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EDITORIAL

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Dear Readers
this edition provides an update on some skeletal and extra-skeletal effects of vitamin D.
As you know, so-called stress fractures are caused by repetitive loads and mechanical stresses that exceed the bone tissue's ability to repair itself and are especially common among athletes, military personnel and individuals who engage in strenuous physical activity. Well, patients who suffer from it frequently present vitamin D deficiency and it is also known that adequate vitamin D levels accelerate bone callus formation and improve the quality of bone regeneration. This seems to be attributable to a dual role of vitamin D: the immunomodulation role in the first acute inflammatory phase of "fracture healing" and the mineralisation role.
The second article contains an important update on the possible role of vitamin D in reducing the risk of developing type 2 diabetes. The rationale has long been there: vitamin D also has intranuclear receptors in pancreatic beta cells and could therefore play a role in glucose homeostasis. Observational studies have indeed documented an association between hypovitaminosis D and the presence of type 2 diabetes, but interventional studies with vitamin D supplementation have so far reported conflicting results on glycaemic control and insulin resistance in subjects with prediabetes. Furthermore, there were few studies to date in the general population and on the possible role of genetic variants of the vitamin D receptor. Hence the importance of a recent large prospective cohort study that observed a significant association between circulating 25(OH)D levels above 75 nmol/L and reduced risk of developing type 2 diabetes compared to subjects with 25(OH)D levels below 25 nmol/L, regardless of prediabetes status and especially in the presence of certain genetic polymorphisms. This has been considered in the *Endocrine Society's* new vitamin D guidelines[1] that, in recommendation no. 10, suggests vitamin D supplementation, in addition to lifestyle correction, in individuals at high risk of prediabetes to reduce the risk of progression to type 2 diabetes.
The same new guideline [1] recommends for the first time vitamin D supplementation in children and adolescents up to 18 years of age not only to prevent rickets but also to reduce the risk of respiratory tract infections, recognising the specific extra-skeletal benefit of vitamin D.
Another important and original acknowledgement of an extra-skeletal benefit by the same guidelines [1] is the sixth recommendation, which recommends vitamin D supplementation in all individuals over 75 years of age due to the possibility of reducing the risk of mortality. This reminds me of the report I had made to the Italian Medicines Agency (AIFA) in my capacity then as President of the Italian Society of Osteoporosis, Mineral Metabolism and Skeletal Diseases (SIOMMMS) in relation to note 96 [2]: I pointed out that the note neglects the elderly by not providing them, regardless of the 25(OH)D determination, with supplementation by the National Health Service, despite the fact that they are understandably and notoriously at risk of chronic deficiency. Among the effects of note 96 on the prescription of vitamin D, as later reported by AIFA [3], there was indeed a reduction in the use of vitamin D, even in the elderly [3], a fact

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that I find worrying and not an expression of improved appropriateness of use.
The new recommendation in the recent guidelines [1] on vitamin D supplementation in all elderly people also reminds me of the Project started in the Veneto Region 20 years ago [4]which envisaged vitamin D supplementation in the entire elderly population, particularly during the winter months.

What do you think?
Enjoy reading!

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Vitamin D deficiency, stress fractures and post-traumatic recovery

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Summary

Vitamin D is essential for intestinal absorption of calcium and phosphate, as well as for maintaining good muscle performance and optimal immune function. In fact, consistently low vitamin D levels impair skeletal mineralisation and increase the risk of bone fractures. Among these, stress fractures, caused by repeated mechanical stress, have been associated with vitamin D deficiency and are a common problem among athletes and military personnel. Correcting and maintaining adequate vitamin D levels, together with optimising calcium levels, is one of the most effective strategies for strengthening the skeleton and, consequently, preventing the risk of fractures. Therefore, this review offers an overview of the mechanisms by which vitamin D affects bone health and post-traumatic recovery, providing a solid basis for future research and clinical interventions.

