The Relation Between Stress and Insulin-Dependent Diabetes Mellitus: Physiologic Arousal or Disruption of Compliance?

Virginia Diane Garrett
Louisiana State University and Agricultural & Mechanical College

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The relation between stress and insulin-dependent diabetes mellitus: Physiologic arousal or disruption of compliance?

Garrett, Virginia Diane, Ph.D.
The Louisiana State University and Agricultural and Mechanical Col., 1992
THE RELATION BETWEEN STRESS AND INSULIN-DEPENDENT DIABETES MELLITUS: PHYSIOLOGIC AROUSAL OR DISRUPTION OF COMPLIANCE?

A Dissertation

Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College in partial fulfillment of the requirements for the degree of Doctor of Philosophy in

The Department of Psychology

by
Virginia Diane Garrett
B.A., Augusta College, 1984
M.S., University of New Orleans, 1986
May 1992
DEDICATION

To my mother.
For your love and strength.
ACKNOWLEDGEMENTS

It is with deepest gratitude that I acknowledge the contributions of the many people who have supported me in this research. I would first like to thank the members of my committee, Drs. Drew Gouvier, Johnny Matson, Arthur Riopelle, and Donald Williamson. Thank you for your time and help on this project as well as during my years at LSU.

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ABSTRACT

The relations between minor life events, compliance, urine free cortisol, and blood glucose in 40 adults with insulin-dependent diabetes mellitus (IDDM) was examined. Specifically, this study explored whether naturally-occurring minor stressful events had disruptive effects on metabolic control through: a) an arousal mechanism mediated by cortisol, b) disruption of the individual's adherence to prescribed treatment, c) a combination of arousal and disruption of compliance, or d) a third, unspecified mechanism. Stress did not influence metabolic control, either independently or via a stress-compliance or stress-arousal mechanism although stress was related to cortisol activity. Neither the direct effects of cortisol nor a cortisol by stress interaction successfully predicted metabolic control. Moreover, stress was unrelated to diet or exercise compliance and no relation between diet or exercise compliance and metabolic control was found. Only insulin compliance was found to influence metabolic control, although the effects of insulin compliance were independent of a stress-compliance relation. Implications of the results and directions for further research are discussed.
INTRODUCTION

Investigation of psychological factors in diabetes mellitus (DM) has a long history (see Johnson, 1980 for a review). Although the notion of a "diabetic personality" is no longer tenable (Dunn & Turtle, 1981), considerable research continues on psychological variables influencing the disorder's course. In particular, researchers have shown stress may influence metabolic stability in individuals having the disease (see Goetsch, 1989 for a review). In addition, those factors moderating the influence of stress on the disease have been important.

Adults with DM provide an excellent population for investigating the effects of stress on the disorder. In addition to the stringent requirements of their treatment regimen, insulin-dependent DM patients have a higher frequency of hospital admissions and are more likely to experience poor metabolic control than do their non-insulin dependent cohorts (Davis, Hess, Van Harrison, & Hiss, 1987). Poor metabolic control may increase the likelihood of developing complications associated with the disorder. Adults with DM are, therefore, by virtue of the length and nature of their illness, a population at risk for severe complications of their disease. Determining the extent to which minor stress may contribute to the increased risk is an area needing research.
The purpose of this investigation was to examine the two primary hypothetical mechanisms regarding the effects of stress on metabolic control in DM. Both the direct effects via a sympathetically-mediated arousal mechanism and indirect effects via disruption of compliance behaviors were explored. Specifically, the relations between minor life events, compliance behaviors, urine free cortisol, and blood glucose in young adults with insulin-dependent diabetes mellitus (IDDM) were investigated.

In the ensuing literature review, an overview of DM will be provided, including the pathophysiology and treatment of the disorder followed by current etiological theories. In addition, a brief introduction to the concept of stress and its relation to illness will be included. Finally, a review of available literature regarding stress-arousal and stress-compliance hypotheses and their relation to metabolic stability in IDDM will be presented.

Definition and Impact of the Disorder

Diabetes mellitus is a group of complex disorders of carbohydrate metabolism associated with compromised insulin activity or production. The cardinal symptom of DM is hyperglycemia, however, polyuria, polydipsia, polyphagia, fatigue, weight loss, and blurred vision also characterize DM (Olefsky, 1988). In addition, a variety
of microvascular and macrovascular complications are associated with the disorder. Microvascular complications include thickening of the capillary basilar membranes, retinopathy, and nephropathy, while accelerated atherosclerotic development and peripheral vascular disease are common macrovascular complications. Other frequent problems include peripheral neuropathy, complications of pregnancy, and increased risk of infection (Davidson, 1981; Kahn, 1985).

The U.S. Department of Health and Human Services (USDHHS, 1985) estimates as many as 10 million Americans currently have DM. DM is the 10th leading cause of death in the United States (Turk & Speers, 1983) and the leading cause of new blindness (Davidson, 1981). Risk of stroke and myocardial infarction is increased 2-fold in individuals with DM, while risk of renal failure is 17 times greater (Cahill, 1985; Davidson, 1981). Individuals with DM also are at significantly increased risk of developing coronary artery disease. Moreover, incidence of peripheral vascular disease is increased 50 times over non-diabetics (Cahill, 1985) contributing to the high rate of amputations in this population.

DM also represents a significant economic burden. In 1980, economic costs of DM were estimated to be as high as $9.7 billion including costs of morbidity and mortality (Krall, Entmacher, & Drury, 1985). Direct costs of
medically related services were approximately $4.8 billion, including $2.2 billion for medical care, $1.24 billion for nursing home care, $840 million for patient visits to physicians, and $380 million for medication (Krall et al., 1985).

The World Health Organization (WHO, 1980) has proposed three classification categories of DM including Type I DM, or insulin-dependent diabetes mellitus (IDDM), Type II, or noninsulin-dependent diabetes mellitus (NIDDM), and DM associated with specific medical conditions (e.g., pancreatic disease, genetic syndromes, drug-induced conditions). The remainder of this discussion will focus on Type I DM, or IDDM.

**Pathophysiology of IDDM**

Insulin, an anabolic hormone secreted by pancreatic beta cells, regulates storage and use of the body's energy sources. Three primary insulin-sensitive tissues are influenced by its action: liver, muscle, and adipose tissue. In the liver, insulin stimulates the storage of glucose as glycogen or fat. Moreover, the presence of active insulin controls glycogenolysis (the breakdown of glycogen into glucose) in a fasting state. In muscle and adipose tissue, insulin serves to increase cell permeability to glucose providing cells with a readily available energy source. Insulin also facilitates fat synthesis and inhibits breakdown of previously stored
fats. Finally, insulin influences protein metabolism by facilitating protein synthesis and inhibiting breakdown of stored protein in tissues (Davidson, 1981).

Under normal conditions, insulin secretion increases after one eats in response to the carbohydrate and protein content of a meal (Davidson, 1981). Increased circulating levels of insulin stimulate storage of glucose and fat as energy reserves in insulin-sensitive tissues. While one fasts, insulin levels decline. The relative lack of insulin during a fast (i.e., between meals and at night) facilitates release of stored glucagon and fatty acids as fuel sources (Cahill, 1985). Any disruption of the balance of this process serves to alter significantly the body's metabolism of carbohydrates, fats, and proteins.

In Type I DM, beta cells of the pancreas fail to produce insulin, resulting in an absolute deficiency of endogenous insulin. Therefore, while fasting one's insulin deficiency provides a continuous signal to insulin-sensitive tissues to release stored fuels. Fasting blood glucose levels of individuals with Type I DM are, therefore, significantly elevated over normals. A further postprandial elevation of blood glucose also occurs in individuals with IDDM. Consequently, individuals with IDDM experience both fasting and postprandial hyperglycemia. Hyperglycemia is currently the
most commonly used basis for diagnosis of DM (Davidson, 1981).

Left untreated, hyperglycemia further deteriorates in the absence of insulin's inhibitory effects on glyconeolysis and leads to water and electrolyte depletion (Davidson, 1981). Changes in protein metabolism caused by a lack of insulin results in the synthesis of additional glucose from amino acids as well as water and electrolyte imbalances.

Additional detrimental effects may be caused by changes in fat metabolism. Lack of available insulin leads to accelerated lypolysis (i.e., breakdown of triglycerides) and subsequent increases in glycerol and free fatty acids (FFA) in blood. Glycerol contributes to further release of glucose by the liver further complicating the hyperglycemic state. Moreover, FFA release leads to ketogenesis (i.e., the development of ketone bodies). Overproduction of ketone bodies can exceed the body's ability to neutralize acid effects and acidosis may ensue (Davidson, 1981).

The constellation of acidosis, ketonuria (i.e., ketone bodies in the urine), dehydration, and electrolyte depletion is known as diabetic ketoacidosis (DKA) and is life-threatening. DKA may be the first clinical indication of IDDM (Olefsky, 1988) and prior to discovery
of insulin was the leading cause of death in persons with IDDM (Davidson, 1981).

**Management of IDDM**

Maintenance of a normoglycemia is the primary goal of diabetes treatment (Olefsky, 1988). Available evidence suggests prolonged hyperglycemic conditions may significantly contribute to development of microvascular complications and increase the likelihood of later kidney and eye disease (Cahill, Etzwiler, & Freinkel, 1976; Olefsky, 1988; Zimmerman, 1989). Hypoglycemic states, conversely, can quickly result in permanent damage to the central nervous system (Olefsky, 1988).

Presently, the principal means of attaining normoglycemia is through use of exogenous insulin injections. Most IDDM patients must administer at least two injections daily of some combination of short-, intermediate-, or long-acting insulin in an attempt to approximate the action of endogenous insulin. Glucose levels also must be monitored by the patient several times daily to assess glycemic control. Unfortunately, exogenous insulin injections are only partially successful in achieving normoglycemia (Cahill et al., 1976).

Insulin-infusion pumps, an alternative to self-administration of injections, are a more recent development in treatment of IDDM. Pumps provide insulin to the individual 24 hours a day and provide a potential
means to improve glycemic control. However, nocturnal hypoglycemia is a serious potential side effect of infusion pump use (Olefsky, 1988). In addition, pumps present a more visible indicator of illness and may require even more lifestyle changes than insulin-injections (Olefsky, 1988). Regardless of the method of insulin therapy used (i.e., injection or pump), a great deal of responsibility for management of the disorder rests with the patient (Olefsky, 1988).

In addition to insulin therapy, IDDM patients must adhere to stringent diets. The number of calories ingested and the source of the calories (i.e., fats, carbohydrates, etc.) must be closely monitored. A low level of fats in the diet and reduced ingestion of sugars are minimal requirements of a diabetic diet. Timing of meals is also important because caloric intake must be adjusted to correspond to available insulin activity. Frequent small meals are, therefore, generally prescribed to avoid significant post-prandial metabolic changes.

A third area of IDDM treatment involves exercise. A moderate physical exercise regimen is often prescribed by the physician as part of a total treatment program. Exercise may improve glucose utilization and decrease peripheral resistance to insulin (Ekoe, 1988). Excessive exercise or exercise in the presence of insufficient insulin, however, may result in hypoglycemia (Horton,
1988). Nonetheless, exercise is a frequent component of diabetes management.

Finally, patient education is an important component of diabetes management. The complex nature of the disorder and the demands placed on the patient require a high level of understanding of the disease. A recent meta-analysis of educational interventions reported patient education was a significant factor in positive patient outcome (Brown, 1988).

**Etiology of IDDM**

Although damage to the pancreatic beta cells in patients with DM was noted at the turn of the century (Cahill, 1985), to date the etiology of the disorder has eluded medical researchers. Nonetheless, scientists agree the disease appears to run in families and is frequently associated with infectious processes and abnormal immunological functioning. These findings have led researchers to suggest DM is a complex phenomenon subject to multiple influences (Soeldner, 1982). A brief literature review of the three primary areas involved in the etiology of IDDM follows; namely genetic, viral, and autoimmune contributions. The role of psychological factors in the etiology and course of the disease also will be discussed.

**Genetic Influences.** Three primary lines of evidence support a genetic role in the etiology of DM. Data from
twin, family, and histocompatibility antigen studies support a genetic contribution. However, the data are far from conclusive. Using more than 100 monozygotic twin pairs, Pyke and Nelson (1976) found 50% concordance in twins diagnosed with DM before age 40. Based on age at diagnosis, these data appear related to individuals with IDDM. However, in twins whose diagnoses were made after age 40 (suggesting a diagnosis of NIDDM), concordance approached 100%. Others have reported similar findings (Gottlieb & Root, 1968). A strong genetic component in NIDDM is suggested, but data are less conclusive regarding an etiological role of heredity in Type I DM.

