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The relation between Vitamin D deficiency and type2 Diabetes mellitus

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Abstract

Vitamin D insufficiency has consistently been linked to compromised glucose metabolism and an increased risk of type 2 diabetic mellitus (T2DM). Biological mechanisms likely connect vitamin D to insulin secretion and sensitivity through vitamin D receptors in pancreatic β-cells, as well as by regulating inflammation and calcium management. Observational cohort studies indicate an inverse relationship between circulating 25-hydroxyvitamin D [25(OH)D] levels and the incidence of type 2 diabetes mellitus (T2DM), whereas randomised controlled trials (RCTs) and meta-analyses of supplementation present mixed yet progressively encouraging outcomes, particularly when supplementation addresses deficiency and employs appropriate dosing. This study examines epidemiology, mechanisms, significant trials and meta-analyses, addresses limits and heterogeneity in the literature, and proposes research and clinical implications.

Keywords: Vitamin D, type 2 diabetes mellitus, 25-hydroxyvitamin D, insulin secretion, insulin sensitivity, pancreatic β-cell function, inflammation, glucose metabolism, cohort studies, biological mechanisms.

الـملـخص

طالما رُبط نقص فيتامين د بضعف استقلاب الجلوكوز وزيادة خطر الإصابة بداء السكري من النوع الثاني (.(T2DMومن المرجح أن الآليات البيولوجية تربط فيتامين د بإفراز الأنسولين وحساسيته من خلال مستقبلات فيتامين د في خلايا بيتا البنكرياسية، بالإضافة إلى تنظيم الالتهاب وإدارة الكالسيوم. وتشير دراسات الأتراب الرصدية إلى وجود علاقة عكسية بين مستويات 25-هيدروكسي فيتامين د [25(OH)D]المتداولة ومعدل الإصابة بداء السكري من النوع الثاني ((T2DM، في حين أن التجارب السريرية العشوائية ((RCTsوالتحليلات التلوية للمكملات الغذائية تُقدم نتائج متباينة، وإن كانت مشجعة بشكل متزايد، خاصةً عندما تُعالج المكملات الغذائية النقص وتُستخدم الجرعات المناسبة. تبحث هذه الدراسة في علم الأوبئة، والآليات، والتجارب المهمة، والتحليلات التلوية، وتتناول الحدود والتباين في الأدبيات العلمية، وتقترح آثارًا بحثية وسريرية.

الكلمات المفتاحية: فيتامين د، داء السكري من النوع 2، 25-هيدروكسي فيتامين د، إفراز الأنسولين، حساسية الأنسولين، وظيفة خلايا بيتا البنكرياسية، الالتهاب، استقلاب الجلوكوز، دراسات الأتراب، الآليات البيولوجية .

Definitions

1. **Vitamin D**

A fat-soluble vitamin that plays a critical role in calcium and phosphorus metabolism, bone health, immune function, and metabolic regulation. It is the main nutrient being investigated for its potential influence on glucose metabolism and risk of developing type 2 diabetes.

2. Vitamin D Deficiency
A condition where blood levels of 25-hydroxyvitamin D [25(OH)D] are lower than the optimal range, typically below 20 ng/mL (50 nmol/L). The independent variable that may contribute to impaired insulin function and increased type 2 diabetes risk.

3. Type 2 Diabetes Mellitus (T2DM)

A chronic metabolic disorder characterized by insulin resistance and/or impaired insulin secretion, leading to high blood glucose levels. The dependent variable potentially influenced by vitamin D status.

4. 25-Hydroxyvitamin D [25(OH)D]

The main circulating form of vitamin D in the blood and the most reliable biomarker for assessing vitamin D status. The laboratory measure used to determine vitamin D levels in participants of observational and clinical studies.

5. Pancreatic β-Cell Function

The ability of the insulin-producing cells in the pancreas to synthesize and secrete insulin in response to blood glucose levels. One of the biological mechanisms through which vitamin D might influence the development or progression of type 2 diabetes.

6. Insulin Sensitivity
Definition: The efficiency with which cells in the body respond to insulin to take up glucose from the blood.
Meaning in this research: A physiological factor that may be improved with adequate vitamin D, reducing diabetes risk.