INTRODUCTION

Vitamin D is an essential nutrient that plays a crucial role in maintaining bone health. Its importance is well documented not only for the prevention of bone diseases, but also for its role in the modulation of the immune system, muscle contraction and the prevention of chronic diseases. However, vitamin D deficiency is a widespread problem globally, influenced by various factors, including seasonality, latitude, obesity, malnutrition, as well as acute inflammation and infection that may reduce serum vitamin D levels [1].

Vitamin D deficiency has been associated with a higher incidence of bone fractures, including stress fractures, caused by repetitive loads and mechanical stress, which are common among athletes, military personnel and individuals who engage in strenuous physical activity. The ability of bone to repair these micro-damages depends largely on the availability of essential nutrients, including vitamin D. Numerous evidences suggest that vitamin D deficiency may impair bone mineralisation, increasing susceptibility to fractures caused by stress. Furthermore, post-traumatic recovery from stress fractures is a complex process that requires adequate nutritional support to ensure effective healing. Vitamin D plays a key role in bone regeneration and fracture healing, accelerating the recovery process, improving the quality of bone callus and reducing immobilisation time [2].

In a context where the prevalence of vitamin D deficiency is increasing, it is essential to fully understand its implications on bone health and to identify the best practices for its management. Therefore, our review aims to explore the role of vitamin D in stress fracture prevention and post-traumatic recovery by analysing the association between vitamin D deficiency and increased incidence of fractures, as well as the benefits of vitamin D supplementation in the healing process.

ROLE OF VITAMIN D IN BONE HEALTH

Vitamin D is a fat-soluble vitamin crucial for regulating calcium and phosphorous metabolism. Vitamin D can be obtained through exposure to sun, which induces skin synthesis of vitamin D_3 or cholecalciferol, and through the intake of foods and supplements containing vitamin D_2 or ergocalciferol, and vitamin D_3. In the organism, vitamin D is converted in the liver to 25-hydroxyvita-

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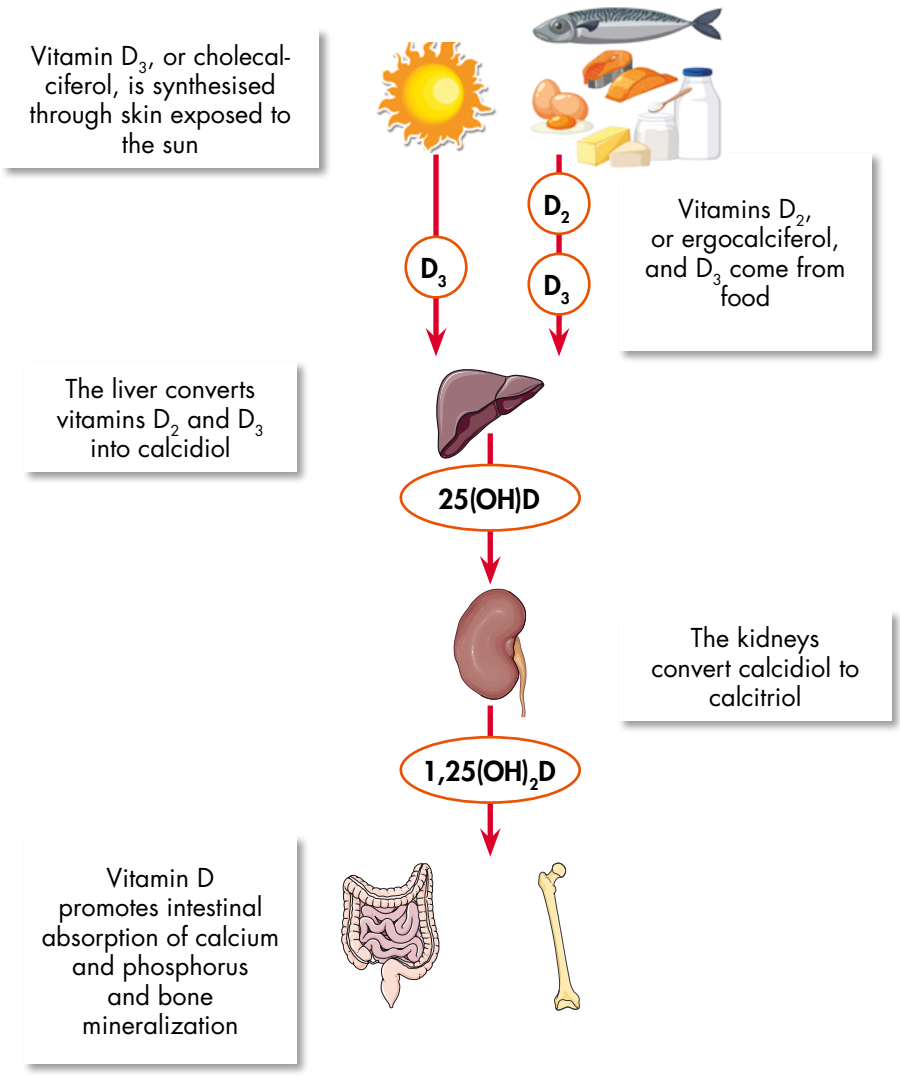