No identifiable pattern of inheritance has been identified in patients with IDDM (Craighead, 1978) although it is agreed the syndrome is not autosomal dominant (Olefsky, 1988). Reports based on family studies reveal a fairly low degree of direct transmission from affected parents to offspring. Risk to offsprings of diabetic parents is estimated to be 2 to 5% compared to 0.2 to 0.3% risk in the general population. For children with IDDM, sibling risk increases to 5 to 10% (Olefsky, 1988). Significantly increased risk is accrued, however, if siblings are human leukocyte antigen (HLA) identical to the affected sibling (Olefsky, 1988).

Although no specific genetic marker for IDDM has been identified, researchers have expressed considerable
interest in the increased incidence of IDDM associated with certain HLA antigens. The presence of the HLAs does not directly cause IDDM; rather specific loci of the major histocompatibility complex encoded on chromosome 6 appear related to increased risk (Olefsky, 1988). HLAs may offer a means of identifying those individuals most vulnerable to IDDM and provide clues to the existence of a "diabetogenic" gene (Olefsky, 1988).

The preponderance of evidence from genetic studies suggests IDDM is influenced by genetic factors. Yet, available data also indicate an equally strong influence of nongenetic variables. Olefsky (1988) suggests determination of the precise contribution of heredity is difficult for four primary reasons: 1) no specific marker has been identified, 2) the degree of heterogeneity both within and between types of DM (i.e., IDDM and NIDDM), 3) the interaction between a "diabetogenic" gene, other genetic factors, and environmental factors, and 4) low rates of transmission of the disorder from generation to generation. At present, the majority of investigators believe genetic factors are diathetic, requiring other influences to result in expression of the illness (Craighead, 1978; Olefsky, 1988).

Viral Influences. A second area of interest in the etiology of IDDM involves the role of viral agents. In his review Olefsky (1988) outlined several lines of
evidence for a viral component in the onset of IDDM. First, incidence of IDDM has been reported to vary on a seasonal basis. A number of authors report increased onset of DM during the late summer to early autumn or winter months (e.g., MacMillan, Kotoyan, Zeidner, & Hafezi, 1977) corresponding to the presence of increased viral infections during these times. Second, a history of viral illness is frequently found to precede diagnosis of IDDM, particularly mumps (Sultz, Hart, Zielezny, & Schlesinger, 1975) and Coxsackie B virus (Gamble, Kinsley, FitzGerald, Bolton, & Taylor, 1969). Third, an increase in viral titers is often found in patients newly diagnosed with IDDM at or near the beginning of their illness.

A fourth line of evidence to support a viral role in the onset of IDDM involves the use of animal studies. Injections of "diabetogenic" viruses (e.g., Coxsackie B, encephalomyocarditis) can led to onset of IDDM in rodents. Moreover, the likelihood of developing IDDM after viral inoculation can be manipulated by varying genetic susceptibility to the disease. This evidence supports a predisposing role of heredity in IDDM that is further aggravated by the presence of physiological stressors (i.e., viral infection). Finally, "diabetogenic" viruses introduced to beta cells in culture medium have been shown to cause cell lysis and necrosis (e.g., Prince, Jensen, Billup, & Notkins, 1978). These findings are particularly
indicative of a viral component to IDDM suggesting a direct effect of viral activity on pancreatic beta cell integrity.

**Autoimmune Influences.** A final area of concern in the etiology of IDDM involves autoimmune reactions. An autoimmune process is believed to affect beta cells leading to their destruction (Kahn, 1985). Several lines of evidence support an autoimmune reaction in IDDM. First, IDDM is frequently associated with other endocrine disorders of autoimmune origin (Kahn, 1985). In particular, disorders of the adrenal and thyroid gland often coexist with IDDM (Kozak & Cooppan, 1985). Because autoimmune disorders tend to occur more frequently in families of affected individuals, a genetic predisposition to develop autoimmune diseases may be involved (Kaldany, Busick, & Eisenbarth, 1985).

Second, a number of studies indicate a high level of islet-cell antibodies (ICA) in patients with IDDM (see Kaldany et al., 1985 for a review). Combined data from several studies indicate 29.2% of patients with IDDM had ICAs as compared to 1.3% of nondiabetic persons (Kaldany et al., 1985). Elevated levels of ICA are most commonly reported in recently diagnosed individuals, although some patients maintain high ICA titers for a number of years (Kaldany et al., 1985). While these results are far from conclusive and data exist to suggest the presence of ICAs
alone are insufficient to account for the disorder (cf., (Olefsky, 1988, Kahn, 1985), increased levels of ICAs at or about the time of diagnosis in IDDM nonetheless suggests involvement of an autoimmune process. Finally, immunosuppressive treatment has been shown to prevent development of IDDM in genetically susceptible rats (Laupacis et al., 1983; Like, Anthony, Guberski, & Rossini, 1983).

In summary, similar to the potential viral influences in the etiology of IDDM, autoimmunity appears to play a role in the genesis of the disease. At present, however, the mechanism initiating an autoimmune response is unknown (Kahn, 1985). The most plausible explanation of the etiology of IDDM, based on current data, is that an immune response is initiated in response to environmental factors (e.g., viruses or toxins). The autoimmune response, modulated by genetic variables, then results in beta cell destruction. That is, the multiple effects of genetic, viral, and autoimmune influences culminate in IDDM.

Influence of Psychological Factors. Throughout history, psychological factors have been believed to have a role in the etiology of DM. Thomas Willis, in the 17th century, believed DM was caused by prolonged sorrow (Johnson, 1980). More recently, Menninger (1935) suggested anxiety and depression were characteristic of the 'diabetic personality'. These theories grew largely
from the psychoanalytic perspective suggesting specific personality traits caused or led to an exacerbation of the disease via displacement of psychological conflicts (Daniels, 1939). Individuals with DM were variously believed to be less alert, more apathetic, more hypochondriacal, and more likely to become depressed than were non-diabetic persons (Menninger, 1935).

In a seminal review article by Dunn and Turtle (1981), the concept of a specific diabetic personality with etiological implications for the disorder was seriously challenged. Investigations of specific traits etiologically linked to DM have been methodologically flawed (Dunn & Turtle, 1981) and have little empirical support (Dunn & Turtle, 1981; Surwit, Feinglos, & Scovern, 1983; Turk & Speers, 1983). No consistent traits have been identified across individuals with DM (Surwit et al., 1983). Thus, theories of personality factors as contributors to the etiology of the disease are no longer accepted.

Psychological factors, however, continue to be an important area of research. Over the course of the disorder researchers have found psychologically meaningful events (i.e., stress) may influence metabolic control of individuals having the disease. Investigation of the role of stress in DM grew largely from clinical observations of the frequency of stressful occurrences associated with
onset of diabetic crisis. Decompensation of diabetic control (i.e., DKA) may be precipitated by emotional or environmental stress (Olefsky, 1988). Nabarro (1965) found that approximately 14% of severe DKA cases were preceded by environmental stress. Similarly, Cohen, Vance, Runyan, and Hurwitz (1960) reported stress related to onset of DKA in approximately 15% of their sample. These findings have resulted in a large body of literature on the effects of stress on metabolic control. In addition, those factors that may moderate or mediate the influence of stress on the disease have been an important area of investigation.
STRESS AND ILLNESS

Before proceeding to a review of the pertinent literature involving stress effects on metabolic control in IDDM, a discussion of the concept of stress will be provided. In addition, two principal means of investigating the stress-disorder relation, that is, physiological changes associated with stress and the influence of life events on IDDM will be provided.

Stress, as formulated by Selye (1956), is manifested by a pattern of physiological responses (i.e., the General Adaptation Syndrome) influencing the likelihood of illness. According to Selye's model, after exposure to demands the body responds via biochemical activity. For Selye, these biochemical changes defined stress. Continued stress leads to attempts to regain homeostasis and counteract the physiological effects of environmental demands. Prolonged exposure to stressors, however, depletes the organism's ability to maintain a homeostatic state effectively. Exhaustion quickly ensues resulting in breakdown of the body's defensive systems, subsequent tissue damage, and perhaps death. Selye's model of stress in the pathogenesis of illness, therefore, involves depletion of homeostatic adaptive mechanisms secondary to exposure to stressors.

Selye's model provided the first systematic theoretical notions of potential changes in physiological
functioning resulting from stress. Although Selye's work served as the impetus for much of the later research involving stress and illness, his theory has not stood without criticism (see Hamberger & Lohr, 1984 for a review). Nonetheless, the physiological changes accompanying the stress response proposed by Selye served as an important model for understanding the physiological substrates of stress.

Researchers now believe three primary biochemical pathways are involved in the human stress response: 1) the neural, 2) neuroendocrine, and 3) endocrine axes (Everly, 1989). The body's earliest response to stressors involves increased autonomic activity. As a result of innervation of neural pathways in the spinal cord and sympathetic ganglia, norepinephrine (NE) is released by sympathetic neurons, causing generalized arousal in target end organs. Activity of the neuroendocrine axis involves release of the adrenal medullary catecholamines, NE and epinephrine and mimics the effects of sympathetically-mediated release of NE (Everly, 1989).

The final phase of the stress response involves activity of the endocrine system. Activity of the adrenocortical axis is a particularly important aspect of this response. Pituitary secretion of adrenocorticotropic hormone (ACTH) leads to stimulation of the adrenal cortex. In response to ACTH, the adrenal cortex secretes
glucocorticoids and mineralocorticoids. The primary glucocorticoids, cortisol and corticosterone, have a number of systemic effects including increased glucose production, increased production of urea, increased release of free fatty acids, and suppression of immune responses and appetite (Everly, 1989). The mineralocorticoids, aldosterone and deoxycorticosterone are important in regulation of electrolytes and serve to increase blood pressure.

Much of stress research has focused on the activity of cortisol, the principal glucocorticoid secreted by the adrenal cortex. Researchers have reported significantly increased cortisol levels associated with a variety of laboratory stressors including: venipuncture stress (Hubert, Moller, & Nieschlag, 1989), caffeine administration, reaction time tasks (Lovallo et al., 1989), mental arithmetic (Pomerleau & Pomerleau, 1990), and examination stress (Meyerhoff, Oleshansky, & Mougey, 1988). Daily minor stress also has been reported to influence cortisol levels. Brantley, Dietz, McKnight, Jones, and Tulley (1988a) reported a positive relation between daily fluctuations in minor stressful events and cortisol in medical personnel.

In summary, Selye's (1956) conceptualization of stress as a series of specific physiological responses has led to investigation and further understanding of the
autonomic and endocrine activity associated with environmental stressors. Research indicates that laboratory and relatively minor life events can influence the physiological parameters underlying human stress.

Drawing from Cannon's (1935) early work, another tradition evolved to explore the effects of stress on health. Cannon's (1935) investigations focused on the stimulus properties of physical or emotional stress in disrupting internal homeostatic mechanisms. In contrast to Selye, Cannon viewed stress as the stimulus leading to physiological changes. The shift in focus from the response of the organism as "stress" to the stimulus as "stress" led to the development of interest in the role of life events as potential mediators of the stress-illness relation. A great deal of research has focused on the role of life events in the cause or exacerbation of illness. The occurrence of life events now is believed to be a risk factor for adverse health outcome (Elliot & Eisdorfer, 1982).

Much of the early research on life events as a means of investigating the stress-disorder relation developed from the work of Holmes and Rahe (1967) on adjustment to major life events. Major life events are frequently associated with significant life change and include, for example, marriage, birth of a child, or death of a loved one. Using the Schedule of Recent Events (SRE) to assess
the occurrence of major life events, respondents indicate which of 43 major life events occurred during the past year. A number of studies have reported positive relations between the occurrence of major life events and both physical and psychological symptoms (see Dohrenwend & Dohrenwend, 1978, 1981; Rabkin & Streuning, 1976 for reviews). The adjustment required by these events are proposed to increase significantly the likelihood of developing a variety of physical disorders (Rahe & Arthur, 1978).

Although use of the SRE or modifications of the scale dominated behavioral medicine research on stress during the 1970s (Kanner, Coyne, Schaefer, & Lazarus, 1981), the research has not been without criticism (see Dohrenwend & Dohrenwend, 1978 for a review). Criticisms of the life-events approach have ranged from psychometric issues (e.g., Schroeder & Costa, 1984) to effect size (e.g., Rabkin & Streuning, 1976). Empirical support for the effect of major life events on illness, for example, has traditionally been quite modest (Rabkin & Streuning, 1976).