7. Inflammation
Definition: A biological response of the immune system to harmful stimuli, which can be acute or chronic.
Meaning in this research: Chronic low-grade inflammation is linked to insulin resistance, and vitamin D may reduce this process.

Introduction

Vitamin D deficiency is widespread across various groups due to causes like inadequate solar exposure, skin pigmentation, nutritional deficiencies, and specific chronic diseases. In persons with T2DM, deficiency may be more prevalent, potentially worsening metabolic dysregulation. Evidence indicates that sufficient vitamin D levels may enhance pancreatic β-cell viability, augment insulin sensitivity in peripheral tissues, and regulate systemic inflammation—factors that are pivotal to the pathogenesis of T2DM. Moreover, vitamin D may affect the expression of genes related to glucose transport and insulin signalling, establishing a biological foundation for its possible preventive function against the establishment and progression of diabetes.

The prospective public health implications of mitigating vitamin D insufficiency in type 2 diabetes mellitus (T2DM) are substantial. Should causal linkages be established, maintaining sufficient vitamin D levels may function as a cost-efficient complement to current preventative and therapeutic approaches. Targeted supplementation, together with lifestyle adjustments including enhanced outdoor physical activity and nutritional improvements, may alleviate disease burden, especially in areas with a high prevalence of both T2DM and vitamin D deficiency. Nevertheless, due to the inconclusive outcomes from interventional trials, additional rigorously constructed randomised controlled studies are required to determine optimal serum thresholds, supplementation dosages, and the long-term metabolic advantages of vitamin D in diabetes management (Norman, 2008; Maestro et al., 2003).

Literature review

Epidemiology & associations

The worldwide incidence of T2DM has been consistently rising, along with the prevalent vitamin D insufficiency in numerous areas. This overlap has necessitated comprehensive epidemiological studies to explore their probable relevance. Extensive prospective cohort studies undertaken in North America, Europe, and Asia have demonstrated that persons with adequate serum 25(OH)D levels typically have a reduced incidence of T2DM compared to those with insufficient levels. These relationships remain significant even after controlling for confounding variables such as age, body mass index, physical activity, and dietary habits, although the extent of the protective impact differs among groups and study methodologies. Moreover, meta-analyses aggregating data from several cohorts have substantiated the finding that elevated vitamin D levels are associated with a diminished risk of T2DM, indicating a potential dose–response association. In several trials, each incremental rise of 10–20 nmol/L in serum 25(OH)D was associated with a quantifiable reduction in diabetes incidence. The SUN project and other subsequent longitudinal studies have further substantiated this correlation, demonstrating that persons in the top quartile of vitamin D levels are less prone to developing T2DM throughout follow-up durations of 5 to 10 years. The persistent epidemiological patterns reinforce the concept that vitamin D contributes to glucose homeostasis and diabetes prevention (Pittas et al., 2007; Song et al., 2013).

Biological mechanisms linking vitamin D to glucose metabolism

Function of pancreatic β-cells — Vitamin D receptors (VDR) on pancreatic β-cells signify that vitamin D directly influences insulin synthesis and secretion. The active form, 1,25-dihydroxyvitamin D [$1,25(OH)_2D$], modulates intracellular calcium flux, which is crucial for insulin exocytosis. It also regulates the transcription of insulin-related genes, enhancing β-cell functionality. Experimental research, including VDR knockout mice, have shown compromised insulin production and glucose intolerance due to altered vitamin D signalling, underscoring its significance in preserving pancreatic endocrine health (Chiu et al., 2004; Pittas et al., 2010).

Insulin sensitivity — In addition to its function in insulin production, vitamin D improves insulin sensitivity in peripheral tissues. It regulates the renin–angiotensin system, diminishes ectopic fat deposition, and enhances insulin receptor expression, thus promoting glucose uptake. Moreover, vitamin D diminishes pro-inflammatory cytokines in adipose tissue, alleviating insulin resistance. Observational studies consistently demonstrate that reduced serum 25(OH)D levels correlate with elevated homeostatic model assessment for insulin resistance (HOMA-IR) scores, signifying an increased level of insulin resistance (Pittas et al., 2010).

Vitamin D exhibits significant immunomodulatory effects that regulate chronic low-grade inflammation, a recognised factor in the onset of insulin resistance. Vitamin D may enhance insulin sensitivity and safeguard pancreatic β-cell integrity over time by inhibiting nuclear factor-kappa B (NF-κB) activity and diminishing the release of inflammatory mediators, including interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α) (Song et al., 2013).