FIGURE 1.
Vitamin D synthesis and metabolism.

min D [25(OH)D] or calcidiol, the main circulating form. In the kidneys, 25(OH)D is converted into its active form, 1,25-dihydroxyvitamin D [$1,25(OH)_2D$], known as calcitriol, which acts on specific receptors in various tissues, contributing to the maintenance of homeostasis [3] (Fig. 1).
Numerous studies have shown that adequate levels of vitamin D are associated with increased bone mineral density, a key indicator of strength and endurance of the bones. It promotes the intestinal absorption of calcium and phosphorus, which are necessary for the mineralisation of the bone matrix. However, under conditions of vitamin D deficiency, calcium absorption is inefficient, causing hypocalcaemia. This condition stimulates the secretion of parathormone (PTH), which mobilises calcium from the bones to maintain serum calcium levels, causing bone demineralisation and increasing the risk of fractures [4].
Vitamin D increases the expression of calcium-binding proteins in the gut, facilitating the trans-cellular transport of calcium into the bloodstream. Furthermore, it acts directly on bone cells, stimulating the activity of osteoblasts and reducing the activity of osteoclasts. These actions are associated with the presence of the vitamin D receptor (VDR), a nuclear receptor that, by binding to calcitriol, regulates the expression of genes involved in calcium metabolism, cell growth and immune function. In fact, VDR dysfunction can alter tissue homeostasis, contributing to the onset of musculoskeletal disorders, including osteoporosis and sarcopenia [5]. In addition, severe vitamin D deficiency can cause rickets in children, a condition characterised by defects in bone mineralisation leading to skeletal deformities, while in adults it can cause osteomalacia, a condition in which the mineralisation of newly-formed bone is inadequate, causing muscle weakness and widespread bone pain [6].
Numerous epidemiological and clinical studies support the role of vitamin D in fracture prevention. In particular, a meta-analysis of randomised clinical trials showed that vitamin D supplementation, especially when combined with calcium, significantly reduces the risk of fractures in the elderly suffering from vitamin D deficiency [7]. Another study showed that patients with stress fractures frequently have insufficient vitamin D levels, suggesting that proper supplementation could prevent such injuries [8]. Overall, this evidence confirms the role of vitamin D in maintaining optimal bone mass and suggests the need to monitor and maintain adequate vitamin D levels, especially in individuals at risk, through adequate sun exposure, a balanced diet and, if necessary, vitamin D supplementation.