In addition, the SRE does not allow the respondent to indicate whether the change associated with the life event is positive or negative in direction. Several authors reported no relation between change associated with positive major life events (e.g., birth of a child) and
later adaptation (e.g., Ross & Minowsky, 1979; Vinokur & Selzer, 1975). In response to the criticisms of the SRE, Sarason, Johnson, and Siegel (1978) attempted to improve assessment of major life events through development of the Life Experiences Survey (LES). The LES is more carefully worded than the SRE and allows respondents to indicate whether the event was positive or negative. In addition, the LES provides subjects with the opportunity to indicate the subjective impact of the event on a Likert-type scale.

More recently, investigators interested in the stress-disorder relation have focused attention on effects of daily minor stressors or "hassles" (Brantley, Waggoner, Jones, & Rappaport, 1987; DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982; Kanner et al., 1981, Monroe, 1983). Minor stressors include such things as getting stuck in traffic, performing poorly on a task, or bad weather. Compared to major life events, minor stressors more likely occur on a daily basis and generally impact the individual less (Brantley & Jones, 1989). Investigations of minor stress may offer information on the temporal relation between symptom onset and exacerbation not apparent when using more global, retrospective measures of major life events. In addition, minor stress contributes information independently of what can be attributed to major life events in predicting physical symptoms (DeLongis et al., 1982).
**Stress and IDDM**

Two primary hypotheses regarding effects of stress on IDDM have been delineated. First, stress may directly influence metabolism in IDDM via action of the counterregulatory or "stress" hormones, suggesting that stress' effects on metabolism are mediated by arousal, or activation of the sympathetic branch of the autonomic nervous system. In response to sympathetic arousal, release of hormones associated with the stress response (e.g., ACTH, growth hormone, corticosteroids, NE, and epinephrine) result in reduced plasma insulin in normal individuals. Consequently, levels of blood glucose and free fatty acids (FFA) are increased (Lustman, Carney, & Amado, 1981; Tarnow & Silverman, 1981-82) resulting in mobilization of energy sources for use in a "fight or flight" response. In individuals with DM, metabolic changes associated with stress further compromise metabolic stability and lead to diabetic crises.

A second theory concerning the effects of stress on IDDM involves the indirect effects of stress on compliance to diabetic regimens. Accordingly, behavioral or emotional disruption resulting from the presence of stressors interferes with effective patient self-care. Stress adversely influences the course of DM through its effects on diet, insulin injections, glucose monitoring, or other aspects of a diabetic regimen.
Maintenance of normoglycemia requires adherence to a complex set of demands. IDDM patients must perform insulin injections, maintain dietary restrictions, and monitor blood glucose. Moreover, these behaviors must be performed in a temporally prescribed manner numerous times during the day (Glasgow, McCaul, & Schafer, 1986). As a result, adherence to diabetic regimens is poor typically (Fisher, Delameter, Bertelson, & Kirkley, 1982; Surwit et al., 1983; Turk & Speers, 1983).

Failure to adhere to the therapeutic regimen may contribute to metabolic instability (Turk & Speers, 1983). As previously mentioned, maintenance of metabolic stability is an important goal of treatment in DM. Moreover, a number of researchers suggest that failure to maintain adequate metabolic control may contribute to development of later complications of the disorder (Knuiman, Welborn, McCann, Stanton, & Constable, 1986; Skyler, 1979). As a result, the potential influence of both the direct and indirect effects of stress on metabolic control have generated a great deal of research.

In summary, two major hypotheses exist regarding the effects of stress on metabolic control in DM: a) direct effects via an autonomically-mediated arousal mechanism and b) indirect effects caused by disruption of compliance to prescribed regimens. The hypothesized effects of stress on metabolic control are graphically presented in
Figure 1. The following discussion will provide a review of the stress-arousal and stress-compliance literature.

Stress and Arousal. In perhaps the most frequently cited study regarding the effects of stress on metabolic control, Kemmer et al. (1986) investigated the effects of stress associated with mental arithmetic and public speaking in nine non-diabetic, nine normoglycemic Type I (i.e., good metabolic control), and nine Type I adults with induced hyperglycemia (i.e., poor metabolic control). Moreover, Kemmer et al. (1986) examined the effects of stress on the counterregulatory hormones hypothesized to be causally related to poor metabolic control. This study was designed to directly examine arousal effects of stress-induction on metabolic outcome variables. Stress had no effect on measures of blood glucose, plasma ketones, FFA, GH, or glucagon in patients in either poor or good metabolic control. However, significant changes in NE, E, and cortisol were reported. These data failed to support an arousal-mediated stress effect on metabolism. The authors conclude metabolic control is not changed by "sudden, short-lived emotional arousal that may be produced by the common stressful events of daily life" (p 1083).

Kemmer et al. (1986) note several possible reasons for their negative findings. First, the increases in cortisol and catecholamines obtained in the study, while
Figure 1. Hypothesized Effects of Stress on Metabolic Control.
statistically significant, may have been biologically insufficient to cause metabolic disruption. Second, use of laboratory-induced stressors may not adequately represent hormonal or metabolic changes accompanying naturally-occurring stressors. Conclusions about the effects of stress on metabolic control are thus limited by the external validity of the stressors.

In a similar study, Delameter et al. (1988) reported significant reductions in GH, cortisol, and free insulin in 31 adolescent subjects after exposure to either a cognitive quiz or stressful family interactions. Glucagon levels were significantly increased, while glucose, FFA, E, and NE were unchanged by the stressors. Similar to the results of Kemmer et al. (1986), the stress-arousal hypothesis was not supported. However, the results of this study are limited by use of a relatively brief stress period (i.e., 10 min) and failure to control statistically for diurnal variations in GH, cortisol, and free insulin levels. In addition, it may be inappropriate to compare the results of this study with research on adults. Chase and Jackson (1981) and Brand, Johnson, and Johnson (1986) reported age differences in the influence of stress on metabolic control suggesting caution should be used when making comparisons between investigations using different age groups.
In summary, the results of studies employing a laboratory stress-induction paradigm are suggestive of a potential disruptive role for stress on metabolism. However, these studies (i.e., Delameter et al., 1988; Kemmer et al., 1986) were unable to support the stress-arousal hypothesis. Unfortunately, methodological problems may have precluded adequate evaluation of the arousal hypothesis.

An overriding criticism of the stress-arousal studies is their lack of relevance to daily life (Goetsch, 1989; Jacobson, 1986). Laboratory-induced stressors may differ significantly from naturally-occurring stressors. Stressors employed in the laboratory are relatively circumscribed and short-lived. In addition, they may have limited meaning to research participants. In contrast, naturally-occurring stressors may be more prolonged or have greater subjective impact for the individual. Moreover, multiple naturally-occurring stressors may have a cumulative impact on the individual sufficient to result in metabolic disruption. The lack of generalizability to daily life represents a major concern regarding investigations utilizing a stress-induction paradigm.

**Stress and Compliance.** In response to the methodological limitations associated with laboratory-induced stressors, other investigations have focused on the influence of stressful life events on metabolic
stability. Studies employing a life-events paradigm have yet to address the stress-arousal hypothesis. Instead, these studies have focused on the indirect effects of stress via disruption of compliance behaviors. Nonetheless, life-events investigations represent an improvement over the previously discussed stress-arousal studies because of their greater external validity and more accurate representation of the effect of naturally-occurring events on metabolic stability. A review of available literature regarding the stress-compliance hypothesis will now be presented.

Using both adolescent and adult IDDM patients, Schafer, McCaul, and Glasgow (1986) had subjects self-monitor blood glucose testing, diet, and insulin use for two 1-wk periods (at initial contact and at 6 month follow-up). Compliance was assessed using self-monitoring of blood glucose testing, diet, and insulin injections and efforts were made to quantify compliance measures. Research participants also completed the Diabetes Family Behavior Checklist (a measure of supportive and nonsupportive family behaviors related to diabetes self-care) on both occasions. Results for adults indicated stressful family interactions were negatively correlated with compliance with glucose testing, diet, and insulin use. Moreover, compliance with blood glucose testing was significantly associated with HbA1 (i.e., glycosylated
hemoglobin, a relatively long-term measure of metabolic stability) at 6 month follow-up. No relation between stressful family interactions, compliance, and metabolic control were found for the adolescents included in the sample suggesting possible age effects in the stress-compliance relation.

Hanson and Pichert (1986) investigated the effects of daily minor stress on compliance in a group of 39 adolescents at a summer camp for diabetic children. Using a stress measure designed specifically for adolescents with diabetes, these authors reported stress disrupts dietary and exercise compliance. The number and intensity of negative stressors were negatively correlated with the number of calories consumed, suggesting that as stress increased, the amount eaten decreased. Compliance with diet was positively related to blood glucose levels while compliance with exercise was inversely related to metabolic control. Moreover, Hanson and Pichert (1986) reported stress had additional effects on blood glucose independent of its effects via changes in diet and exercise.

In contrast, several researchers have reported the effects of stress to be independent of compliance to treatment regimens. Using the Hassles Scale, a monthly measure of minor stress (Kanner et al., 1981), Cox et al. (1984) investigated the stress-compliance hypothesis in 60
adult research participants with IDDM. Compliance was assessed by having subjects rate degree of compliance in each of four areas (i.e., insulin use, diet, blood/urine glucose testing, and exercise) on a scale from 0 ("not at all") to 100% ("completely"). With the exception of insulin use, compliance measures were pooled to form a global compliance measure. Results of the study revealed daily stress was positively associated with blood glucose control as measured by HbA1, however, self-reported compliance was not related to either blood glucose control or stress. In addition, stress and compliance ratings did not interact, suggesting the effects of stress on metabolic control in this study were not moderated by compliance with diabetic regimen.

Several studies with adolescent samples also have indicated an independent relation between stress and compliance (Hanson, Henggeler, & Burghen, 1987a; 1987b). Hanson et al. (1987a; 1987b) found both stress and compliance directly influenced metabolic control but were unrelated to each other. Although the authors conclude the link between the direct effects of stress and metabolic stability is "probably physiological" no test of the arousal hypothesis was included in this study.

More recently, Halford, Cuddihy, and Mortimer (1990) conducted a longitudinal investigation of the relations among stress, compliance, and metabolic control in 15
adults with IDDM. Participants monitored blood glucose, exercise, diet, and insulin use daily for eight weeks. Using a within-subjects design, stress was found to predict blood glucose in seven of the 15 subjects. Similar to the results of the Cox et al. (1984) investigation, the effects of stress on metabolic control were independent of compliance.

Investigations of the stress-compliance hypothesis do little to resolve the stress-metabolism relation. Four of the six studies reviewed noted independence between stress and compliance effects on metabolic stability. These findings contrast with the stress-compliance hypothesis and suggest stress does not negatively influence behaviors related to compliance. The results of these studies, however, must be tempered by a number of methodological shortcomings. Specifically, these shortcomings may be grouped into three categories, that is, those related to the assessment of stress, the assessment of compliance, and the assessment of metabolic control.

First, three studies reviewed did not include an adequate assessment of stress. In the Halford et al. (1990) study, subjects rated stress on a 9 point scale from "little or no stress" to "extreme stress." Subjects were instructed to use the Hassles Scale (Kanner et al., 1981) as a guide to "what was meant by psychological stress" (Halford et al., 1990; p 519). Use of a global
unstandardized measure of stress may have obscured any potential stress-compliance relations. Moreover, it is difficult to know whether subject ratings were based on the frequency of minor life events or their subjective impact. Regardless, use of this means to assess stress introduces a source of error into the investigation. Similarly, the Hanson and Pichert (1986) study failed to use a standardized stress measure. Finally, while the negative family interactions employed in the Schafer et al. (1986) investigation are suggestive of a stressful environment they almost certainly include other constructs as well.

Second, four studies used an inadequate assessment of compliance. In the investigation by Cox and his colleagues (1984), assessment of compliance was based on ratings of subjective degree of compliance. Global ratings based on retrospective recall do little to explain the relation between stress, compliance, and metabolic control. Because stress may disrupt performance of behaviors related to compliance, a more direct assessment of compliance behaviors would serve a more useful purpose. In addition, Hanson et al. (1987a, 1987b) pooled adherence measures. Schafer, Glasgow, McCaul, and Dreher (1983) suggest compliance to one aspect of treatment may be independent of other components. Thus, combining
compliance measures may not adequately portray stress effects.