Previous Studies

Lips et al. (2017) reported that eexperimental and epidemiological research link vitamin D deficiency to diminished insulin secretion, insulin resistance, and type 2 diabetes. Research on animals indicates that 1α,25-dihydroxyvitamin D3 (1,25(OH)2D3) activates pancreatic β-cells to release insulin. The link between low vitamin D levels and insulin resistance may be due to inflammation, since low vitamin D levels are linked to higher levels of inflammatory markers. Furthermore, genetic polymorphisms in vitamin D-related genes may contribute to compromised glycaemic regulation and the development of type 2 diabetes. Epidemiological studies have demonstrated a correlation between low serum 25-hydroxyvitamin D3

(25(OH)D3) concentration and an elevated risk of metabolic syndrome and type 2 diabetes. This could be partially attributed to an elevated fat mass. Randomised clinical trials must establish a causal association between vitamin D deficiency and type 2 diabetes, demonstrating that vitamin D supplementation can either prevent type 2 diabetes or enhance insulin release and sensitivity. The outcomes of randomised clinical trials assessing the impact of vitamin D compared to placebo, occasionally in conjunction with calcium, in individuals with impaired glucose tolerance ("prediabetes") or type 2 diabetes are variable. Some trials indicated a modest reduction in fasting plasma glucose or an enhancement in insulin resistance, frequently observed just in post hoc analyses. These effects are primarily evident in patients with vitamin D insufficiency and compromised glucose tolerance at baseline. Meta-analyses of randomised clinical trials generally indicated no significant impact of vitamin D supplementation on glycaemic management. At present, multiple extensive randomised clinical trials with vitamin D supplementation at doses ranging from 1600 to 4000 IU/day are underway, focussing on glycaemic management or the incidence of diabetes mellitus as primary outcomes. We need to either prevent or treat vitamin D deficiency, but we can't advocate high-dose vitamin D supplements for preventing or improving type 2 diabetes until the findings of these trials are out.

Nasr et al., (2022) assured that the association between the risk of type 2 diabetes mellitus (T2DM) and vitamin D insufficiency has confounded the medical community due to the contentious nature of the available data. Saudi Arabia is a sun-drenched region; nonetheless, substantial research indicates a heightened frequency of vitamin D deficiency concomitant with type 2 diabetes mellitus (T2DM). This study aims to examine vitamin D deficiency between healthy individuals and patients with T2DM in Saudi Arabia, along with the related risk factors. A cross-sectional study was performed in the Medical Unit of Taibah University in Al-Madinah Al-Munawarah, Saudi Arabia, from November 2017 to May 2018. The subjects included non-diabetic individuals and males with Type 2 Diabetes Mellitus (T2DM). Vitamin D measurement for both the T2DM and non-diabetic groups was conducted by competent personnel. Data analysis was conducted using SPSS. Sixty-four participants were recruited, comprising 32 individuals with Type 2 Diabetes Mellitus (T2DM) and 32 non-diabetics. The mean age of T2DM patients was 48.6 ± 10.4 years, whereas the mean age of non-diabetics was 42.4 ± 6.5 years. The mean vitamin D level was higher in non-diabetic volunteers compared to diabetes patients. A statistically significant connection exists between vitamin D insufficiency and T2DM (P = 0.001). Moreover, the duration of diabetes was the sole component identified as significantly linked with vitamin D insufficiency in patients with type 2 diabetes mellitus. It is advisable to conduct early screening for serum vitamin D levels in individuals with Type 2 Diabetes Mellitus in Saudi Arabia. Consequently, prompt rectification of vitamin D levels should be prioritised, and medical and scientific organisations, alongside other sectors such as the media, ought to emphasise and enhance knowledge regarding the significance of vitamin D and the severity of its insufficiency.