STRESS FRACTURES AND POST-TRAUMATIC RECOVERY: THE ROLE OF VITAMIN D

Stress fractures are injuries caused by repeated micro-trauma that exceeds the repair capacity of bone tissue. Vitamin D deficiency is a significant risk factor for the development of these fractures, as this vitamin is crucial for the health and adaptation of bones to mechanical stresses. This type of fracture is common in the lower limbs, where the bones bear the weight of the body and repeated impacts during activities such as running and jumping [9].
Several studies have shown that individuals with low vitamin D levels have an increased risk of stress fractures, especially among athletes, because they are exposed to repeated loads, and military personnel, subjected to intense physical activities. In particular, it has been shown that changes in training protocol, equipment used or the start of a new sport, especially in non-professional athletes, are frequent causes of stress injuries, suggesting the importance of vitamin D supplementation during periods of intense training or service [10].
Post-traumatic recovery from bone fractures

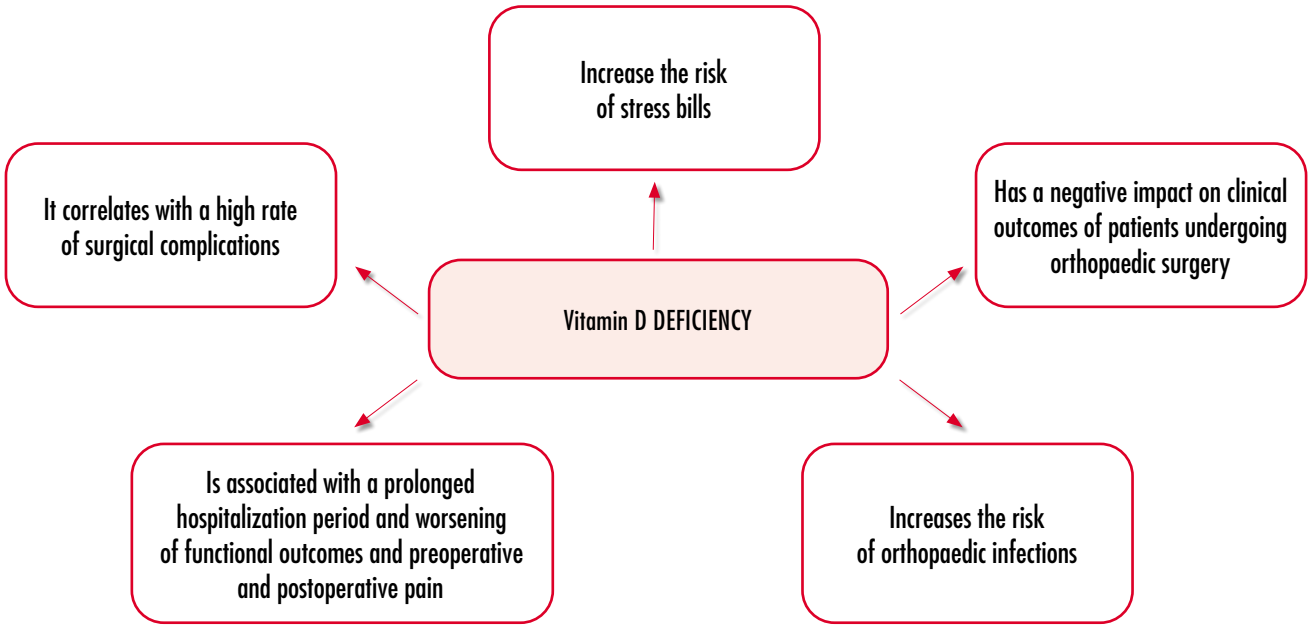

FIGURE 2.
Vitamin D deficiency: consequences in the orthopaedic field.