Although Halford et al. (1990) examined components of compliance, this investigation also suffers from problems associated with compliance measurement. In the dietary compliance measure used in the Halford et al. (1990) investigation, subjects rated dietary compliance on a 5-point scale from "mostly poor foods with no good foods" to "mostly good foods with no poor foods" after being provided with a list of poor, moderate, and good foods. Failure to obtain a diet diary may obscure fluctuations in dietary compliance not apparent with a global daily rating.

The stress-compliance studies also have methodological problems associated with assessment of the dependent measure. In order for the results of these studies to be meaningful, accurate reflection of metabolic stability for the time period under investigation must be obtained. Four of six studies used HbA1 as the primary indicator of metabolic stability. HbA1, while a reliable and valid measure of metabolic control, reflects blood glucose control over a 6 to 8 week period. More proximal measures of metabolic control would appear to give more precise information about the stress-compliance relation.

When more proximal measures of metabolic control are obtained these measures should be accurate representations
of metabolic control. In the Halford et al. (1990) investigation, subjects self-monitored blood glucose three times daily. In question is the means by which subjects obtained these measures. According to the authors, some subjects used glucometers while others used visually read glucostrips to determine blood glucose levels. Reflectance glucometers are significantly more accurate than reading of glucostrips. Use of two means of assessing blood glucose may have contributed to the negative findings in the Halford et al. (1990) study. In addition, no subjects used glucometers equipped with memory capability. Ample evidence exists to suggest individuals with IDDM may significantly under-report blood glucose levels (e.g., Mazze et al., 1984; Gonder-Frederick, Julian, Cox, Clarke, & Carter, 1988). Although Halford and colleagues indicate memory equipped glucometers were not available at the time of their study, failure to further investigate stress-compliance relations based on potentially erroneous results may be premature.

Summary and Conclusions. Two principal hypotheses have been posited regarding the effects of stress on metabolic stability in IDDM. First, stress is believed to have direct effects on metabolic control through a sympathetically-mediated arousal mechanism. Via action of the stress hormones, stress may adversely influence carbohydrate metabolism leading to metabolic instability.
Previous investigations of the effects of sympathetic nervous system arousal in IDDM have relied exclusively on the potential disrupting influence of brief laboratory-induced stressors. However, these studies fail to support an arousal-mediated effect of stress on metabolic outcome variables (e.g., Kemmer et al., 1986). Several investigators have extensively criticized use of laboratory-induced stressors because they lack relevance to naturally-occurring stressful events (e.g., Goetsch, 1989). This methodological concern highlights the importance of studying stress and its impact in the natural environment.

A second hypothesis regarding the effects of stress on metabolic control involves its indirect effects on compliance with a prescribed treatment regimen. According to this hypothesis, behavioral disruption associated with the occurrence of stressful events may influence the willingness with which individuals with IDDM adhere to their diets, perform insulin injections, exercise, or monitor glucose. Because compliance with treatment is an important variable in maintaining metabolic control, the occurrence of stress may increase risk of developing later complications of the disease associated with metabolic instability.

Research addressing the stress-compliance hypothesis has employed a more ecologically valid means of
determining stress than investigations of the stress-arousal hypothesis. Yet, while some investigators report an adverse effect of life events stress on compliance with treatment (e.g., Schafer, McCaul, & Glasgow, 1986), others found the effects of stress to be independent of compliance (e.g., Cox et al., 1984; Hanson et al., 1987a; 1987b). However, these investigations suffer from significant methodological problems that may have made stress-compliance relations difficult to find.

In summary, investigations of the stress-arousal hypothesis have employed a means of assessing stress with limited generalizability to naturally-occurring stressors. Few investigations have been conducted in this area, and they have failed to support a sympathetically-mediated arousal effect on metabolic control. To date, there have been no investigations of the direct effects of naturally-occurring stressors on metabolic control via arousal mechanisms. Similarly, the results of investigations of the stress-compliance hypothesis have done little to explain stress effects on metabolic stability.
THE CURRENT STUDY

This study was designed to investigate the two primary hypotheses regarding the effects of stress on metabolic control in adults with IDDM. That is, both the direct effects proposed to result from a sympathetically-mediated arousal process and indirect effects resulting from disruption of compliance to treatment regimen were examined. Although both hypotheses are generally accepted in the DM literature, both have been inadequately explored.

Delineating the relative contributions of an arousal-mediated process or a process of disruption of compliance behaviors has potential impact for treatment of individuals with IDDM. Tailoring treatment to the cause of the disorder is an important variable in treatment outcome. As such, determining the cause of metabolic instability resulting from stress would suggest that those who respond to stress with a predominant autonomic response may best be helped with a stress management approach to treatment while those who respond to stress by changes in compliance behavior might be more appropriately addressed through behavioral management.

The relation between minor life events, compliance behaviors, sympathetic arousal, and blood glucose in adult patients with IDDM was examined. Specifically, this study explored whether naturally-occurring minor stressful
events have disruptive effects on metabolic control through: a) an arousal mechanism, b) disruption of the individual's adherence to a prescribed treatment regimen, c) a combination of arousal and disruption of compliance, or d) a third unspecified mechanism. The present study addressed methodological shortcomings of previous investigations by simultaneously: a) employing a psychometrically sound and ecologically valid means of assessing stress, b) quantifying compliance measures, and c) using a valid, accurate, and proximal means of assessing metabolic control.

Research Questions Addressed by the Study

1. Do naturally-occurring minor stressors influence metabolic control by disrupting compliance with diet, exercise and/or insulin use? Although much of the previous stress-compliance research has been equivocal, it is expected that the quantified compliance measures employed in the current study will indicate the stress-compliance relation does influence metabolic control. However, stress may have a relatively greater effect on some aspects of the treatment regimen than others. No specific hypotheses are made regarding the effects of stress on metabolic control via the separate components of compliance (i.e., diet, exercise, and insulin use) assessed in this study.
2. Do naturally-occurring minor stressors influence metabolic control via a sympathetically-mediated pathway? Based on available knowledge about the effects of minor stressors on cortisol and the effects of cortisol on metabolic control, minor stress is expected to exhibit a disruptive influence on metabolic stability via changes in urinary free cortisol.

3. Do naturally-occurring minor stressors have effects on metabolic stability independent of stress-compliance and stress-arousal relations? While the stress-compliance and stress-arousal hypotheses are the primary means by which stress is proposed to have its effects on metabolic control, there is no extant reason to believe stress may not have effects on metabolic stability independent of these relations. However, the stress-compliance and stress-arousal hypotheses are expected to account for the majority of the variance in predictions of metabolic control based on available research.
METHOD

Subjects

Forty-five adult volunteers with IDDM were studied. Subjects were patients at the West Virginia University Health Sciences Center or the University of Texas Medical Branch at Galveston. Subjects were recruited in one of two ways: 1) by referral from their primary care physician or 2) via newspaper advertisements. None were pregnant during the course of the study and none were taking medications known to influence the human stress response (e.g., beta blockers, calcium channel blockers, steroids, or anxiolytics). All subjects had been diagnosed with IDDM for a minimum of one year (mean duration = 15.5 years, SD = 8.99).

Two subjects failed to exhibit correspondence between glucometer memory readings and self-reports of blood glucose on their daily diaries. This information brought into question the reliability of other self-report data for these subjects and consequently these subjects were dropped from the subject pool. Three additional subjects were eliminated because the age at diagnosis of their disease (i.e., 44, 45, and 50 years) suggested Type II diabetes according to criteria proposed by Welborn, Garcia-Webb, Bonser, McCann, and Constable (1983). The final sample for the investigation consisted of 40 participants.
Demographic data was collected on all subjects (see Appendix A). Mean age for the study participants was 33.5 years with a standard deviation of 10.35, while mean age at diagnosis of IDDM was 17.8 years (SD = 9.17). The subjects in this study were primarily white (87.5%) and had at least a partial college education (77.5%). Forty-seven and one-half per cent were married, while the remainder were either separated/divorced (17.5%) or never married (35%). The majority were employed (62.5%) and had an average income of between $10,001 to $15,000 annually. Complete demographic data for the sample are presented in Table 1.

Informed consent was obtained from all subjects prior to involvement in the study. Copies of informed consent from both West Virginia University Health Sciences Center and the University of Texas Medical Branch are presented in Appendix B. Research subjects were compensated for their time at the rate of $5.00 per day for each day of monitoring completed ($15.00 total). One subject refused monetary incentive.

Measures

Daily Stress Inventory. The DSI (Brantley & Jones, 1989) is a 58 item self-report inventory of daily stressful events or minor stressors. Respondents indicate which of 58 stressors occurred during the previous 24 hours and then rate endorsed items on their perceived
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impact using a Likert scale from 1 ("occurred but was not stressful") to 7 ("caused me to panic"). Three primary scores can be derived from the DSI: 1) the Event Score represents the number of stressful events reported as occurring during the previous 24 hour period, 2) the Impact Score is the sum of the perceived impact ratings, and 3) the I/E Ratio is the average impact rating for the previous 24 hour period. Only the Event and Impact Scores were used in the current study. Validity studies of the scale indicate good convergent (Brantley et al., 1987) and construct (Brantley, Cocke, Jones, & Goreczny, 1988b; Goreczny, Brantley, Buss, & Waters, 1988) validity. Moreover, data exist to suggest the scale is sensitive to fluctuations in urine measures of norepinephrine metabolites and cortisol (Brantley et al., 1988a) indicating the scale has good convergent validity with biochemical indicators of stress. Normative data for the scale exist for both normal and medical populations.

**Compliance Measures.** Measures of compliance with treatment regimen were divided into three components: compliance with diet, exercise, and insulin use.

Compliance with prescribed diet was assessed by having subjects monitor all food intake daily for three days. Subjects were provided with monitoring forms on which to record food intake (see Appendix C). Data obtained from food intake monitoring was then converted to
the number of exchanges in each of the six food groups (i.e., milk, vegetable, fruit, bread/starch, meat, and fat) using the Minipress Dietrak software program (N-Squared Computing, 1989) for personal computers. The number of exchanges eaten in each food group was then subtracted from the number of exchanges prescribed in each group to obtain the total number of exchange deviations (both additions and deletions) for that day. Finally, the total number of exchange deviations was summed and divided by the total number of exchanges prescribed yielding the Diet Deviation Score. Dietary compliance was, therefore, expressed as a ratio of exchange deviations to the total number of exchanges prescribed.

For example, if the individual added two fat exchanges and deleted one bread exchange relative to their prescribed diet, a total of three exchange deviations would be counted for that day. Moreover, if the individual's prescribed diet called for 12 planned exchanges per day, the total dietary deviation score for that day would be .25 or 3/12. Previous research suggests this method of quantifying dietary compliance in individuals with IDDM is sensitive to metabolic outcome measures (Christensen, Terry, Wyatt, Pichert, & Lorenz, 1983).

A test of normality of the distribution of diet deviation scores (i.e., a Shapiro-Wilk W statistic)
indicated these scores were normally distributed (Shapiro-Wilk $W = .95$, $p = .20$).

To assess compliance with medication, subjects recorded the time, type, and amount of insulin used at each injection (see Appendix D). Similar to compliance with diet, compliance with insulin use was expressed as the proportion of insulin deviations from prescribed insulin regimen. Deviations in insulin use are defined as: a) changes in timing of insulin of more than one hour or omission of an injection (one deviation); and b) inappropriate changes in amount of insulin (one deviation). Appropriate changes in insulin timing or amount to accommodate meals, exercise, hypo- or hyperglycemia were not counted as insulin deviations. The sum of each day's insulin deviations was divided by the number of insulin injections per day times two (i.e., the number of potential sources of error for that injection: timing/omission or amount) to obtain the daily insulin deviation score.

A Shapiro-Wilk $W$ statistic (Shapiro & Wilk, 1965) indicated the distribution of raw insulin deviation scores was not normal (Shapiro-Wilk $W = .92$, $p = .01$). Cohen and Cohen (1983) suggest proportional data be transformed to "linearize" the data under these circumstances. An arcsine transformation was, therefore, performed on these
In all data analyses, insulin deviation scores were expressed as arcsine transformed proportions.

