as a result of the activation of these negative thinking patterns. The content of the depressed individual's cognitions will be reflected by either or all of the above three themes. The presence of such depressive cognitions is thought to result from what Beck has termed faulty information-processing.

2.2. Faulty information-processing

It is the systematic errors in thinking of the depressed person which maintains their belief in the validity of their negative concepts about the self, the world and the future, despite the presence of contradictory evidence (Beck, 1967). In Beck's (1984) response to the paper by Simons, Garfield and Murphy (1984), he proposed that the site of action of both pharmacotherapy and cognitive therapy is the information-processing system, and suggests that "any intervention which affects information-processing will show both cognitive and biochemical changes" (p. 1113). Beck et al. (1979) described how the thinking patterns of depressed

individuals may be conceptualized as "primitive", as opposed to "mature" modes of organizing reality. Depressed individuals tend to make broad, global judgments about their experiences, and may often present with extreme, negative, categorical, absolute, fixed and judgmental means of processing information (Beck et al., 1979), arbitrary inferences, selective abstractions, overgeneralizations, and magnifications (Beck, 1967), which result in negative and extreme emotional responses. "More mature thinking automatically integrates life situations into many dimensions or qualities, in quantitative rather than qualitative terms, and according to relative rather than absolutistic standards" (Beck et al., 1979, p. 14).

2.3. Schemas

Early in life, individuals develop a wide variety of concepts and attitudes about themselves and the world. Some of these concepts are anchored to reality and form the basis for healthy personal adjustment. Others deviate from reality and create a vulnerability to possible psychological disorders (Beck, 1967).

One's self-concept consists of a cluster of attitudes about the self, some favourable, others not, which develops out of generalizations made about the self, on the basis of one's interactions with the environment, the attitudes and opinions communicated to one by others, and from one's identifications with key figures, such as parents, siblings and friends. Once formed, these attitudes or

concepts influence subsequent judgments (through which such attitudes are often further reinforced) and subsequently become more firmly set until they become structuralized as permanent formations in one's cognitive organization. These cognitive structures or schemas (Beck, 1967), in contrast to cognitive processes (which are transient), are thus deep (often unconscious), stable, organized, representations of past experiences, which provide the basis for screening, differentiating and coding environmental stimuli that impinge on a person, and which thereby mold data into cognitions (defined as any ideation with verbal or pictorial content). Examples of positive or self-enhancing attitudes associated with adaptive cognitive schemas include "I'm capable", "I'm attractive", "I can get what I want", "I can understand problems and solve them" (Beck, 1967, p.276). Negative or self-diminishing attitudes stemming from dysfunctional schemas include "I am weak", "I am inferior", "I am unlovable", "I can't do anything right" (Beck, 1967, p.276).

A schema may be inactive or latent for a long period of time. Schemas associated with particular events or circumstances will be activated by the onset of such an event or stressor. The kinds of schemas activated by and employed subsequent to the onset of the event, will determine the interpretations and personal meanings made, and the subsequent affective and behavioural responses observed. Beck (1967) suggested that in psychopathological states such as depression, patient's conceptualizations of specific situations are distorted to fit the prepotent dysfunctional schemas. It is thus these core

schemas which are believed to underlie the over generalized, negative, dichotomous, and dogmatic means of processing information and the subsequent cognitions associated with depression. Beck thus attributed vulnerability to depression to a constellation of enduring negative attitudes about the self, the world and the future. These attitudes, activated by an appropriate set of conditions then dominate the way in which information is processed and lead to the typical depressive symptoms observed.

3. A theoretical framework for understanding depression in diabetes, based on Beck's cognitive model of depression

Talbot and Nouwen's (2000) review of the link between depression and diabetes partly illustrated the way in which researchers, thus far, have attempted to uncover the psychosocial link between these two conditions. While some epidemiological work has been done and a few descriptive studies have been published which have linked certain psychosocial variables such as the diagnosis of diabetes (Kovacs, Obrosky, Goldston, & Drash, 1997; Palinkas, Barret-Connor, & Wingard, 1991), the onset of comorbid complications (Bernbaum, Alpert, & Kuckro, 1988; Peyrot & Rubin, 1989; Wuslin & Jacobson, 1989; Wuslin, Jacobson, & Rand, 1986; Wuslin, Jacobson, & Rand, 1993), illness intrusiveness (Devins, 1994; Karlson & Agardh, 1997; Talbot, Nouwen, Gingras, Belanger, & Audet, 1999), and lower levels of social support (Littlefield, Rodin, Murray, & Craven, 1990), with the onset of depressed mood among diabetics, little has

been done in an attempt to conceptualize the psychodynamic issues relating to the onset and maintenance of depression in diabetes. The objective of the following section is to combine what is already known about diabetes and depression, as reviewed in Talbot and Nouwen (2000), with Beck's cognitive model of depression in order to conceptualize the relationship between these two conditions.

Figure 1 provides a diagrammatic summary of a conceptualization of depression and diabetes from the perspective of cognitive theory, by illustrating and highlighting potential pathways between diabetes specific psychosocial variables and the activation of latent schemas or faulty information processing. Much of the research thus far has focused almost exclusively on the acute and chronic psychosocial stressors and potential biochemical determinants of depression among diabetics. The remainder of this paper will thus aim to illustrate the link between such variables and the activation of latent schemas and faulty information-processing. A proposal regarding the content of the depressed diabetic's cognitions about the self, the world and the future will also be presented.