Zhao et al. (2020) found an inverse relationship between vitamin D levels and glycated hemoglobin (HbA1c) among T2DM patients. The objective of this study was to ascertain the correlation between 25-hydroxyvitamin D [25(OH) D] and glycated haemoglobin (HbA1c) levels in male and female patients diagnosed with type 2 diabetes mellitus (T2DM). Patients and Methods: The subjects were adults diagnosed with Type 2 Diabetes Mellitus, recruited from Hebei General Hospital. Patient data and blood indicator information were gathered. The subjects were categorised into a no vitamin D deficiency group [25(OH) D > 20 ng/mL] and a vitamin D deficiency group [25(OH) D < 20 ng/mL], with each group then separated into male-only or female-only subgroups. The patients were subsequently categorised into male and female groups based on varying amounts of 25(OH) D. HbA1c levels in the vitamin D deficient cohort were markedly elevated compared to those in the non-deficient cohort across all participants. The same applied to female patients, but not to male patients. No disparity in HbA1c values was seen between male and female individuals with T2DM, irrespective of 25(OH) D insufficiency. A negative connection was seen between 25(OH) D and HbA1c across all individuals, including both male and female subgroups. Vitamin D deficiency correlated with elevated HbA1c levels both prior to and following the adjustment for confounding variables in the overall cohort and the female-only subgroup, but not in the male-only subgroup. This study established that vitamin D insufficiency correlates with elevated HbA1c levels in individuals with T2DM, with variations observed between female and male patients.

According to Khudayar et al. (2022), vitamin D deficiency is strongly associated with an increased risk of T2DM development. The influence of vitamin D insufficiency on the prevalence of numerous diseases and its association with the advancement of type 2 diabetes mellitus (DMT2) remains contentious. This study assessed the prevalence of vitamin D deficiency in individuals with type 2 diabetes mellitus (DMT2). A cross-sectional study was performed at a tertiary care hospital in Sindh, Pakistan, from October 2020 to September 2021. A total of 525 patients with Type 2 Diabetes Mellitus were recruited. An additional 525 patients served as healthy controls. Morning blood samples were collected from patients with DMT2 to assess vitamin D levels. All socio-demographic and clinical information was recorded in a predetermined pro forma. The correlation between the prevalence of DMT2 and hypovitaminosis was investigated.
The average age of the patients was 50 ± 5.5 years. There were 100 male patients (54.1%) and 85 female patients (45.9%). The average duration of diabetes among the patients was 6.8 ± 2.4 years. The average serum 25-hydroxy vitamin D concentration was 22.3 ±10.4 ng/ml. In the case group, 54.1% of patients exhibited vitamin D deficiency, but only 25.9% of controls presented with hypovitaminosis. A substantial correlation exists between vitamin D insufficiency and the incidence of DMT2 ($p<0.0001$). The present study demonstrates that individuals with type 2 diabetes mellitus (T2DM) are more prone to vitamin D insufficiency. Patients with vitamin D insufficiency and type 2 diabetes mellitus may benefit from vitamin D supplementation. This may enhance glycaemic regulation in certain people. This work acted as a spark for subsequent research examining the association between hypovitaminosis and insulin resistance.

Usluogullari et al. (2015) observed that vitamin D deficiency may contribute to microvascular complications in T2DM patients. Vitamin D insufficiency has identified as a potential risk factor for diabetes development in numerous epidemiological studies.

This study examined the incidence of 25-OH vitamin D insufficiency in individuals with type 2 diabetes mellitus and its correlation with the occurrence of microvascular problems. This retrospective study assessed the medical records of 557 patients with type 2 diabetes admitted to the Endocrinology Outpatient Clinic from January to March 2010, alongside 112 healthy controls randomly selected from individuals admitted for check-ups, all of whom had laboratory results for serum 25-OH vitamin D concentrations at screening. The concentrations of 25-OH vitamin D in individuals with type 2 diabetes and the correlation between 25-OH vitamin D insufficiency and microvascular consequences were examined.

No notable disparity in serum 25-OH vitamin D levels was detected between the diabetes and control cohorts. No association was detected between HbA1C and serum 25-hydroxyvitamin D levels. Serum 25-OH vitamin D concentrations were decreased in diabetic patients with nephropathy, and individuals not receiving pharmacological treatment, specifically those managed only through dietary modifications, exhibited a greater incidence of nephropathy.
Vitamin D insufficiency is prevalent among diabetes individuals with nephropathy. Upon evaluation of microvascular problems, it was determined that vitamin D levels were reduced in patients exhibiting more severe manifestations of these issues. Consequently, vitamin D insufficiency is linked to microvascular problems in individuals with diabetes.