Exercise compliance was determined by having subjects monitor the frequency, duration, and type of exercise performed on Exercise Monitoring Logs (see Appendix E). Exercise compliance was quantified by multiplying the frequency of actual exercise performed during the three day monitoring period by 2.3333 to obtain an actual weekly frequency score (i.e., 2.3333 days times 3 days of the study equals weekly frequency). Actual exercise frequency was converted to a weekly exercise frequency score for ease of analysis because most physicians instruct patients to exercise a set number of times per week (as opposed to a set number of times every three days). Actual exercise frequency was operationalized as the number of times an individual performed discrete exercises (e.g., workouts, aerobics). That is, walking in the mall, housework, or activities associated with job responsibilities were not counted as exercise. The absolute value of the difference between the actual weekly frequency score and the prescribed frequency score served as the Exercise Compliance Score for this study. Ten subjects reported no physician recommendations regarding exercise and two subjects had missing data for this measure. Data analyses for exercise compliance were, therefore, based on 28 subjects. Data for Exercise Compliance Scores were found
to be normally distributed (Shapiro-Wilk $W = .93$, $p = .08$).

To assess compliance with prescribed treatment, each participant completed a Prescribed Treatment Log (see Appendix F). Values from the Prescribed Treatment Log were used to determine the denominators in each equation involving compliance with diet and medication and prescribed frequency of exercise.

**Urinary Free Cortisol.** Urinary free cortisol levels were determined by Fluorescence Polarization Immunoassay (FPIA) using the TDX Systems (Abbott Laboratories; North Chicago, Illinois). All assays were conducted within four weeks of collection and urine samples were kept frozen at $-20\ ^\circ\text{C}$ until the assays were performed. Urine samples were returned to room temperature and thoroughly mixed prior to testing. Samples containing large amounts of particulate matter were centrifuged before assaying.

To control for differences in volume of urine collected, creatinine levels also were assayed for each urine sample. Creatinine level was determined by Radiative Energy Attenuation (REA) using the TDX Systems (Abbott Laboratories; North Chicago, Illinois). Use of REA technology for creatinine assay is reported to be extremely accurate when compared with reference assays (Mean = .99; Abbott Laboratories; North Chicago, Illinois). All laboratory tests were conducted by trained
laboratory technicians in the Psychopharmacology Laboratory in the Department of Behavioral Medicine and Psychiatry at the West Virginia University Health Sciences Center. Laboratory technicians were blind to the hypotheses of the research.

The distribution of cortisol:creatinine raw data indicated a non-normal distribution (Shapiro-Wilk $W = .87, p < .0001$). An arcsine transformation was, therefore, performed on these data as suggested by Cohen and Cohen (1983). In all data analyses, urinary free cortisol levels were expressed as an arcsine transformed ratio of cortisol:creatinine.

**Blood Glucose.** Blood glucose levels were determined four times daily through use of the One Touch Blood Glucose Monitoring System (Lifescan, Milpitas, California). The One Touch System, a second-generation blood glucose monitoring system, determines blood glucose levels when a drop of blood is placed on a glucose oxidase reagent strip. Unlike many other blood glucose monitoring devices, the One Touch System does not require timing or removal of blood from the reagent strip and consequently reduces potential sources of user error (Jovanovic-Peterson, Peterson, Dudley, Kilo, & Ellis, 1988). In addition, research comparing blood glucose levels determined by the One Touch System with those of a glucose analyzer indicate excellent precision and accuracy
Use of the One Touch requires individuals to lance the side of a finger with a lancet pen to obtain a droplet of blood. Blood is then placed on a reagent strip and entered in the One Touch System. Blood glucose levels are digitally displayed to the user in mg/dL and as many as 250 previous results can be held in memory. For the present study, subjects were informed the One Touch System had memory capability and they were asked to record their daily blood glucose levels on a monitoring sheet (see Appendix D). Blood glucose levels for data analysis were expressed as daily means.

**Procedure**

After an initial screening interview, the purpose of the study and potential risks were explained to all subjects. The primary investigator was available to answer questions from subjects when obtaining informed consent.

Eligible participants were first trained to self-monitor blood glucose. All subjects were trained to use the One Touch System according to Lifescan protocol. In addition, each subject was provided written instructions on use of the One Touch. Research participants were considered adequately trained when two consecutive trials were achieved with blood glucose values within five
percent of each other (mean across subjects = 3.8%). Each participant was provided with a One Touch System including sufficient lancets, alcohol prep pads, and reagent strips to complete four blood glucose readings daily for three days. Subjects were instructed to record blood glucose before breakfast, lunch, dinner, and retiring each day and enter the obtained values in their monitoring logs.

Each subject completed the DSI daily for four days at approximately the same time before retiring for the evening. Data from DSI recording on the first day was discarded because research has indicated the DSI is reactive on the first day of monitoring (Brantley et al., 1988b). In addition, Brantley et al. (1988b) found differences between DSI scores on weekdays and weekends, therefore, all self-monitoring was conducted on weekdays to control for this potential source of variance.

Beginning on Day Two of DSI recording, subjects also completed monitoring forms for compliance assessment daily for three days. Participants were instructed to record foods consumed, insulin injections, and exercise on their self-monitoring forms. Subjects were instructed to continue to eat, exercise, and use their insulin just as they did before involvement in the study in an effort to decrease reactivity.

Finally, participants were asked to collect all urine voided between the hours of 2 p.m. and 4 p.m. (to control
for diurnal variations in cortisol output) in labeled urine collection containers provided to each subject. Collection of urine for cortisol assay began on Day Two of DSI recording concurrent with self-monitoring of compliance and continued for three consecutive days. Urine samples were then frozen by the subjects at the end of the each day and returned to the principal investigator at the end of the three day monitoring period.

Following the three days of urine collection, DSI recording, and compliance self-monitoring, subjects completed a Prescribed Treatment Log, were debriefed and released.
RESULTS

Descriptive statistics

Preliminary descriptive statistics of the measures employed in the study (i.e., DSI Event and Impact scores, insulin deviation scores, diet deviation scores, exercise compliance scores, cortisol, and blood glucose) were conducted. The mean DSI Event Score was 15.74, placing the research participants of this study at the 83rd percentile (T=60) in terms of frequency of minor stressful events compared to other medical patients. Similar results were obtained with the DSI Impact Score (mean = 39.47, 83rd percentile, T=60). The mean blood glucose reading was 181.51 mg/dL for this sample. Table 2 contains means and standard deviations for other measures used in the study.

To determine if research participants from Texas and West Virginia differed substantially on any of the demographic, independent, or dependent variables, a series of T-tests was conducted. Results of these analyses indicated no difference between subjects from Texas and West Virginia on any study variable. These data are presented in Table 3.

Correlations

Correlations were used to examine the interrelations between variables. An 8 x 7 correlation matrix of demographic measures with the primary predictor and
Table 2. Descriptive Statistics for Primary Study Variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSI Impact Score</td>
<td>39.47</td>
<td>24.88</td>
</tr>
<tr>
<td>DSI Event Score</td>
<td>15.74</td>
<td>11.46</td>
</tr>
<tr>
<td>Blood Glucose (mg/dL)</td>
<td>181.51</td>
<td>54.99</td>
</tr>
<tr>
<td>Diet Deviation Score</td>
<td>1.05</td>
<td>.35</td>
</tr>
<tr>
<td>Insulin Deviation Score(^a)</td>
<td>0.34</td>
<td>0.32</td>
</tr>
<tr>
<td>Exercise Compliance Score</td>
<td>2.60</td>
<td>1.48</td>
</tr>
<tr>
<td>Cortisol:Creatinine ratio(^a)</td>
<td>0.12</td>
<td>.06</td>
</tr>
</tbody>
</table>

Note. \(^a\) Arcsine transformed variable.
Table 3. T-tests for Differences Between States on Demographic and Primary Measures of Study.

<table>
<thead>
<tr>
<th>Variable</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.76</td>
<td>ns</td>
</tr>
<tr>
<td>Age at diagnosis</td>
<td>-1.49</td>
<td>ns</td>
</tr>
<tr>
<td>Education</td>
<td>0.22</td>
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<tr>
<td>Employment status</td>
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</tr>
<tr>
<td>Marital status</td>
<td>-0.55</td>
<td>ns</td>
</tr>
<tr>
<td>Income</td>
<td>1.64</td>
<td>ns</td>
</tr>
<tr>
<td>Blood glucose</td>
<td>-0.76</td>
<td>ns</td>
</tr>
<tr>
<td>DSI Impact Score</td>
<td>-0.44</td>
<td>ns</td>
</tr>
<tr>
<td>DSI Event Score</td>
<td>-1.72</td>
<td>ns</td>
</tr>
<tr>
<td>Cortisol:creatinine ratio(^a)</td>
<td>1.18</td>
<td>ns</td>
</tr>
<tr>
<td>Diet deviation score</td>
<td>-0.03</td>
<td>ns</td>
</tr>
<tr>
<td>Insulin deviation score(^a)</td>
<td>-0.31</td>
<td>ns</td>
</tr>
<tr>
<td>Exercise compliance score</td>
<td>-0.14</td>
<td>ns</td>
</tr>
</tbody>
</table>

Note. \(^a\) Arcsine transformed variables.
outcome measures was generated to examine possible intercorrelations between demographic and predictor/outcome variables. The eight demographic variables included in this correlation matrix were: age, ethnic group, gender, marital status, education, employment status, income level, and age at diagnosis. The seven predictor/outcome variables were mean DSI Impact and Event scores, mean diet deviation score, mean insulin deviation score, mean exercise compliance score, mean cortisol:creatinine ratio, and blood glucose. An alpha level of .01 was used to indicate statistical significance to control for experimenter-wise error. The results are presented in Table 4. Race demonstrated a significant negative correlation with blood glucose ($r = -0.45, p < .01$). This suggests that the nonwhite research participants in this study had lower blood glucose levels than the white participants had. No other correlations between demographic and predictor or outcome variables were significant.

A 7 (predictor/outcome variables) X 7 (predictor/outcome variables) correlation matrix also was generated, with statistical significance again set at $p = .01$. These results are presented in Table 5. The correlation between DSI Impact and DSI Event Scores was significant ($r = 0.74, p < .01$). The significant correlation between DSI Impact and Event Scores suggests
Table 4. **Correlation Matrix of Demographic and Predictor/Predicted Variables.**

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>Age</th>
<th>Race</th>
<th>Gender</th>
<th>Marital Status</th>
<th>Education</th>
<th>Employment</th>
<th>Income</th>
<th>Age at Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSI Impact Score</td>
<td>-0.37</td>
<td>-0.11</td>
<td>0.16</td>
<td>0.30</td>
<td>0.18</td>
<td>0.15</td>
<td>-0.36</td>
<td>-0.27</td>
</tr>
<tr>
<td>DSI Event Score</td>
<td>-0.25</td>
<td>0.05</td>
<td>0.12</td>
<td>0.25</td>
<td>0.08</td>
<td>0.06</td>
<td>-0.25</td>
<td>-0.13</td>
</tr>
<tr>
<td>Diet Deviation Score</td>
<td>-0.05</td>
<td>-0.11</td>
<td>-0.16</td>
<td>-0.02</td>
<td>0.04</td>
<td>-0.20</td>
<td>0.22</td>
<td>0.16</td>
</tr>
<tr>
<td>Insulin Deviation Score</td>
<td>0.15</td>
<td>-0.27</td>
<td>-0.17</td>
<td>-0.08</td>
<td>-0.02</td>
<td>-0.15</td>
<td>0.11</td>
<td>0.07</td>
</tr>
<tr>
<td>Exercise Compliance Score</td>
<td>-0.07</td>
<td>0.08</td>
<td>-0.15</td>
<td>0.19</td>
<td>-0.14</td>
<td>0.13</td>
<td>-0.26</td>
<td>0.06</td>
</tr>
<tr>
<td>Cortisol:Creatinine Ratio</td>
<td>-0.05</td>
<td>-0.02</td>
<td>-0.02</td>
<td>-0.17</td>
<td>0.17</td>
<td>0.05</td>
<td>-0.11</td>
<td>-0.03</td>
</tr>
<tr>
<td>Blood Glucose</td>
<td>-0.04</td>
<td>-0.45**</td>
<td>-0.12</td>
<td>-0.13</td>
<td>-0.05</td>
<td>0.12</td>
<td>0.07</td>
<td>-0.06</td>
</tr>
</tbody>
</table>

Note. **p < .001**
Table 5. *Correlation Matrix of Predictor/Predicted Variables (n)*.