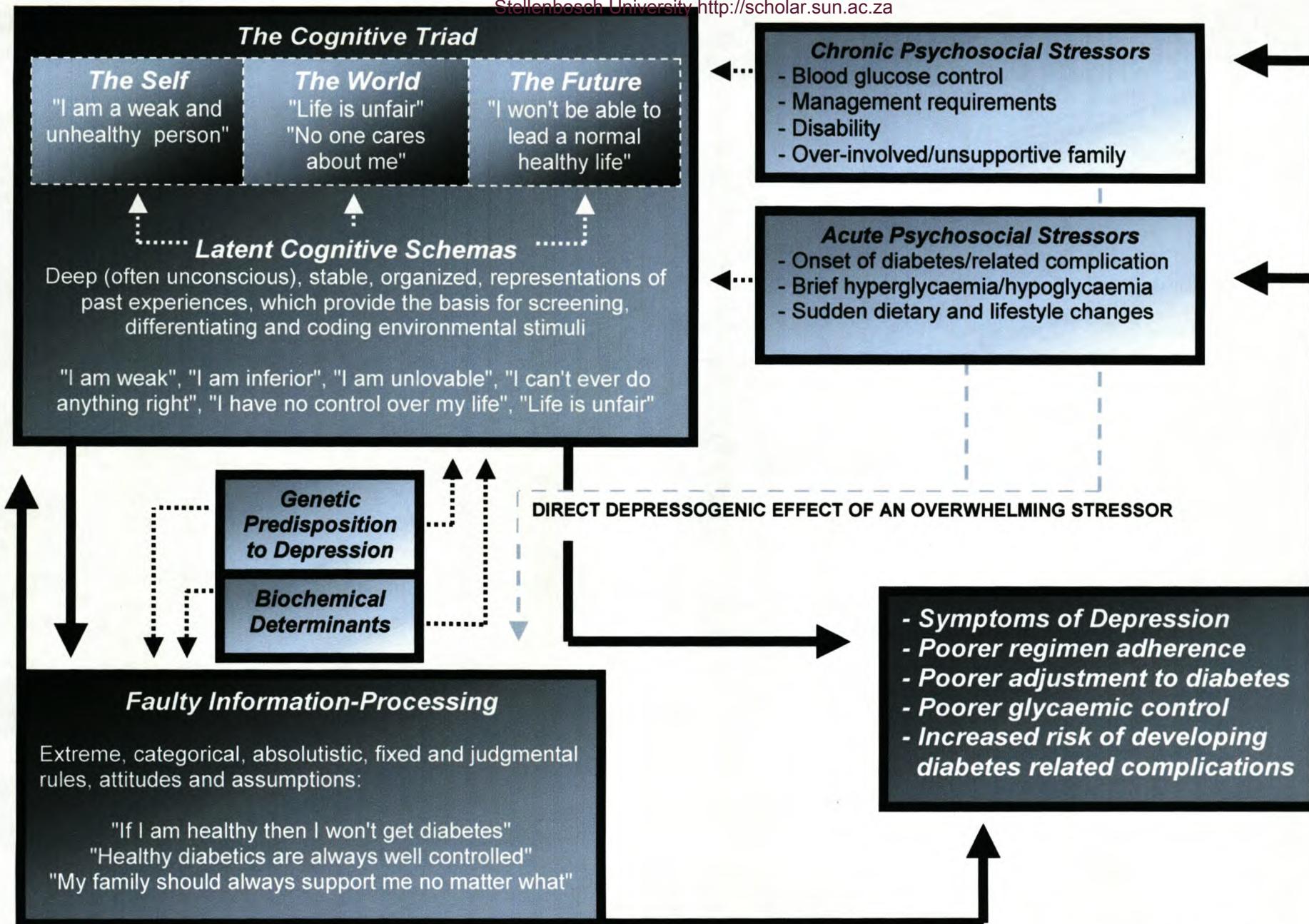


Figure 1. A diagrammatic summary of a theoretical conceptualization of depression in diabetes based on Beck's (1967, 1979) cognitive model of depression.

3.1. Relationship between precipitating and predisposing factors to depression in diabetes

Talbot and Nouwen (p. 1559, 2000) summarized and highlighted the role that certain psychosocial variables may play in the link between diabetes and depression. The theoretical implications of Beck's cognitive model would however suggest, that while precipitating psychosocial factors are certainly always present and associated with the onset of MDD or depressive symptoms, they are seldom the sole determinants thereof, and rather act as one aspect among a multitude of contributing factors. In addition, precipitating or mediating "depressogenic" factors such as poor social support, illness intrusiveness or an external locus of control, may themselves be symptomatic of an underlying premorbid psychological pathology, or may serve as evidence of underlying predisposing attitudes or depressogenic cognitive schemas. It would therefore follow that the clue to understanding and predicting the onset of depression among diabetics may lie in understanding both the influence of precipitating as well as predisposing factors (figure 1).

