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Associations of circulating 25(OH)D with cardiometabolic disorders underlying type 2 diabetes mellitus in an Aboriginal Canadian community

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ABSTRACT

Aims: To investigate the associations of 25-hydroxyvitamin D (25(OH)D) with insulin resistance (IR), beta-cell function and metabolic syndrome (MetS) in a First Nations population.
Methods: We conducted a cross-sectional analysis using data from the Sandy Lake Health and Diabetes Project (2003–2005). A total of 390 participants (>12 y) were assessed for 25(OH)D, fasting glucose, insulin, lipids, blood pressure, inflammatory markers, anthropometric and lifestyle variables and a 75-g oral glucose tolerance test was administered. IR was calculated using the Matsuda insulin sensitivity index (IS_{OGTT}) and the computational homeostasis model assessment of IR (HOMA2-IR). Beta-cell function was calculated using the insulinogenic index (IGI) divided by HOMA-IR (IGI/IR) and the insulin secretion sensitivity index-2 (ISSI-2). The 2009 harmonized criteria were used to define MetS.
Results: Higher 25(OH)D was associated with a decreased prevalence of dysglycemia (OR = 0.71 95% CI, 0.51–0.97 per SD increase). In addition, there were significant associations of 25(OH)D with measures of insulin action (IS_{OGTT}; beta = 0.31; 95% CI, 0.12, 0.49; HOMA2-IR; beta = −29; 95% CI −0.46, −0.11 and beta-cell function (ISSI-2; beta = 0.15; 95% CI, 0.02, 0.28). The prevalence of MetS was 41%. There was a decreased risk (OR = 0.73, 95% CI 0.56, 0.94) of

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MetS per SD increase in baseline 25(OH)D. Finally, there was a significant positive association of 25(OH)D with adiponectin (beta = 0.16; 95% CI = 0.01, 0.31).
Conclusions: These results support a potential role for vitamin D metabolism in the natural history of T2DM among Aboriginal Canadians, although carefully designed randomized trials will be required to establish causality.
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1. Introduction

Type 2 diabetes mellitus (T2DM) has emerged as a critical public health problem for Aboriginal Canadian communities [1]. The prevalence of T2DM in this population has been steadily increasing since the 1950s, and particularly high rates have been documented in First Nations communities [2,3]. Although important progress has been made in the scientific, health promotion and policy arenas to address this health crisis [4–6], recent data suggest that the prevalence and incidence of T2DM continue to increase [7].

T2DM is characterized and predicted by a number of underlying physiological disorders including insulin resistance (IR), pancreatic beta-cell dysfunction and the metabolic syndrome (MetS) [8]. While several important risk factors for T2DM have been identified including central obesity, family history, physical inactivity and suboptimal dietary patterns [1], major gaps remain in our understanding of the causes of this disease. Recent research has suggested that low vitamin D status, which is known to cause rickets and other bone disorders, may have a potential role in the etiology of T2DM [9] and its underlying disorders, including insulin resistance, beta-cell function and the MetS [10]. While limited research has indicated that vitamin D levels are low in Aboriginal communities, likely due to diet transition, low sun exposure, and other factors [11], the relationship between vitamin D status and diabetes and its physiological traits has been investigated in only two previous studies which have yielded inconsistent results [12,13]. Further, no studies have investigated the potential association of 25(OH)D with MetS in this population.

In addition to the measurement of total 25(OH)D in assessing vitamin D status, there has been increasing interest in measuring bioavailable and free 25(OH)D. Recent studies on bone mineral density [14] and renal osteodystrophy [15] found that bioavailable 25(OH)D was a better biomarker for vitamin D status than total 25(OH)D; however to date no study has investigated these measures in the context of T2DM. Therefore, given these knowledge gaps, the objective of this study was to explore the association of different 25(OH)D measures with IR, beta-cell dysfunction and MetS in a First Nations population at risk for T2DM.

2. Subjects

The Sandy Lake Health and Diabetes Project is a population-based study designed to determine the etiology of diabetes and its associated risk factors in an Aboriginal Canadian community. This study was approved by both the Sandy Lake First Nation Band Council and the University of Toronto Ethics Review Committee. Between 2003 and 2005, 485 individuals participated in an evaluation of diabetes and its associated risk factors including obesity, impaired glucose tolerance and impaired fasting glucose [16]. After excluding participants who did not have information on relevant outcome measures, 387 men and women remained in the analysis examining the association of serum 25(OH)D, the primary measure of vitamin D status, with the prevalence of dysglycemia. Analyses of associations between 25(OH)D and IR and beta-cell function were restricted to those without known T2DM at the time of participant assessment ($n = 361$). In the end, analyses of associations between 25(OH)D and MetS was undertaken on 390 participants (Supplementary material, Fig. S1).

3. Materials and methods

Blood samples were collected after an 8- to 12-h overnight fast [16], and a 75-g oral glucose tolerance test (OGTT) was administered, with additional blood samples collected at 30-min and 2-h postload for glucose and insulin measurements. Glucose concentration was determined using the glucose oxidase method. Specific insulin was measured using the Elecsys 1010 (Roche Diagnostics, Basel, Switzerland) immunoassay analyzer and electrochemiluminescence immunoassay. For the assessment of IR and β-cell function, validated indices based on glucose and insulin values from the OGTT were used. The IS_{OGTT} (Matsuda) index was used as a measure of insulin sensitivity (the inverse of IR), defined as $10{,}000/\sqrt{(FPG \times FPI) \times (G \times I)}$, where FPG = fasting plasma glucose, FPI = fasting plasma insulin, G = mean glucose, I = mean insulin [17]. This index is a measure of whole body insulin sensitivity, and it has been validated against the euglycemic-hyperinsulinemic clamp technique [17]. The Homeostasis Model of Assessment of Insulin Resistance (HOMA-IR) was also calculated, which is defined as $FPG \times FPI/22.5$ [18]. This index has also been validated against the clamp and it is considered as a measure of hepatic IR [19]. The HOMA2-IR index was obtained from the HOMA Calculator v2.2.3 [20].

Beta-cell function was estimated using the insulinogenic index divided by HOMA-IR (IGI/IR), which is calculated as the ratio of (30 min insulin—fasting insulin) to (30 min glucose—fasting glucose) [21]; it has been validated against 1st phase insulin secretion during intravenous glucose tolerance testing [21] and has been shown to predict the development of T2DM [22]. The Insulin Secretion Sensitivity Index 2 (ISSI-2), which is

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