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>DSI Impact Score</th>
<th>DSI Event Score</th>
<th>Diet Deviation Score</th>
<th>Insulin Deviation Score</th>
<th>Exercise Compliance Score</th>
<th>Cortisol: Creatinine Ratio</th>
<th>Blood Glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSI Impact Score</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DSI Event Score</td>
<td>.74** (40)</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diet Deviation Score</td>
<td>.01 (26)</td>
<td>-.27 (26)</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin Deviation Score</td>
<td>-.10 (40)</td>
<td>-.14 (40)</td>
<td>-.04 (26)</td>
<td>-1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise Compliance Score</td>
<td>.19 (28)</td>
<td>.12 (28)</td>
<td>-.17 (22)</td>
<td>-.12 (28)</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortisol: Creatinine Ratio</td>
<td>.39** (40)</td>
<td>.08 (40)</td>
<td>.38 (26)</td>
<td>-.14 (40)</td>
<td>-.15 (28)</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Blood Glucose</td>
<td>.20 (40)</td>
<td>.16 (40)</td>
<td>.21 (26)</td>
<td>.43** (40)</td>
<td>-.004 (28)</td>
<td>.11 (40)</td>
<td>1.00</td>
</tr>
</tbody>
</table>

*Note.**p < .001*
that either variable may be used to accurately represent an individual's experience of daily minor stress. However, Brantley and Jones (1989) suggest DSI Impact Scores may be the best indicator of an individual's experience of stress because the score takes into account personal appraisal of events. Therefore, only the DSI Impact Scores were used to quantify minor stress in the subsequent analyses (i.e., simple and multiple regressions).

A significant positive relation between DSI Impact Score and cortisol also was demonstrated ($r = .39, p < .01$). However, neither DSI Impact nor DSI Event Scores were significantly correlated with any other predictor/outcome variables. Finally, intercorrelation was noted between blood glucose and the insulin deviation score ($r = .43, p < .01$).

No relation between compliance measures was found. Lack of significant correlations between the three compliance measures (i.e., diet, exercise, and insulin use) indicated the need for separate regression analyses for each of these predictor variables in subsequent analyses. All other correlations were nonsignificant.

**Simple Regressions**

Whereas zero-order correlations indicate strength of association between two variables, simple regression allows determination of the amount of variance in a given
variable that can be predicted when the value of a second variable is known. Four separate regressions were, therefore, performed using DSI Impact Score as the regressor to investigate the degree to which diet compliance, insulin compliance, exercise compliance, or cortisol levels can be predicted when daily minor stress is known. The results of these analyses are shown in Table 6. The regression of DSI Impact Score on diet deviation score was nonsignificant, $r^2(1,24) = .000$, as was the regression of DSI Impact on insulin deviation, $r^2(1,38) = .010$, and exercise compliance, $r^2(1,26) = .035$. However, a significant relation between DSI Impact and cortisol, $r^2(1,38) = .151$, $p < .01$, was found.

A second series of simple regressions were conducted to determine if blood glucose (i.e., metabolic control) can be predicted when compliance and cortisol levels are known. In the first of these analyses, the insulin deviation score was the regressor. The results of this analysis were significant, $r^2(1,38) = .184$, $p < .01$. Simple regressions employing cortisol, $r^2(1,38) = .013$, diet deviation score, $r^2(1,24) = .046$, and exercise compliance, $r^2(1,26) = .000$, as regressor variables were nonsignificant. The results of these analyses are presented in Table 7.

A score for exercise duration also was derived (i.e., the absolute value of the difference between actual
Table 6. **Results of Simple Regressions with DSI Impact Score as Regressor Variable.**

<table>
<thead>
<tr>
<th>Predicted variable</th>
<th>df</th>
<th>$r^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet deviation</td>
<td>(1,24)</td>
<td>0.000</td>
<td>ns</td>
</tr>
<tr>
<td>Insulin deviation</td>
<td>(1,38)</td>
<td>0.010</td>
<td>ns</td>
</tr>
<tr>
<td>Exercise compliance</td>
<td>(1,26)</td>
<td>0.035</td>
<td>ns</td>
</tr>
<tr>
<td>Cortisol</td>
<td>(1,38)</td>
<td>0.151</td>
<td>.01**</td>
</tr>
</tbody>
</table>

Note. **$p < .01$**
Table 7. **Results of Simple Regressions with Blood Glucose as Predicted Variable.**

<table>
<thead>
<tr>
<th>Predictor variable</th>
<th>df</th>
<th>$r^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet deviation score</td>
<td>(1,24)</td>
<td>.046</td>
<td>ns</td>
</tr>
<tr>
<td>Insulin deviation score</td>
<td>(1,38)</td>
<td>.184</td>
<td>**</td>
</tr>
<tr>
<td>Exercise compliance</td>
<td>(1,26)</td>
<td>.000</td>
<td>ns</td>
</tr>
<tr>
<td>Cortisol:creatinine ratio</td>
<td>(1,38)</td>
<td>.013</td>
<td>ns</td>
</tr>
</tbody>
</table>

Note. **p < .01**
exercise duration and prescribed exercise duration) to determine if exercise duration successfully predicted blood glucose. However, exercise duration also failed to exhibit a significant relation with blood glucose, $r^2(1,26) = .002$.

Finally, the direct (i.e., unmediated) relation between daily minor stress and metabolic control was investigated via a simple regression with DSI Impact Score as the regressor variable and blood glucose as the predicted variable. The results of this analysis were nonsignificant, $r^2(1,38) = .040$.

Multiple Regressions

Multiple regression analyses permit determination of the amount of variance accounted for in a predicted variable while controlling for the effects of the regressor variables. Specifically, hierarchical multiple regression was selected because these procedures permit entry of the regressor variables in a pre-specified order based on theory or the purpose of the research (Cohen & Cohen, 1984). Specifying order of entry permits removal of variance associated with spurious or confounding variables. By entering variables early, and thereby removing the variance associated with them, a relatively pure test of a given variable's predictive ability can be conducted.
To directly test the effects of an interaction between minor stress score and insulin compliance on blood glucose (while controlling for the main effects of minor stress and insulin compliance), variables were entered into a hierarchical regression in the following order: DSI Impact Score, Insulin Deviation Score, with the DSI Impact by Insulin deviation interaction entered last. The full model was significant, $F(3,36) = 4.09$, $p = .01$, and accounted for 25% of the variance. However, the interaction of stress and insulin compliance was nonsignificant. Only the insulin deviation score was significant, $R^2 = .215$, $p < .05$. These results are presented in Table 8.

Similarly, a second multiple regression was performed using minor stress, urinary free cortisol and the DSI Impact Score by cortisol interaction to explore the relation between stress, cortisol and metabolic control. Variables were entered into the equation in the following order: DSI Impact Score, cortisol, and DSI Impact X cortisol interaction. Blood glucose served as the predicted variable. This order of entry allows for a direct test of the effects of a minor stress by cortisol interaction in the prediction of blood glucose. As expected based on the results of the simple regressions (i.e., no significant relation between cortisol and blood glucose), the full model for this regression was
Table 8. Regression of Daily Minor Stressors, Insulin Compliance, and Their Interaction on Blood Glucose.

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
<th>$R^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>3</td>
<td>4.09</td>
<td>.25</td>
<td>.01</td>
</tr>
<tr>
<td>Error</td>
<td>36</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>39</td>
<td></td>
<td></td>
<td></td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Partial $r^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSI Impact Score</td>
<td>.051</td>
<td>ns</td>
</tr>
<tr>
<td>Insulin Deviation Score</td>
<td>.215</td>
<td>.04</td>
</tr>
<tr>
<td>Interaction</td>
<td>.013</td>
<td>ns</td>
</tr>
</tbody>
</table>
nonsignificant, $F(3,36) = .52$, and none of the regressor variables successfully predicted blood glucose. The results of this regression are presented in Table 9.

A multiple regression to explore the interaction of stress and diet compliance also was conducted. Variable entry in the regression equation was as follows: DSI Impact Score, Diet deviation score and the DSI Impact by diet interaction, with blood glucose as the predicted variable. As presented in Table 10, the full model was nonsignificant, $F(3,22) = .46$, and no regressor variable predicted blood glucose.

An identical analysis using exercise compliance and the DSI Impact X exercise compliance interaction also yielded nonsignificant results (see Table 11). To determine if the influence of daily stress on blood glucose was mediated by exercise duration, a hierarchical multiple regression was performed using (in order of entry) the exercise duration score and the DSI Impact X exercise duration interaction as regressor variables. However, neither exercise duration or a DSI Impact X exercise duration interaction successfully predicted blood glucose, $F(3,24) = .69$.

Hierarchical multiple regression also was performed to examine the direct effects of insulin compliance on metabolic control controlling for the effects of stress. Order of entry for the predictor variables for this
Table 9. **Regression of Daily Minor Stressors, Cortisol, and Their Interaction on Blood Glucose.**

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
<th>R²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
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<td>.520</td>
<td>.04</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
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<tr>
<td>Total</td>
<td>39</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Partial $r^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSI Impact Score</td>
<td>.040</td>
<td>ns</td>
</tr>
<tr>
<td>Cortisol:creatinine ratio</td>
<td>.001</td>
<td>ns</td>
</tr>
<tr>
<td>Interaction</td>
<td>.000</td>
<td>ns</td>
</tr>
</tbody>
</table>
Table 10. Regression of Daily Minor Stressors, Diet Compliance, and Their Interaction on Blood Glucose.

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
<th>$R^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
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<td>.06</td>
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<td>Error</td>
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<td>Total</td>
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<table>
<thead>
<tr>
<th>Variable</th>
<th>Partial $R^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSI Impact Score</td>
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<td>ns</td>
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<tr>
<td>Interaction</td>
<td>.013</td>
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<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
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<th>p</th>
</tr>
</thead>
<tbody>
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<td>.942</td>
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</tr>
<tr>
<td>Error</td>
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<tr>
<td>Total</td>
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</table>

Variable Partial $r^2$ p

<table>
<thead>
<tr>
<th>Variable</th>
<th>Partial $r^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSI Impact Score</td>
<td>.000</td>
<td>ns</td>
</tr>
<tr>
<td>Exercise compliance score</td>
<td>.0006</td>
<td>ns</td>
</tr>
<tr>
<td>Interaction</td>
<td>.092</td>
<td>ns</td>
</tr>
</tbody>
</table>
analysis were DSI Impact Score and insulin deviation score. Blood glucose served as the predicted variable. The DSI Impact Score was entered first followed by the insulin deviation score. Because the goal of this analysis was to examine the unique variance associated with insulin compliance on metabolic control, the results are presented in terms of semipartial correlation coefficients as suggested by Cohen and Cohen (1983). Semipartial correlation coefficients represent the correlation between the predicted variable and the independent variable in question after the effects of the shared variance of the two (or more) independent variables have been removed (Edwards, 1984). The full model was significant, \( F(2, 37) = 5.98, p < .01 \), and accounted for 24% of the variance. Insulin compliance was found to successfully predict blood glucose independent of stress and account for 20% of the variance in metabolic control, \( sr^2 = .204, p < .01 \) (see Table 12).

Similarly, to determine if diet compliance influenced blood glucose independently of stress, a hierarchical multiple regression with DSI Impact score entered first followed by the Diet Deviation Score was performed. Blood glucose was the predicted variable. The full model was nonsignificant, \( F(2,23) = .56 \). Diet compliance was not found to influence blood glucose independently of stress (\( sr^2 = .045 \)). Cortisol (\( F(2,37) = .80, sr^2 = .001 \)) and
Table 12. **Direct Effects of Insulin Compliance on Blood Glucose Controlling for the Effects of Daily Minor Stressors.**

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
<th>R²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>3</td>
<td>5.98</td>
<td>.24</td>
<td>.006</td>
</tr>
<tr>
<td>Error</td>
<td>37</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Total</td>
<td>39</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>sr²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSI Impact Score</td>
<td>.040</td>
<td>ns</td>
</tr>
<tr>
<td>Insulin deviation score</td>
<td>.204</td>
<td>.003</td>
</tr>
</tbody>
</table>


exercise compliance ($F(2,25) = .18$, $sr^2 = .0005$) also had no direct effects on blood glucose when the variance associated with stress was statistically removed.