3.1.1 Precipitating factors

Both acute and chronic stressors may precipitate the onset of a major depressive episode. Beck (1967, p. 280) noted that an individual "may develop some form of psychological disturbance when exposed to an overwhelming stress, even if it

does not strike at a specific sensitivity". Diabetic individuals may thus develop depressive symptoms if the management requirements of their condition or the complications or difficulties associated with it are experienced as overly taxing. The unremitting difficulty associated with having to contend with unstable blood glucose levels, for example, may be an example of one such chronic stressor. Such an experience may easily leave an individual feeling helpless and demoralized and would not necessarily require the activation of some underlying cognitive vulnerability in order for depressive symptoms to develop (figure 1). The concept of illness intrusiveness (Karlson & Agardh, 1997; Talbot, et al., 1999; Devins, 1994) is considered as one of the most significant mediating factors in the relationship between such chronic disease variables and depressed mood (Talbot & Nouwen, 2000).

Acute stressors such as the onset of diabetes, severe hypoglycaemic episodes, the diagnosis of a comorbid condition associated with micro or macrovascular complications, or adverse social experiences associated with the diagnosis of diabetes may also precipitate the onset of a MDD. Newly diagnosed individuals are confronted with significant lifestyle changes and with new diabetes management requirements such as blood glucose testing and the administration of insulin or oral hypoglycaemic agents. These factors alone may have a significant impact on illness intrusiveness. The way in which newly diagnosed diabetics perceive the impact of their diagnosis or its implications may thus act as a critical determinant to the onset of depression. For example, if an individual

perceives their diagnosis as an indication of how they have failed to lead a healthy life, or if the management requirements of the condition are perceived as being too threatening, overly time consuming or unattainable, then such individuals may be at risk for developing a depressive disorder. Individuals who perceive their diagnosis as a "death sentence", or as "the end of a normal happy lifestyle" may also be at an increased risk for developing depression.

3.1.2 Predisposing factors

Do diabetics who develop depression carry a cognitive vulnerability towards developing depression, just as in the non-diabetic population? Beck's model would suggest that they do, and that much of the difference between those who develop depression and those who do not, lies in cognitive vulnerability.

Lustman, Clouse, Carney and Griffith (1987) found that depressed diabetics tend to have a positive family history for depression (27 %) in comparison to non-depressed diabetics who generally do not (3%). These statistics are in line with what one would expect from depressed patients who do not present with a chronic medical illness (Winokur & Pitts, 1965). Kovacs et al. (1997) also found that youths with type 1 diabetes who had mothers with a history of MDD, were at a greater risk of developing MDD themselves. These findings alone are indicative of the potential contribution of genetic vulnerability or a predisposition to the onset of depression. Cognitive theory would attribute a cognitive vulnerability or

predisposition to depression, to the presence of underlying depressogenic cognitive schemas, as is illustrated in figure 1, in addition to any genetic or physiologically based predispositions (Beck, 1983). The above-mentioned relationships clearly indicate the role of predisposing factors in the onset of depression. While none of the studies referenced in the above text assessed for cognitive vulnerability or the presence of underlying depressogenic schemas, it can be postulated that cognitive vulnerability may have acted as a contributing factor among those depressives who were predisposed to depression through a family history of depression.

What sort of schemas might be associated with the onset of depression among diabetics? Would such underlying predispositions include unique diabetes related dysfunctional schemas or would they be typical of the depressogenic schemas found within non-diabetic depressed populations? Investigations aimed at answering these questions would assist in the development of a more thorough psychodynamic conceptualization of depression in diabetes.

It is proposed that the experience of living with diabetes from a young age (pre-adolescence) may be associated with a predisposition to depression if the experience of living with diabetes for the young diabetic leads to the development of dysfunctional attitudes and core beliefs about the self, the world or the future. The development of generalized and stable attitudes about the self, the world and the future, would however normally precede the diagnosis of type 2 diabetes,

and type 1 diabetes if diagnosed during adolescence or early adulthood. The average age of onset of MDD among type 1 diabetics of 22.1 years (Lustman, Griffith & Clouse, 1988) is generally preceded by the average age of onset of diabetes (Talbot & Nouwen, 2000). This would suggest that if diabetics vulnerable to depression become depressed, it may be the result of the onset of diabetes or some other combination of precipitating variables, perhaps associated with diabetes (such as illness intrusiveness or an added vulnerability to depression due to alterations in biochemical systems), which may then lead to the activation of some already preformed cognitive schema, which itself may have developed completely independently of diabetes. This proposal would however require further empirical investigation if it were to be validated. Beck (1967, p.279) suggested that "a physical disease or abnormality may sometimes act as a precipitating event to depression", but that an individual "must be particularly sensitive to the situation and must have a pre-depressive constellation to react with a clinical depression".

