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HbA_{1c} levels as a function of emotional regulation and emotional intelligence in patients with type 2 diabetes

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ABSTRACT

Aims: Understanding the role of emotion in glycemic control may be critical for the long-term treatment of patients with type 2 diabetes (T2D). In this study we investigated the relationship between measures of emotional regulation and emotional intelligence and HbA_{1c} levels in adult patients with T2 diabetes.

Methods: 100 adult patients with T2 diabetes completed assessments of emotional regulation (i.e., affect intensity/lability) and emotional intelligence and were then correlated with HbA_{1c} levels with several relevant covariates.

Results: HbA_{1c} levels were significantly associated with affect intensity (AI: $r = .24$, $p = .018$) and with emotional intelligence (EI: $r = -.29$, $p = .004$), but not affect lability. These results were the same even after adding income, state depression scores, insulin-dependent status, serum cholesterol, diabetes literacy and self-care as covariates (AI: $\beta = .33$, $p = .001$; EI: $\beta = -.31$, $p = .002$). Diabetes self-care, but not diabetes literacy, was also associated with HbA_{1c} levels ($\beta = -.29$, $p = .003$).

Conclusions: These data suggest that aspects of emotional regulation and emotional intelligence play a role in glycemic control in adult patients with T2 diabetes and do so even in the context of several variables relevant to diabetes. If so, interventions that can reduce affect intensity and/or increase emotional intelligence may represent a new strategy in the glycemic control of adult patients with T2 diabetes.

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1. Introduction

Type 2 diabetes (T2D) is a major public health problem affecting millions of individuals worldwide [1]. Critically, less than 50% of patients with T2D achieve a glycemic target of $HbA_{1c} < 7.0\%$ and are at elevated risk to suffer from the medical complications of T2 diabetes, including early death.

A variety of factors, including clinical inertia, polypharmacy, overly complex medication regimens, health disparities, and psychiatric disorders, all of which are thought to reduce treatment compliance, have been suggested to account for inadequate glycemic control [2]. The last factor has been an ongoing focus of study because patients with T2 diabetes have substantial co-morbidity with various psychiatric disorders [3,4]. Specifically, depression has been very extensively studied as it has been estimated that patients with T2 diabetes are twice as likely to suffer from depression than the general population [5]. The burden of diabetes and comorbid depression is very significant as it has been linked with more diabetes-related complications, poorer adherence to dietary recommendations, poorer quality of life, poorer glycemic control, worse overall diabetes management and increased mortality [5–7]. Given all of these devastating consequences, coupled with the increasing rates of comorbid depression and T2 diabetes, multiple studies have been conducted to determine the exact mechanism by which these two conditions are related.

There has been much specific focus on the impact by which depression adversely impacts glycemic control, though there are no clear conclusions. Several pharmacologic and non-pharmacologic intervention studies have been conducted and, although these studies report reduced rates of depression, the impact on glycemic control is mixed with several studies reporting no association between improved depression and glycemic control [8,9]. Thus, targeting depression, for example, may not be enough to improve glycemic control [10,11], though attention to various constructs related to depression may be more effective in this regard [12].

Multiple psychological factors have been studied ranging from depression-related symptoms such as anhedonia to social-cognitive variables such as social support in depressed individuals with T2 diabetes. Arigo et al. [6] showed that social support mediated the relationship between depressive symptoms and glycemic control. In addition, apathy and perceived control are other psychological constructs implicated in glycemic control regardless of the severity of depression [7,13]. Diabetes-specific emotional distress has also been extensively studied and is reported as a better predictor of diabetes self-care than depression [7,14]. Recently, Nef et al. [15] reported that symptoms related to reduced positive affect (anhedonia) were linked to poorer glycemic control, while symptoms related to negative affect (dysphoria and anxiety) were not. Thus, depression, itself, may not be sufficient to explain less than optimal glycemic control in patients with T2 diabetes. However, given the strong predictive relationships of the more emotion/affect based constructs (i.e. emotional distress and anhedonia) the study of emotionality factors could provide an explanation regarding a biopsychosocial mechanism underlying glycemic control in patients with T2 diabetes.

Such emotionality factors may be defined as identifying, evaluating, and controlling emotions and their expression so as to not impair psychosocial function [16]. Processes involved in the intensity, and/or lability, of emotion fall under the area of emotional regulation (ER) while processes involved in identifying, understanding, and repairing emotions fall under the area of emotional intelligence (EI). While each of the above mentioned predictors (i.e. distress, anhedonia) is relevant to emotional regulation and emotional intelligence, such constructs have rarely been studied in adult patients with T2 diabetes. The one study that did assess emotional intelligence (though not emotional regulation) reported that T2D patients with higher levels of emotional intelligence skills used more effective coping strategies and scored higher on measures of emotional awareness, self-motivation, and managing emotions and stress [17]. In this intervention study, patients who participated in and emotional intelligence skills training program obtained improved emotional intelligence skills and improved glycemic control with decreased levels of anxiety and emotional burnout. To the best of our knowledge, this study [17] is the only investigation that has examined the relationship between emotional intelligence and glycemic control in patients with T2 diabetes. Related to emotional intelligence, Hughes et al. [12] reported that low emotional processing, along with low self-control, was associated with the poorest HbA_{1c} levels in adolescents with T1 diabetes. Further, this study noted that emotion processing better predicted HbA_{1c} levels than other variables such as self-efficacy and self-reported adherence to medical regimens [12]. There are no current studies examining the link between glycemic control and emotion regulation and emotional intelligence in patients with T2 diabetes and depression.

Thus, this study was designed to examine the relationship between psychometric measures of emotional regulation and emotional intelligence and glycemic control in adult patients with T2 diabetes. This is the first study to examine these specific variables in patients with T2 diabetes. We hypothesized a positive relationship between HbA_{1c} levels and impaired emotion regulation (affect intensity and affective lability), an inverse relationship between HbA_{1c} levels and emotional intelligence. In addition, we hypothesized that these emotion variables will account for a significant degree of variance in HbA_{1c} levels even in the context of other factors thought relevant to glycemic control.

2. Methods

2.1. Subjects

One hundred adult subjects with T2 diabetes were enrolled in this study. A sample size of 100 was chosen because it is capable of detecting medium-sized (e.g., r or $\beta = .25$) relationships between variables with 80% power at a two-tailed α of .05. Subjects were patients at the Kovler Diabetes Center (KDC) at the University of Chicago Medical Center (UCMC). Eligibility included a T2D diagnosis of at least one year, $\geq$21 years in age, and ability to read and write in English. Subjects were recruited between July 2013 and June 2015 by a research assistant in collaboration with the physicians and nurses at KDC. All