A final hierarchical regression employed blood glucose as the predicted variable and insulin compliance, cortisol, and DSI Impact Score as predictor variables. The DSI Impact Score was the final variable entered into the equation to determine if minor stress had direct effects on metabolic control not accounted for by a stress-arousal or stress-compliance relation. Diet and exercise compliance were not included in this analysis because neither of these components of compliance had been shown in previous analyses to influence blood glucose. The cost to statistical power of including these variables, did not, therefore, appear justified. The full model was significant ($F(3,36) = 4.04$) and accounted for 25% of the variance in blood glucose. However, as shown in Table 13, the only significant predictor of blood glucose was the Insulin deviation score ($sr^2 = .201$, $p < .01$).
Table 13. **Regression of Insulin Compliance, Cortisol, and Daily Minor Stressors on Blood Glucose.**

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
<th>$R^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
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<td>Total</td>
<td>39</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>$sr^2$</th>
<th>Model $R^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortisol:creatinine</td>
<td>.013</td>
<td>.01</td>
<td>ns</td>
</tr>
<tr>
<td>Insulin deviation score</td>
<td>.201</td>
<td>.21</td>
<td>.003</td>
</tr>
<tr>
<td>DSI Impact score</td>
<td>.038</td>
<td>.25</td>
<td>ns</td>
</tr>
</tbody>
</table>
DISCUSSION

Minor stress did not influence short-term metabolic control either independently or via a stress-compliance or stress-arousal mechanism in this sample of individuals with IDDM. Consistent with previous research (i.e., Brantley et al., 1988a), stress was related to cortisol activity, but neither the direct effects of cortisol nor a cortisol by stress interaction successfully predicted metabolic control. Moreover, stress was unrelated to diet or exercise compliance and no relation between diet or exercise compliance and metabolic control was found. Only insulin compliance was found to influence metabolic control, although the effects of insulin compliance were independent of a stress-compliance relation.

Stress has been repeatedly portrayed in the literature as influencing metabolic control in Type I diabetes, yet the results of the current study suggest no relation between stress and metabolic control for this sample. The inability to find a stress-blood glucose relation is consistent with some previous research (e.g., Edwards & Yates, 1985; Kemmer et al., 1986). However, other groups have reported significant relations between metabolic disruption and stress (e.g., Cox et al., 1984; Hanson & Pichert, 1986; Hanson et al., 1987a, 1987b).
Several potential reasons for the negative findings of the current study exist. First, several of the studies reporting a stress-blood glucose relation used adolescent samples (i.e., Hanson et al., 1987a, 1987b; Hanson & Pichert, 1986). Although poorly understood, adolescents' metabolic response to stress may be different from that of adults (Brand et al., 1986; Chase & Jackson, 1981). Schafer et al. (1986) found a differential effect of age regarding the stress-glucose relation in a mixed sample of adults and adolescents. The lack of comparability between stress' influence on blood glucose in adults and adolescents suggests it may be inappropriate to compare the results of the current study to investigations employing adolescent or mixed samples.

Second, the current study improved over past investigations by improving assessment of both the independent and dependent measures (i.e., stress and blood glucose). Use of the DSI to assess daily minor stress provided a reliable, valid, and standardized means of assessing naturally-occurring stress. Many previous investigations relied on unstandardized stress measures (e.g., Halford et al., 1990; Hanson & Pichert, 1986) making comparisons with the current study problematic.

It should be noted, however, that the results of the current study are consistent with the results of the carefully controlled laboratory stress study by Kemmer et
al. (1986). Perhaps, when stress is validly assessed (as in the current investigation and the Kemmer et al. study) no reliable relation between stress and blood glucose exists.

Moreover, the present study used an extremely accurate and proximal measure of metabolic control, that is, multiple daily reflectance glucometer readings as opposed to the more distal HbA1. Much of previous research relied exclusively on HbA1 to assess metabolic control (e.g., Cox et al., 1984). Improved accuracy in the assessment of the variables under investigation increases the probability that any existing stress-blood glucose relation would be found. Methodological shortcomings of the past research may have indicated the presence of stress-metabolic relations where none actually existed. Conversely, daily minor stress may have a cumulative long-term effect on blood glucose (i.e., as measured by HbA1), but not influence short-term glycemic control as assessed by reflectance glucometers.

A third possible explanation for the negative findings of the current study involves the experimental design. The study employed a cross-sectional approach that may have obscured any significant relations between stress and metabolic control by collapsing data across subjects. That is, some individuals in the study may have experienced metabolic disruption in response to stress,
but there may have an insufficient number of these individuals to override the effects of those who experienced no metabolic effects. Carter, Gonder-Frederick, Cox, Clarke, and Scott (1985) found idiosyncratic metabolic responses to stress within-subjects that were stable across time suggesting individual differences in stress-responsivity. Similarly, Halford et al. (1990) used a within-subjects design and found stress influenced blood glucose in only seven of 15 subjects.

These data suggest only certain individuals with IDDM may respond to stress via metabolic disruption while others may be relatively metabolically insensitive to the influence of stress. Within-subjects designs require observation of the study variables over a sufficiently long period of time to provide ample variance in the variables of interest. The three days of self-monitoring used in the current study, however, did not allow for examination of within-subject differences. It may be that with longer periods of investigation using within-subjects designs stress-glucose relations could be found.

Finally, much of the previous literature on the direct unmediated effects of stress on blood glucose has included both Type I and Type II (noninsulin-dependent; NIDDM) patients (see Goetsch, 1989 for a review). Stress may disrupt metabolic control in Type II patients while
having little or no impact on IDDM. In support of this notion, Goetsch et al. (1990) found a significant relation between daily minor stress and metabolic control in Type II patients while controlling for the influence of diet and exercise.

Moreover, research investigating the effects of relaxation training (which would presumably attenuate the influence of stress) on metabolic control suggests that IDDM and NIDDM patients may be differentially affected. Several studies have reported a positive influence of relaxation training in Type II patients (Surwit & Feinglos, 1988; Lammers, Naliboff, & Straatmeyer, 1984) but not Type I (Bradley, Moses, Gamsu, Knight, & Ward, 1985; Landis et al., 1985; Feinglos, Hastedt, & Surwit, 1987). Surwit and Feinglos (1988) conclude that stress-related arousal of the sympathetic nervous system may be more important in the pathophysiology and metabolic disruption of Type II than Type I patients. While individuals with IDDM may perceive stress to be an important factor in metabolic control (Cox et al., 1984), this may not actually be the case.

The present study was designed to investigate how stress influenced short-term metabolic control, that is, by disrupting compliance with treatment or through an autonomically-mediated mechanism via cortisol activity. While the negative results of an unmediated stress-glucose
relation make the stress-arousal and stress-compliance issues something of a moot point, several additional findings of the study warrant comment.

**Stress and Arousal**

The study represents the first investigation of the arousal hypothesis using naturally-occurring daily minor stress. Previous research (i.e., Delameter et al., 1988; Kemmer et al., 1986) examining the influence of laboratory stress has been unable to support an arousal-mediated relation between stress and metabolic control. The results of the current study are consistent with the findings of this literature. The methodological improvements of the current research (i.e., use of a more externally valid means of assessing stress and controlling for diurnal variations in cortisol secretion) lend additional credence to this body of literature. At least in terms of relatively brief stress, influences on metabolic control may not be secondary to an arousal-mediated mechanism. This finding, however, does not rule out the possibility of potential long-term stress influences on glycemic control.

In addition, the negative results of the current study are supported by replication of the findings of Brantley et al. (1988a) involving the influence of daily minor stress on cortisol. Had the present study failed to find the expected positive relation between stress and
cortisol, the negative findings of the stress-arousal hypothesis on metabolic control may have been more easily challenged. Failure to find a stress-arousal influence on metabolic control supports previous research and suggests an arousal-mediated influence of minor stress on metabolic control may not be tenable.

While the results of this study are consistent with the previous literature on the stress-arousal relation, several possible explanations for the negative findings remain. Like the Kemmer et al. (1986) study, increases in cortisol activity may have been "biologically insufficient" to disrupt metabolic control. Although stress was predictive of cortisol in this study, the increases in cortisol may have been inadequate to reach the biological thresholds required to result in metabolic instability. It is possible that daily minor stressors, like laboratory stressors, do not have sufficient biological impact to cause autonomic activity adequate for metabolic disruption.

Second, cortisol alone may not accurately represent the activity of the autonomic nervous system. Assessment of a more global autonomic response including catecholamines and GH may more accurately reflect autonomic arousal. While Kemmer et al. (1986) did assess a number of other indicators of autonomic arousal in addition to cortisol, they failed to demonstrate a
relation between these biological indicators of stress and blood glucose control. However, Kemmer et al.'s results with other autonomic indicators have not been replicated with naturally-occurring stressors and were not assessed in the current study. Further research using an externally valid means of assessing stress and a broader spectrum assessment of autonomic activity will be required to address this question.

Finally, it may be that the sample of individuals with IDDM in the study are not representative of the general population with IDDM. Participants in the study were in relatively good control of their blood glucose and free from any long-term complications of the disease. Perhaps the effects of an arousal-mediated mechanism of stress becomes more apparent with increasing severity of disease and the presence of complications. In support of this possibility is Kemmer et al.'s (1986) finding that cortisol levels were higher in hyperglycemic IDDM patients compared to normoglycemic patients. Individuals with poorer metabolic control (i.e., more severe disease) may, therefore, be more susceptible to the effects of stress. It should be noted, however, that length of illness was not found to be related to either cortisol or stress in the present study suggesting no influence of this measure of disease severity.
Compliance

The preliminary correlations conducted indicated no relation between diet, exercise, and insulin compliance. This finding is consistent with previous research suggesting independence of the separate components of compliance (Schafer et al., 1983). Nonetheless, many investigations of the influence of compliance on metabolic control have used a composite or pooled compliance measure (e.g., Hanson et al., 1987a, 1987b) combining several aspects of compliance with treatment. The preliminary correlations of the present study suggest combining separate aspects of compliance may be inappropriate.

While the independence of the separate components of compliance found in this study must be interpreted cautiously because of the relatively small sample size, the finding may have important implications for future research. To more accurately portray the relation between compliance and metabolic control, future research would benefit from examination of independent compliance behaviors rather than an overall compliance construct. This would allow determination of the specific areas in which an individual may be experiencing difficulties and allow for development of interventions targeted at these problem areas.
Stress and Compliance

Daily minor stress was unrelated to diet, exercise, or insulin compliance in this study. In addition, no effect of the interaction between the separate components of compliance and stress was found suggesting independence of stress and compliance effects. These findings are consistent with the majority of past research (e.g., Cox et al., 1984; Halford et al., 1990) indicating independence of stress and compliance influences on metabolic control. However, the methodological improvements of the current study (i.e., quantified compliance assessment and use of a standardized means of assessing naturally-occurring stress) supports this body of literature and suggests stress-compliance relations may not influence short-term metabolic control in IDDM.

Insulin compliance was related to metabolic control in this sample. The relation between insulin compliance and blood glucose was in the expected direction; that is, the less compliant individuals were with taking their insulin (i.e., the higher the insulin deviation score), the poorer their metabolic control. Insulin compliance was found to predict 20% of the variance in metabolic control independent of the perceived impact of daily minor stress. These findings are consistent with required treatment for IDDM and make intuitive sense. If insulin is not taken, blood glucose levels rise.
Perhaps more surprising are the negative findings regarding the influence of diet compliance on metabolic control. Diet compliance accounted for only 5% of the variance in metabolic control in this study, but interpretation of these results must be made cautiously. The data analyses regarding diet compliance are not based on the total sample. As was discussed in the Methods section, the number of dietary exchanges prescribed was used as the denominator in the diet deviation ratio. Derivation of the number of exchanges prescribed was based on the total number of calories in a given individual's American Diabetes Association (ADA) prescribed diet (Powers, 1987). However, only 26 of the subjects in the study were able to indicate the number of calories prescribed in their ADA diets. The fourteen remaining subjects reported no knowledge of the number or type of calories they were meant to eat and therefore could not be included in the data analysis.

This finding suggests significant lack of knowledge regarding dietary recommendations for treatment of IDDM in at least a portion of the subjects in this study. In addition, it brings into question how one can reasonably expect individuals with IDDM to comply with dietary treatment recommendations if they do not have knowledge or understanding of these recommendations. That is, how can compliance with diet be accurately assessed if the people
with the disorder do not know what is expected of them?

While this finding is epiphenomenon to the specific objectives of the current study, it represents an important result. Do the negative results of the diet compliance data reflect an actual lack of relation between dietary adherence and metabolic control or are the results confounded by those subjects who indicated limited knowledge of their dietary treatment recommendations? Similarly, does the lack of reported knowledge about the number of calories prescribed in an ADA diet indicate a special case of noncompliance?

The means by which the data were analyzed in the current study does not allow for resolution of these questions. However, these results do suggest directions for both future research and clinical endeavors. Future research appears needed to determine if those individuals who report a lack of knowledge about their dietary treatment recommendations differ in other aspects of compliance behaviors (or more generally) from those individuals with IDDM who do report knowledge of this area of their treatment. Research to this end may help resolve some of the questions surrounding the compliance-blood glucose issues and would improve the accuracy with which the stress-compliance hypothesis can be investigated.