3.1.3 Dysfunctional attitudes, rules and assumptions: evidence of faulty information-processing

Beck (1984, p. 1113) proposed that the site of action of both pharmacotherapy and cognitive therapy is the information-processing system, and suggested that "any intervention which affects information processing will show both cognitive and biochemical changes". Beck (1995) suggested that core beliefs (the content

of underlying schemas) and automatic thoughts are ultimately connected by certain intermediate beliefs which take the form of dysfunctional attitudes, rules and assumptions (see figure 1). It is in the attitudes, rules and assumptions of a depressed individual that one will find the evidence of faulty information-processing. Depressed diabetics may therefore present with attitudes, rules and assumptions which may be extreme, categorical, absolutistic, fixed and judgmental (Beck et al., 1979), thus leading to arbitrary inferences, selective abstractions, overgeneralizations, and magnifications (Beck, 1967). For example, an absolutistic attitude such as "I should be able to maintain good blood glucose control at all times", a categorical assumption such as "You cannot be healthy if you have a chronic illness", or a fixed and judgmental rule such as "A competent individual will always be able to avoid hypoglycaemia" may all demonstrate faulty information-processing and link situational or psychosocial variables with depressogenic schemas, thus resulting in or contributing toward depressive affect.

In summary, precipitating variables, which may present as a combination of genetic, biological and psychosocial factors, but which may, for example, include the chronic stressor of unstable blood glucose, may activate a latent depressogenic schema such as "I am a weak person", via a dysfunctional rule such as "If I am competent, then I will be able to maintain good diabetic control at all times". A hypothesis regarding the kinds of dysfunctional cognitions (re. the

self, the world and the future) with which a depressed diabetic may present is presented in the following section.

3.2 Events or experiences, which may be associated with dysfunctional cognitions about the self, the world, or the future

As discussed earlier, Beck (1967, 1979) proposed that depressed individuals' faulty information-processing, itself the result of the activation of an underlying schema(s) by a combination of precipitating factors, results in the negatively orientated misperceptions and misinterpretations about (1) the self, (2) one's situation or the world, and (3) the future. Although it is assumed that the depressed diabetic's cognitions would also be associated with negative and biased perceptions of the self, the world and the future, it is possible that the exact content of some depressed diabetic's cognitions may be specifically related to diabetes and therefore indicative of the subjective psychological impact of the condition. Other depressed diabetics may however present with cognitions completely unrelated to diabetes itself. It is proposed that by identifying the content of the depressive's cognitions, we may be better equipped to make assumptions about the relative contribution of this chronic condition to the onset of depression, from a psychosocial perspective. While it is probable that the underlying cognitive schemas of the depressed individual will be very similar, if not the same as the underlying schemas or core beliefs of a depressed individual not diagnosed with diabetes, it is speculated that the more superficial automatic

thoughts, attitudes and assumptions may be more specifically related to diabetes among some individuals. While diabetes related experiences may precipitate the onset of diabetes related depressive cognitions, and depressed mood, depressive cognitions may also be precipitated by depressed mood itself (Kwon & Oei, 1992; in Abela & D'Alessandro, 2002). In such a scenario, depressed mood would precipitate faulty information-processing, which itself would then result in the activation of latent schemas and/or associated depressive cognitions about the self, the world and the future.

The following section focuses on the link between specific precipitating factors and the kinds of diabetes related cognitions about the self, the world and the future, that depressed diabetics may present with, as presented in figure 1.

3.2.1 Events or experiences associated with dysfunctional cognitions about the self

Beck noted that cognitions about the self will only result in depressive affect if their underlying meaning points towards the individual's perception of themselves as helpless or unlovable (Beck, 1995).

Being diagnosed as diabetic may act as a strong precipitant to depressed mood (Kovacs et al., 1997). In investigating a sample of 1586 men and women, who were aged 50 years or older, Palinkas et al. (1991) found that individuals who

were aware of their diabetic status (n=93), presented with a 3.7 times higher prevalence of mild to severe depressive symptoms in comparison to those who were not yet aware of their diagnosis of diabetes (n=209), or in comparison to non-diabetics who may have been diagnosed with another chronic condition (n=1284). The newly diagnosed individual's awareness of their diagnosis (Palinkas et al., 1991) and their interpretations of what it means to have been diagnosed with diabetes may act as significant mediating variables in the link between diabetes and depression. Dysfunctional cognitions about the self such as "I am diabetic" and therefore "I am sick" and thus "I am different", "I am weak", "I am disabled", "I am defective", "I am undesirable" or "I am inadequate" may result. These may result from dysfunctional attitudes such as "If you are a strong, healthy and well adjusted person, then you wouldn't develop a chronic medical condition". Diabetics who have developed microvascular (retinopathy, neuropathy, nephropathy) or macrovascular complications (cardiovascular or cerebrovascular pathology) secondary to diabetes, may also present with the above-mentioned cognitions about the self. In addition, individuals diagnosed with such complications may also present with cognitions such as "I should have been able to prevent this complication" and "Because I did not, I am useless". The associations between such precipitants and the presence of dysfunctional cognitions may be mediated by the individual's perceived impact of diabetes, or illness intrusiveness (Devins, 1994; Karlson & Agardh, 1997; Talbot et al., 1999), which refers to the way in which the condition affects the individual's psychological, social, and physical well-being and functioning. Similar cognitions

about the self (as described above) may thus be associated with diabetes if it results in a significant change in the way in which the individual would ordinarily lead his or her life, especially if they perceive their new management requirements and lifestyle patterns as particularly different, embarrassing or limiting. Such experiences may lead to cognitions such as "I am a burden" or "I am not able to do what other people do and am therefore different and of less value as a person".