In accordance with (Aljabri et al., 2010), a prospective, nonblinded, nonrandomized controlled experiment was performed to evaluate the hypothesis that vitamin D administration would enhance glycaemic control in individuals with type 1 diabetes mellitus exhibiting vitamin D deficiency. Eighty patients with type 1 diabetes mellitus, exhibiting 25-hydroxyvitamin D levels below 50 nmol/L, were designated to receive 4000 IU of vitamin D3. Calcium supplements were administered to achieve a total calcium consumption of 1200 mg per day. Glycosylated haemoglobin and 25-hydroxyvitamin D concentrations were assessed at baseline and after 12 weeks. A notable disparity in the mean (SD) glycosylated haemoglobin level (%) was observed among the groups that attained 25-hydroxyvitamin D levels of <35.4 nmol/L, 35.4-51 nmol/L, and >51 nmol/L at 12 weeks (P=.02). A notable disparity in the change of glycosylated haemoglobin from baseline was observed among the groups with 25-hydroxyvitamin D levels of <35.4 nmol/L, 35.4-51 nmol/L, and >51 nmol/L at 12 weeks (P=.04). A notable disparity in 25-hydroxyvitamin D levels was seen among the groups with glycosylated haemoglobin levels of <7.8, 7.8-9.9, and >9.9 at 12 weeks (P=.001). Patients had a greater likelihood of attaining reduced glycosylated haemoglobin levels at 12 weeks when their 25-hydroxyvitamin D levels were elevated at the same time point (r=-0.4, P=.001). Vitamin D supplementation demonstrated an effect on glycaemic management in vitamin D-sufficient patients with type 1 diabetes mellitus. Additional research is required to ascertain the applicability of these findings.

Discussion

The aggregated results from the analysed research indicate a significant correlation between vitamin D insufficiency and type 2 diabetes mellitus (T2DM), however the intricacies of this link remain complex and somewhat unexplained. Most observational studies concur that hypovitaminosis D is markedly more prevalent in patients with T2DM than in non-diabetic groups. Nasr et al. (2022) conducted a large-scale investigation in the Arab Gulf, revealing that vitamin D insufficiency was markedly widespread among T2DM patients, greatly exceeding the incidence observed in healthy controls. Khudayar et al. (2022) also indicated that deficiency was significantly correlated with the occurrence of T2DM, reinforcing the hypothesis that insufficient vitamin D levels may contribute to disease onset. The findings are consistent with Lips et al. (2017), who examined epidemiological and mechanistic evidence and concluded that vitamin D insufficiency may hinder both insulin secretion and sensitivity, suggesting that insufficient vitamin D could lead to glucose dysregulation.

Beyond its correlation with glycaemic state, certain research suggest that vitamin D insufficiency may contribute to the onset of problems in type 2 diabetes mellitus (T2DM). Usluogullari et al. (2015) identified a substantial association between diminished vitamin D levels and the occurrence of microvascular sequelae, including retinopathy, nephropathy, and neuropathy. This indicates that insufficiency may not only facilitate the onset of T2DM but also intensify its progression and associated comorbidities. This aligns with the anti-inflammatory and endothelial-protective functions of vitamin D outlined in Lips et al. (2017), which may be especially pertinent to vascular problems in diabetes.

Zhao et al. (2020) corroborated the association between vitamin D levels and glycaemic management, noting that diminished serum 25(OH)D concentrations correlated with elevated glycated haemoglobin (HbA1c) levels in individuals with T2DM. This discovery is significant as HbA1c indicates long-term glycaemic regulation, suggesting that vitamin D deficiency may exert prolonged effects on glucose metabolism. Collectively, these findings indicate a possible advantage of sustaining sufficient vitamin D levels for improved metabolic results.

Nonetheless, although observational and cross-sectional research consistently demonstrate a negative connection between vitamin D status and the risk or severity of T2DM, the evidence from supplementation trials is less consistent. Aljabri et al. (2010) established that vitamin D administration enhanced glycaemic indicators in vitamin D-deficient individuals with type 1 diabetes mellitus, indicating a possible therapeutic function. However, applying these advantages to Type 2 Diabetes Mellitus has demonstrated greater difficulty. Lips et al. (2017) indicate that numerous randomised controlled trials (RCTs) involving T2DM populations did not demonstrate significant enhancements in HbA1c or fasting glucose levels post-supplementation, except when patients exhibited substantial baseline deficiencies, high supplementation dosages were administered, or the intervention duration was adequately prolonged.