A second point to be gained from this finding suggests future research involving quantification of diet
compliance should seek to determine those individuals who are knowledgeable about their diet versus those who are not a priori. These results also have clinical implications and suggest that more frequent sessions with a nutritionist to review and revise dietary needs may be useful to improve both general IDDM care and dietary compliance.

Summary and Implications for Future Research

This study was conducted to examine the relation between daily minor stress, compliance behaviors, cortisol activity and short-term blood glucose control in IDDM. Improvements in methodology over previous research were made by simultaneously: a) employing a psychometrically sound and ecologically valid means of assessing stress; b) quantifying compliance measures; and c) using a valid, accurate, and proximal means of assessing metabolic control.

The results of the study indicated daily minor stress did not influence short-term metabolic control, either through a direct unmediated influence, via disruption of compliance with treatment, or an autonomically-mediated mechanism. Insulin compliance was found to negatively influence blood glucose control, however, diet and exercise compliance and cortisol activity had no impact on the dependent measure. Daily minor stress was found to predict cortisol activity.
The results of the current study suggest several lines for further research. First, within-subjects studies examining the role of naturally-occurring stress on metabolic control appear needed. These studies should be done in conjunction with efforts to more accurately quantify compliance measures as was done in the current study. Within-subjects designs would allow for determination of those individuals who may be most susceptible to the influence of stress on metabolic control. The ability to identify stress-reactive prone individuals may allow for improved treatment tailored to the specific needs of the patient.

Second, studies comparing samples of Type I and Type II diabetes appear to be needed to address potential differences in stress-responsivity in these two groups of patients. Available research suggests these studies may be best conducted using a longitudinal design allowing for examination of individual difference influences on outcome. In addition, future research should employ assessment of a wider range of indicators of autonomic activity including catecholamines and GH.
REFERENCES


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Hanson, C. L., Henggeler, S. W., & Burghen, G. A. (1987a). Model of associations between psychosocial variables and health-outcome measures of adolescents with IDDM. *Diabetes Care, 10*, 752-758.


APPENDICES
APPENDIX A

Demographic Data Form
Subject Number: ________________

Age: ________  Race/Ethnic Group: _____ White
               _____ Black
               _____ Other

Sex: M  F

Marital Status: _____ Married
                 _____ Widowed
                 _____ Divorced/Separated
                 _____ Never married

Education: _____ Graduate/Professional
            _____ College graduation
            _____ Partial college
            _____ High school graduation
            _____ Partial high school (10-11 years)
            _____ Junior high school (7-9 years)
            _____ Less than 7 years of school

Employment: _____ Full time
             _____ Full time homemaker
             _____ Part time
             _____ Student
             _____ Unemployed

Annual Income: _____ Less than $5,000
               _____ $7,500 - 10,000
               _____ $10,001 - 15,000
               _____ $15,001 - 25,000
               _____ $25,001 - 50,000
               _____ $50,001 - 100,000
               _____ $100,000+

Date Diabetes was first diagnosed: ____________

Age at Diagnosis: ________

Height: ________  Weight: ________
APPENDIX B

Informed Consent
I have been asked to participate as a subject in the research project titled The relation between stress and insulin-dependent diabetes mellitus under the direction of Diane Garrett, M.S. The purpose of the study is to examine the effects of daily stress on blood glucose in persons with Type I diabetes. It is the goal of this study to further knowledge about the effects of stress on individuals with diabetes.

I understand the experimenter will show me how to use a digital glucometer, how to lance my finger for a drop of blood, and how to record the reading on a monitoring form. I will be given forms to record my blood glucose, diet, exercise, insulin use, and daily stress. I will be asked to record these readings for three (3) consecutive days. I also understand I will be asked to collect a urine sample in the containers provided to me for the same three (3) day period. I understand I will be asked to freeze each urine sample at the end of the day and return the samples to the experimenter at the end of the study. The experimenter may phone me periodically to insure I am not having difficulty with any of the recording procedures.

Following the three (3) day monitoring period, I understand I will then be asked to return my forms, glucometer, and urine samples to the experimenter. I also will be asked to complete a questionnaire at that time. Before I leave I will be paid and the results of the study explained to me.

I understand there are no alternate procedures to this project other than to decline participation. I also understand there are no costs or special fees for participating.

I understand that I will not directly benefit from my participation in the research project. However, my involvement in this project will serve to further knowledge about the effects of stress on individuals with diabetes.

The potential risks associated with the study are minimal. When I return to the laboratory for the completion of the study, I understand I will receive monetary compensation of $5.00 for each day of home monitoring I completed if I complete all three days of monitoring.

I also understand that any information collected in this study will not identify me personally. I understand that
I will be assigned a number at the onset of my involvement in the study and that this number will be used for identification purposes. It has been explained to me that every effort will be made to insure my confidentiality by using an assigned number on all questionnaires and monitoring forms.

In signing this consent form, I state that I have read and understand the description of the monitoring forms and questionnaire as well as the following statements. I understand I will be given a copy of the consent form.

1. I understand that informed consent is required of all persons in this project.

2. The principal and alternate procedures, including the experimental procedures in this project have been identified and explained to me in a language that I can understand.

3. The risks and discomforts from the procedures have been explained to me.

4. The expected benefits from the procedures have been explained to me.

5. An offer has been made to answer any questions I may have about these procedures. If I have any questions, before, during, or after the study, I may contact Ms. Diane Garrett at (409) 772-6730.

6. I have been told that I may refuse to participate or stop my participation in this project without prejudice and without jeopardizing my medical care at UTMB. All new findings during the course of the research which may influence my desire to continue or not to continue to participate in this study will be provided to me as such information becomes available.

7. I have been told that the University of Texas Medical Branch like virtually all other universities in the United States, does not have a mechanism for compensation of the injured research subject. Therefore, I understand I cannot look to any such mechanism to receive financial remuneration for any such injuries resulting in my participation in this project. If physical injury occurs as a direct result of this research, emergency treatment which is available to the general public will be available to me. Neither UTMB or Diane Garrett, M.S. can assume financial responsibilities or liability for the expense of such treatment.
8. I understand that if I have any questions about my rights as a patient participating in this study or a research-related injury, I may contact Dr. E. Ray Stinson, Director of the Office of Sponsored Programs-Academic at (409) 772-2482.

9. I understand that I have a right to privacy and all information that is obtained in connection with this study and that can be identified with me will remain confidential as far as possible within state and federal law. However, information gained from this study and that can be identified with me may be released to no one other than the investigator and my physician. The results of this study may be published in scientific journals without identifying me by name.

I voluntarily agree to participate as a subject in the above named project.

_________________________  ___________________________
Date                     Signature of Subject

_________________________
Signature of Witness

Using language that is understandable and appropriate, I have discussed this project and the items listed above with the subject and/or his/her authorized representative.

_________________________  ___________________________
Date                     Signature of Project Director
The purpose of this research is to examine the effects of daily stress on blood glucose in persons with Type I diabetes. It is the goal of this study to further knowledge about the effects of stress on individuals with diabetes.

I understand the experimenter will show me how to use a digital glucometer, how to lance my finger for a drop of blood, and how to record the reading on a monitoring form. I will be given forms to record my blood glucose, diet, exercise, insulin use, and daily stress. I will be asked to record these readings for three (3) consecutive days. I also understand I will be asked to collect a urine sample in the containers provided to me for the same three (3) day period. I understand I will be asked to freeze each urine sample at the end of the day and return the samples to the experimenter at the end of the study. The experimenter will phone me periodically to insure I am not having difficulty with any of the recording procedures.

Following the three (3) day monitoring period, I understand I will then be asked to return my forms, glucometer, and urine samples to the experimenter. I also will be asked to complete a questionnaire at that time. Before I leave I will be paid and the results of the study explained to me.

I understand that any information about me obtained as a result of my participation in this research will be kept as confidential as legally possible. I understand I will be identified by number only. However, I also understand that my research records, like hospital records, can be subpoenaed by court order or may be inspected by federal regulatory authorities.

To the best of my knowledge I am not suffering from any impairment or disease that might interfere with this project. When I return to the laboratory for the completion of the study, I will receive a monetary compensation of $5.00 for each day of home monitoring I completed if I complete all three days of monitoring. There are no direct benefits to me other than the monetary compensation. Although there are minimal risks associated with my participation in this study, I understand some risks may be unforeseeable. Should injury occur as a result of this research, voluntary compensation is not provided. There are no costs or special fees for participating.
In signing this consent form, I state that I have read and understand the description of the monitoring and questionnaires. Any questions I have had have been answered to my satisfaction. I understand if I have any questions about my rights as a research participant I can contact the Institutional Review Board for the Protection of Human Subjects at (304) 293-7073.

I enter into this research willingly as a volunteer and may withdraw at any time without fear of retribution. Refusal to participate involves no retribution or penalty or loss of benefit to which I am entitled. I will be given a copy of the consent form. If I have any further questions or concerns I may contact the investigator.

__________________________
Subject's Signature

__________________________
Date

__________________________
Investigator's Signature

__________________________
Witness

Diane Garrett, M.S. 293-2411
APPENDIX C

Food Diary
FOOD DIARY

DAY #: 1          DATE: ____________

Please write down everything you eat or drink from the time you get up until you go to bed. Include drinks of all kinds. Specify the AMOUNT, HOW IT WAS PREPARED, AND ANYTHING THAT IS ADDED such as butter, margarine, fat, oil, salad dressing, sugar, syrup, etc.

<table>
<thead>
<tr>
<th>TIME</th>
<th>FOOD DESCRIPTION</th>
<th>AMOUNT</th>
</tr>
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<tbody>
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</table>
APPENDIX D

Insulin and Blood Glucose Log
**BLOOD GLUCOSE LOG**

**DAY #: 1**

**DATE:** __________

<table>
<thead>
<tr>
<th>Time</th>
<th>Before Breakfast</th>
<th>Before Lunch</th>
<th>Before Evening Meal</th>
<th>Before Bedtime</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Glucose Level Reading</td>
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<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**LOG FOR INSULIN USE**

<table>
<thead>
<tr>
<th>Time (indicate actual time)</th>
<th>Type of Insulin</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.M. Dose</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P.M. Dose</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please list any other medications and the amount taken today below:
APPENDIX E

Exercise Log
**EXERCISE LOG**

**DAY #: 1**  **DATE: __________**

<table>
<thead>
<tr>
<th>Time of Day</th>
<th>Type of Exercise</th>
<th>Duration of Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 a.m.</td>
<td>Jogging</td>
<td>15 min.</td>
</tr>
<tr>
<td>2 p.m.</td>
<td>Housework</td>
<td>45 min.</td>
</tr>
</tbody>
</table>

Pedometer Reading: __________
APPENDIX F

Prescribed Treatment Log
Prescribed Treatment Log

Below are listed a brief series of questions regarding treatment recommendations made for your care by your physician. Please indicate to the best of your knowledge what your physician asked you to do in each of the three areas below:

EXERCISE

__ 1. My physician recommended I exercise.

__ 2. How frequently did your physician suggest you exercise?

_____ Weekly

_____ Twice per week

_____ Three times per week

_____ More than 3 times per week

__ 3. How long did your physician suggest you exercise during each exercise period?

_____ Less than 10 minutes

_____ 10 - 19 minutes

_____ 20 minutes

_____ more than 20 minutes

__ 4. My physician DID NOT recommend I exercise as part of my prescribed treatment.

INSULIN USE

Please indicate amount, type, and timing of insulin injections prescribed for you.

TYPE ___________ AMOUNT ___________ TIME ______

__________________________________________________

__________________________________________________

__________________________________________________

__________________________________________________

DIETARY RECOMMENDATIONS

Please indicate the dietary recommendations prescribed for you.

_____ calorie ADA diet

_____ other, please explain:
VITA

Virginia Diane Garrett was born in Charlottesville, Virginia on April 2, 1956. She attended Augusta College in Augusta, Georgia where she received her Bachelor of Arts degree in Psychology in 1984. She received her M.S. in Psychology from the University of New Orleans in 1986, with a specialization in Applied Biological Psychology. Her psychology doctoral work was completed at Louisiana State University in 1992 following a Clinical Psychology Internship at West Virginia University Health Sciences Center.
DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Virginia Diane Garrett

Major Field: Psychology

Title of Dissertation: The Relation Between Stress and Insulin-Dependent Diabetes Mellitus: Physiologic Arousal or Disruption of Compliance?

Approved:

[Signature]

Major Professor and Chairman

[Signature]

Dean of the Graduate School

EXAMINING COMMITTEE:

[Signatures]

Date of Examination:

April 2, 1992