The continual daily challenges associated with blood glucose control and good diabetes management may also be associated with the above-described cognitions about the self, especially among brittle type 1 diabetics who may find it extremely difficult to prevent acute fluctuations in blood glucose, an experience which could easily be associated with cognitions such as "I should be able to control my blood glues at all times" but "no matter how hard I try, I can never get my blood glucose under control and therefore I am a failure". Difficulties or "failure experiences" associated with dietary or other lifestyle changes may further contribute to this experience. The association between more demanding regimens and lower levels of adherence, and depressive symptomatology (Padgett, 1993) and the possibility that such experiences may easily lead to the development of learned helplessness (Cox, Gonder-Frederick, & Saunders, 1991) would support this suggestion.

The way in which parents, doctors and other health professionals respond to poor glycaemic control may also have a significant impact on the way in which patients make inferences about themselves in response to their difficulties with achieving good glycaemic control. An autocratic, dogmatic, and critical attitude by health professionals may result in poorly controlled patients thinking "I should be able to control my blood glucose but because I cannot get my blood glucose right, I'm a failure" or "I'm a disappointment".

3.2.2 Events or experiences associated with dysfunctional cognitions about the world

The diagnosis of diabetes, the difficulties or "failure experiences" associated with its management, and the onset of comorbid complications may all leave patients feeling frustrated and irritated with what they perceive as a never-ending battle with diabetes and life. Cognitions such as "bad things only happen to bad people" and having been diagnosed with diabetes just shows that "no matter what I do, I am destined to suffer", or "life is unfair", may result. These individuals may often be left feeling defeated, deprived and helpless in a world, which they may experience as unfair and overly demanding. Individuals who struggle to maintain glycaemic control, despite the efforts of the health professionals involved in their care, may also begin to perceive those involved in the management of their diabetes as "untrustworthy and unreliable". The way in which health professionals interact with their patients will also have a significant impact on how

well supported and understood patients feel (Rubin & Peyrot, 2001). Rubin and Peyrot (2001, p. 470) focused attention on the distressing influence that family members and friends may have if they are perceived as either unsupportive of the patient's efforts to manage diabetes or as overly critical and intrusive. These experiences may leave patients feeling isolated, frustrated, sad or angry.

Thoughts such as "no one really understands" or "no one cares" may be evoked. Such experiences may thus also have a significant effect on how people view their world of relationships.

3.2.3 Events or experiences associated with dysfunctional cognitions about the future

Individuals who struggle with diabetes management, from glycaemic control to the adherence to dietary and exercise prescriptions, may present with future orientated cognitions regarding their condition such as "I will never be able to control my blood glucose" or "I have no control over my blood glucose or whether or not I develop complications" and that "there is no hope for the future".

Diabetes related experiences, which result in a reduction in perceived control or a sense of helplessness and hopelessness, may thus be associated with dysfunctional future orientated cognitions. Cox et al. (1991) speculated that because strict adherence does not guarantee good diabetes control or the avoidance of complications, this is fertile ground for the development of learned helplessness, itself associated with dysfunctional future orientated cognitions. As

a result, idealistic, dogmatic, strict regimen and management goals associated with an attitude such as "I should be able to maintain good blood glucose control at all times" may add to the frequency of perceived failure experiences and the development of associated depressogenic cognitions. The diagnosis of diabetes itself may result in negative expectations by patients such as "I will never be able to lead a normal life again, and that's terrible", "I am destined to develop diabetes related complications" or "I am destined to be sick for the rest of my life". The onset or threat of comorbid complications could leave individuals with thoughts such as "I have no control over my diabetes or my life".

4 An explanation of the increased prevalence, higher recurrence rate and longer duration of MDD among diabetics

If diabetics who become depressed present with the same cognitive vulnerability or underlying dysfunctional schemas, which have developed over time independent of diabetes, as non-diabetics, then to what do we attribute the increased prevalence, higher recurrence rate and longer duration of depression among individuals with diabetes? One potential explanation for this, would be that these individuals, already predisposed to developing depression, may experience higher recurrence rates and longer depressive episodes as a result of the presence of a set of chronic and unremitting stressors, similar to what Beck (1967) defined as psychological strain, which are perhaps unique to the presence of a chronic medical condition such as diabetes. A similar argument may thus

account for the higher prevalence rates of unipolar mood disorders among diabetics. This does not suggest that diabetes is causally associated with the onset of depression. What it does suggest is that those diabetics, who already carry a vulnerability to depression, as with most other non-diabetic depressed individuals, may be at an increased risk of developing depression as a result of the greater number of precipitating factors available for the activation of an underlying depressogenic schema. It is important to highlight that both psychosocial as well as biochemical variables may act as precipitating or maintaining factors in the onset and maintenance of depression. Once a dysfunctional schema is activated, the individual is more likely to process information in a negative, absolutistic, dichotomous, biased way, which would result in the kinds of cognitions, which are associated with the affective, motivational and behavioural symptoms of depression, which themselves then further activate these already active schemas, thus leading to the downward spiral of depression (Beck, 1967). In addition, the manifestation of such symptoms may then further aggravate glycaemic control (Lustman et al., 1997; Lustman, Griffith et al., 1997; Lustman, Griffith, Clouse et al., 1997; Lustman et al., 1986) and other diabetes related variables, thereby further compounding the depressive reaction and increasing the duration of the depressive episode.