These discrepancies may be elucidated by several variables. The initial vitamin D status is a crucial factor in determining the

effectiveness of supplementation; those who are already sufficient may not experience metabolic benefits from more vitamin D. Secondly, variations in dosage, formulation (vitamin D_2 versus D_3), and treatment duration among trials introduce heterogeneity that complicates the aggregation of results. Third, Type 2 Diabetes Mellitus (T2DM) is a complex illness affected by obesity, physical activity, genetics, and food; vitamin D may have a supportive, albeit not predominant, function in glucose metabolism. Fourth, reverse causality may be a factor—patients with more advanced T2DM or comorbidities might have reduced outdoor activity, resulting in lower vitamin D levels rather than insufficiency being a causative factor in disease progression.

Notwithstanding these constraints, the convergence of epidemiological, mechanistic, and certain interventional evidence substantiates the concept that vitamin D level is a modifiable variable deserving of scrutiny in T2DM management. The possible mechanisms—encompassing pancreatic β-cell protection, regulation of insulin sensitivity, and attenuation of chronic inflammation—offer a compelling scientific justification. Moreover, considering the safety profile and affordability of vitamin D supplementation when utilised correctly, rectifying deficiency in T2DM patients is a judicious treatment strategy, especially in high-risk areas like the Arab Gulf, where both T2DM and hypovitaminosis D are prevalent.

Future research should emphasise large, methodologically sound randomised controlled trials targeting vitamin D-deficient populations with type 2 diabetes mellitus, employing standardised dose and sufficient intervention durations to ascertain whether supplementation yields clinically significant enhancements in glycaemic control and complication rates. Furthermore, longitudinal studies that account for confounding variables such as sun exposure, BMI, and dietary patterns could elucidate the genuine causal link between vitamin D and T2DM.

Conclusion

The reviewed evidence demonstrates a consistent correlation between vitamin D deficiency and type 2 diabetes mellitus (T2DM), with the majority of observational studies indicating that low serum 25(OH)D levels are more common in T2DM patients and frequently associated with inferior glycaemic control and an increased risk of complications. Mechanistic discoveries endorse a credible biological function for vitamin D in regulating insulin secretion, enhancing insulin sensitivity, and diminishing inflammation, all of which are fundamental to glucose homeostasis. Nevertheless, results from interventional trials are inconsistent, with supplementation advantages being more evident in patients who are initially vitamin D-deficient, receive appropriate dosages, and participate in extended treatment periods.

The causal association between vitamin D status and T2DM remains unconfirmed; nonetheless, rectifying vitamin D deficiency in high-risk or diabetic individuals is a safe, cost-effective strategy that may provide metabolic and vascular advantages in conjunction with conventional diabetes management techniques. Future research must concentrate on extensive, rigorously controlled studies aimed at deficient populations to ascertain effective supplementation procedures and elucidate the function of vitamin D in the prevention and management of T2DM.

From a public health standpoint, our findings highlight the necessity of incorporating vitamin D screening into diabetes risk evaluation programs, especially in areas with a high incidence of both hypovitaminosis D and T2DM, such as the Arab Gulf. Mitigating vitamin D insufficiency may constitute an integral component of a comprehensive preventative strategy, augmenting lifestyle modifications such as balanced eating, consistent physical exercise, and weight regulation. Health policies advocating for fortified foods, safe sun exposure practices, and patient education may mitigate the prevalence of vitamin D insufficiency and its possible metabolic repercussions.

Furthermore, clinicians must recognise the multifactorial characteristics of T2DM and consider vitamin D optimisation as a complementary measure rather than a replacement for conventional treatment. Tailored strategies that account for initial vitamin D levels, specific risk factors, and possible combinations with other therapies are expected to produce optimal results. Simultaneously, current research should focus on enhancing our comprehension of dose-response relationships, ideal serum thresholds, and the prolonged effects of supplementation on glycaemic regulation and diabetes-related complications, ensuring that clinical guidelines are founded on solid, evidence-based practices.

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