Does this mean that diabetics may require more intensive treatment as a result of the potentially greater number of factors, which may add to the onset and maintenance of the condition? The above speculations would therefore suggest

that an optimal treatment for depression among diabetics would be aimed at both symptom alleviation and at reducing their vulnerability to depression.

5. Summary and recommendations

Depression in diabetes has been associated with a poorer quality of life, poorer regimen adherence, poorer adjustment to diabetes, poorer glycaemic control, and an increased risk of developing diabetes related complications. The reasons for the higher prevalence rates, recurrence rates and longer duration of depressive episodes experienced by those diagnosed with diabetes mellitus are, however, yet to be fully understood.

Talbot and Nouwen (2000) reviewed the link between diabetes and depression. They concluded that, "analysis using the APA guidelines does not support the notion of a mood disorder caused by diabetes" (i.e. a general medical condition) (p. 1558). While the role of certain psychosocial determinants in the onset and maintenance of depression has been investigated, mental health professionals and researchers have neglected the task of conceptualizing the relationship between depression and diabetes from a psychological perspective. The first objective of this assignment was thus to highlight the need for a psychological conceptualization of the relationship between diabetes and depression. A theoretical conceptualization is necessary if the link between diabetes and depression is truly to be understood, and is essential in directing future research.

Beck's cognitive model of depression was utilized as a framework for understanding and conceptualizing the relationship between diabetes and depression among adults. This model of depression conceptualizes depression as a result of the interaction of a variety of genetic, biological and psychosocial precipitants, which together may activate some latent cognitive schema(s), resulting in faulty information-processing, which would subsequently lead to the negatively orientated misperceptions and misinterpretations about (1) the self, (2) one's situation or the world, and (3) the future. Unlike previous research associated with diabetes and depression, Beck's cognitive model introduces the concept of depressogenic schemas, and thereby highlights the role of predisposing variables in the onset and maintenance of depression. Beck's concept of faulty information-processing also provides a means of understanding the mechanism involved in the onset and maintenance of a depressive episode and thus provides the link between a precipitant and the onset of a depressive episode. This link may be seen in the dysfunctional attitudes, rules and assumption with which depressed diabetics present.

In conceptualizing the relationship between diabetes and depression (see figure 1), it was postulated that both chronic variables such as the daily hassles associated with diabetes management, chronically unstable blood glucose, or disability, as well as acute diabetes related variables such as the onset of diabetes, significant dietary and other lifestyle modifications, the onset of a comorbid complication, or a severe hypoglycaemic episode, may act as

precipitating variables in the activation of underlying dysfunctional cognitive schemas and faulty information-processing. Although it is assumed that the depressed diabetic's cognitions would also be associated with negative and biased perceptions of the self, the world and the future, it is proposed that the exact content of some depressed diabetic's cognitions may be specifically related to diabetes and therefore indicative of the subjective psychological impact of the condition. A number of diabetes specific situational variables and the respective cognitions about the self, the world and the future that may be evoked by them were proposed, in addition to some of the dysfunctional rules and attitudes that may precede these cognitions. Depressed diabetics may however also present with cognitions completely unrelated to diabetes. It is proposed that by identifying the content of the depressive's cognitions (automatic thoughts, intermediate beliefs and core beliefs), we may be better equipped to make assumptions about the relative contribution of this chronic condition to the onset of depression, from a psychosocial perspective.

It was postulated that the underlying cognitive schemas of the depressed diabetic would be very similar, if not the same, as the underlying schemas or core beliefs of a depressed individual not diagnosed with diabetes. This is based on the fact that depressogenic cognitive schemas are generally formed well before the average age at diagnosis of either type 1 or type 2 diabetes. Whether diabetes or its treatment requirements can actually cause depression remains to be seen.

Beck has suggested that an individual "may develop some form of psychological

disturbance when exposed to an overwhelming stress, even if it does not strike at a specific sensitivity" (Beck, 1967, p. 280). It is however also possible that diabetes and its correlates may merely act as precipitating variables among those who already carry a cognitive vulnerability to depression.

Apart from facilitating an understanding of the association between diabetes and depression, the objective of a conceptualization of this relationship is also to promote future research in this area. The first step in this process would be to investigate and empirically validate the theoretical assumptions underlying the proposed framework by assessing for and then identifying the role of precipitating, predisposing, and maintaining factors in the onset of depression in diabetes. This may assist in further understanding the higher prevalence rates, longer duration and higher recurrence rates of depression in diabetes. An examination of the cognitive content of the depressed diabetic may assist in identifying the specific role of diabetes and its treatment requirements as precipitants to the onset of depression. Tools such as the Cognitive Response Test (Watkins & Rush, 1983) or the Automatic Thoughts Questionnaire (Hollon & Kendall, 1980) may be useful in this regard. A longitudinal investigation during which the association between cognitive vulnerability and the onset of a depressive episode can be ascertained may also be useful in clarifying the role of cognitive vulnerability in the development of depression among diabetics. Tools such as the Dysfunctional Attitudes Scale (Oliver & Baumgart, 1985; Abela & D'Alessandro, 2002) may be utilized in assessing cognitive vulnerability.

Variables such as age at diagnosis and duration of diabetes should also be examined as they may have important effect on the development of underlying schemas, even later in life.

A better theoretical understanding of the relationship between depression and diabetes would assist in the development of an appropriate empirically based cognitive therapy treatment for depression in diabetes. While only one empirical study (Lustman et al., 1998) has examined the efficacy of cognitive-behavioural treatment for depression in diabetes, the precise approach used in this study was not detailed and may not have followed Beck's (1995) cognitive therapy approach. Research aimed at empirically testing the efficacy of Beck's cognitive therapy approach for the treatment of depression in diabetes is thus also necessary.

References

American Diabetes Association (2003b). Implications of the United Kingdom Prospective Diabetes Study (Position Statement). Diabetes Care, *26*, S25-S27.

American Diabetes Association (2003a). Implications of the Diabetes Control and Complications Trial (Position Statement). Diabetes Care, *26*, S28-S32.

Rubin, R.R., & Peyrot, M. (2001). Psychological issues and treatments for people with diabetes. Journal of Clinical Psychology, *57*, 457-478.

Gavard, J.A., Lustman, P.J., & Clouse, R.E. (1993). Prevalence of depression in adults with diabetes. Diabetes Care, *16*, 1167-1178.

McGill, J.B., Lustman, P.J., Griffith, L.S., Freedland, K.E., Gavard, J.A., & Clouse, R.E. (1992). Relationship of depression to compliance with self-monitoring of glucose. Diabetes, *41(S1)*, 84A.

Lernmark, B., Persson, B., Fishert, L., & Rydelius, P-A.. (1999). Symptoms of depression are important to psychological adaptation and metabolic control in children with diabetes mellitus. Diabetic Medicine, *16*, 14-22.

Lustman, P.J., Griffith, L.S., & Clouse, R.E. (1997). Depression in adults with diabetes. Semin. Clin. Neuropsychiatry, *2*, 15-23.

Lustman, P.J., Griffith, L.S., Freedland, K.E., & Clouse, R.E. (1997). The course of major depression in diabetes. General Hospital Psychiatry, *19*, 138-143.

Lustman, P.J., Griffith, L.S., Clouse, R.E., Freedland, K.E., Eisen, S.A., Rubin, E.H., Carney, R.M., & McGill, J.B. (1997). Effects of nortriptyline on depression and glycaemic control in diabetes: results of a double blind, placebo-controlled trial. Psychosomatic Medicine, 59, 241-250.

Lustman, P.J., Griffith, L.S., Clouse, R.E., & Cryer, P.E. (1986). Psychiatric illness in diabetes mellitus: relation to symptoms and glucose control. Journal of Nervous and Mental Disorders, 174, 736-746.

Kovacs, M., Mukerji, P., Drash, A., & Lyengar, S. (1995). Biomedical and psychiatric risk factors for retinopathy among children with IDDM. Diabetes Care, 18, 1592-1599.

Lloyd, C., Wilson, R., & Forrest, K. (1997). Prior depressive symptoms and the onset of coronary heart disease. Diabetes, 46, 13A.

Lustman, P.J., Griffith, L.S., Freedland, K.E., Kissel, S.S., & Clouse, R.E. (1998). Cognitive-behaviour therapy for depression in type 2 diabetes: results of a randomized controlled clinical trial. Annals of Internal Medicine, 129, 613-621.

O'Kane, M., Wiles, P.G., & Wales, J.K. (1994). Fluoxetine in the treatment of obese type 2 diabetic patients. Diabetes Medicine, 11, 105-110.

Potter Van Loon, B.J.P., Radder, J.K., Frolich, M., Krans, H.M., Zwinderman, A.H., & Meinders, A.E. (1992). Fluoxetine increases insulin action in obese nondiabetic and in obese non-insulin-dependent diabetic individuals. International Journal of Obesity and Related Metabolic Disorders, 16, 79-85.

Van der Does, F.E., De Neeling, J.N., Snoek, F.J., Kostense, P.J., Grootenhuys, P.A., Bouter, L.M., et al. (1996). Symptoms and well-being in relation to glycaemic control in type 2 diabetes. Diabetes Care, *19*, 204-210.

Goodnick, P.J., Henry, J.H., & Buki, V.M.V. (1995). Treatment of depression inpatients with diabetes mellitus. Journal of Clinical Psychiatry, *56*, 128-136.

Talbot, F., & Nouwen, A. (2000). A review of the relationship between depression and diabetes in adults: is there a link? Diabetes Care, *23*, 1556-1562.

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

Beck, A.T. (1967). Depression: clinical, experimental and theoretical aspects. New York: Harper and Row.

Beck, A.T. (1983). Cognitive therapy of depression: new perspectives. In P.J. Clayton, and J.E. Barret (Eds.). Treatment of depression: old controversies and new approaches. New York: Raven Press.

Olinger, L.J., Kuiper, N.A., & Shaw, B.F. (1987). Dysfunctional attitudes and stressful life events: an interactive model of depression. Cognitive Therapy and Research, *11*, 25-40.

Wise, E.H., & Barnes, E.R. (1986). The relationship among life events, dysfunctional attitudes, and depression. Cognitive Therapy and Research, *10*, 257-266.

Kwon, S., & Oei, T.P.S. (1992). Differential causal roles of dysfunctional attitudes and automatic thoughts in depression. Cognitive Therapy and Research, 16, 309-328.

Joiner, T.E. Jr., Metalsky, G.I., Lew, A., & Klocek, J. (1999). Testing the causal mediation component of Beck's theory of depression. Evidence for specific mediation. Cognitive Therapy and Research, 23, 401-412.

Abela, J.R.Z., & D'Alessandro, D.U. (2002). Beck's cognitive theory of depression: a test of the diathesis-stress and causal mediation components. British Journal of Clinical Psychology, 41, 111-128.

Beck, A.T. (1984). Cognition and therapy. Archives of General Psychiatry, 41, 1112-1114.

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

Simons, A.D., Garfield, S.L., & Murphy, G.E. (1984). The process of change in cognitive therapy and pharmacotherapy for depression: changes in mood and cognition. Archives of General Psychiatry, 41, 45-51.

Bernbaum, M., Alpert, S.G., & Kuckro, P.N. (1988). Psychosocial profiles of patients with visual impairment due to diabetic retinopathy. Diabetes Care, 11, 551-557.

Wuslin, L., & Jacobson, A.M. (1989). Visual and psychological function in PDR. Diabetes, 38 (Supp 1), 324A.

Peyrot, M., & Rubin, R.R. (1989). Determinants of depression among diabetic adults. Diabetes, 38 (Supp1), 9A.

Wuslin, L., Jacobson, A.M., & Rand, L.I. (1986). Psychosocial aspects of diabetic retinopathy. Diabetes Care, 10, 367-373.

Wuslin, L., Jacobson, A.M., & Rand, L.I. (1993). Psychosocial adjustment to advanced proliferative diabetic neuropathy. Diabetes Care, 16, 1061-1066.

Karlsøn, B., & Agardh, C.D. (1997). Burden of illness, metabolic control and complications in relation to depressive symptoms in IDDM patients. Diabetic Medicine, 14, 1066-1072.

Talbot, F., Nouwen, A., Gingras, J., Bélanger, A., & Audet, J. (1999). Relation of diabetes intrusiveness and personal control to symptoms of depression among adults with diabetes. Health Psychology, 18, 537-542.

Devins, G.M. (1994). Illness intrusiveness and the psychosocial impact of lifestyle disruptions in chronic life threatening disease. Adv. Renal. Replace. Therapy, 1, 251-263.

Littlefield, C.H., Rodin, G.M., Murray, M.A., & Craven, J.L. (1990). Influence of functional impairment and social support on depressive symptoms in persons with diabetes. Health Psychology, 9, 737-749.

Lustman, P.J., Griffith, L.S., & Clouse, R.E. (1988). Depression in adults with diabetes: results of a five-year follow-up study. Diabetes Care, 11, 605-612.

Kovacs, M., Obrosky, D.S., Goldston, D., & Drash, A. (1997). Major depressive disorder in youths with IDDM: a controlled prospective study of course and outcome. Diabetes Care, 20, 45-51.

Lustman, P.J., Clouse, R.E., Carney, R.M., & Griffith, L.S. (1987). Characteristics of depression in adults with diabetes. Proceedings of the National Institutes of Mental Health Conference on Mental Disorders in General Health Care Settings. Seattle, WA, 127-129.

Winokur, G., & Pitts, F.N. Jr. (1965). Affective disorder. VI. A family history study of prevalences, sex differences and possible genetic factors. Journal of Psychiatric Research, 3, 113-123.

Cox, D.J., Gonder-Frederick, L., & Saunders, J.T. (1991). Diabetes: clinical issues and management. In J.J. Sweet, R.H. Rozensky, & S.M. Tavian (Eds.), Handbook of clinical psychology in medical settings. New York: Plenum Press.

Beck, J.S. (1995). Cognitive therapy: basics and beyond. New York: The Guilford Press.

Palinkas, L.A., Barret-Connor, E., & Wingard, D.L. (1991). Type 2 diabetes and depressive symptoms in older adults: a population based study. Diabetes Medicine, 8, 532-539.

Padgett, D.K. (1993). Socio-demographic and disease related correlates of depressive symptomatology among diabetic patients, Zagreb, Croatia. Journal of Nervous and Mental Disorders, 181, 123-129.

Oliver, J.M., & Baumgart, E.P. (1985). The dysfunctional attitude scale: psychometric properties and relation to depression in an unselected adult population. Cognitive Therapy and Research, 9, 161-167.

Watkins, J.T., & Rush, A.J. (1983). Cognitive response test. Cognitive Therapy and Research, 7, 425-436.

Hollon, S.D., & Kendall, P.C. (1980). Cognitive self statements in depression: development of an automatic thoughts questionnaire. Cognitive Therapy and Research, 4, 383-395.