is also a complex process that requires adequate nutritional support to ensure effective healing. In this context, vitamin D plays a crucial role due to its ability to modulate the activity of osteoblasts and osteoclasts, ensuring an essential dynamic balance for bone repair. Several evidences have shown that adequate levels of vitamin D accelerate bone callus formation and improve the quality of bone regeneration [11]. Therefore, vitamin D deficiency can significantly impair the fracture healing process, causing poor bone callus formation, prolonging healing time and increasing the risk of complications, such as *non-union* (Fig. 2). In this respect, vitamin D-deficient patients with fractures show faster healing and better quality of bone callus if they receive vitamin D supplementation compared to deficient subjects [12]. Another study showed that patients with femur fractures treated with vitamin D and calcium had significantly reduced healing times compared to the control group without supplementation [13]. Therefore, the physiological action of vitamin D is a key element in the post-traumatic healing process, essential both in the inflammatory phase, due to its immuno-modulating properties, and for the formation, mineralisation and remodelling of bone callus.

PREVENTION AND MANAGEMENT OF VITAMIN D DEFICIENCY

Prevention and management of vitamin D deficiency are crucial to maintain bone health and prevent consequences such as stress fractures. In this context, skin synthesis of vitamin D through exposure to sunlight is the main source of vitamin D for many people. It is advisable to expose oneself to the sun for about 15 to 30 minutes a day, although factors such as latitude, season and skin pigmentation may influence the amount of vitamin D produced. In addition, a diet rich in foods containing vitamin D is essential. Some good sources of vitamin D include fish, such as salmon, mackerel and tuna, cod liver oil, egg yolks, beef liver and fortified foods such as milk, orange juice and cereals. Incorporating these foods into the daily diet can help maintain adequate levels of vitamin D [14]. Nevertheless, in many cases, vitamin D supplementation is necessary, especially for people at risk of deficiency, such as the elderly, individuals with limited sun exposure and those with absorption problems. In these individuals, regular monitoring of blood calcidiol levels is important to control and manage vitamin D levels. Blood tests can help determine whether supplementation doses are adequate or whether adjustments are needed. Overall, the prevention of vitamin D deficiency and its adequate supplementation require a multifactorial approach that must include sun exposure, a balanced diet and, when necessary, supplementation [15].

CONCLUSIONS

Vitamin D is essential for bone health, preventing stress fractures and improving post-traumatic recovery. This is especially true for individuals at risk of stress fractures, such as athletes and military personnel, whose intense physical activity subjects the bone tissue to continuous stress and overloads that could favour the development of micro-damage and, consequently, stress fractures. A deficiency of this vitamin impairs bone mineralisation and prolongs healing time. In order to prevent and manage this deficiency, adequate sun exposure, a vitamin D-rich diet and, if necessary, supplementation are recommended. For individuals at risk of deficiency, regular monitoring of vitamin D levels is essential to maintain bone health and reduce the risk of stress fractures.

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Circulating levels of vitamin D and risk of developing type 2 diabetes mellitus: is there a link?

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Diabetes mellitus affects more than 500 million people worldwide and its prevalence, especially type 2 diabetes, has been steadily increasing in recent decades (with an estimated global increase of around 50 per cent in 2045). Globally, deaths due to diabetes and its chronic complications in 2019 are estimated to be around 6 million [1]. Impaired fasting blood glucose and impaired glucose tolerance describe prediabetic conditions. These two conditions, both individually and in combination, are also very frequent worldwide (affecting approximately 7-10% of the global population) and represent not only risk factors for the development of type 2 diabetes mellitus, but also risk factors associated with the development of long-term vascular and kidney function complications [1]. In the absence of effective therapeutic strategies (which are mainly based on lifestyle changes), approximately 5-10% of the population with prediabetes progresses to type 2 diabetes each year.

Vitamin D deficiency/insufficiency has been associated with the coexistence of multiple extra-skeletal chronic diseases (including obesity, cardiovascular disease, certain forms of neoplasia, diabetes and nonalcoholic fatty liver disease (NAFLD)), suggesting the possibility that vitamin D may have multiple beneficial pleiotropic effects at the extra-skeletal level due to the ubiquitous distribution of its specific receptor [2-4]. Vitamin D, in fact, has intranuclear receptors that are expressed on many cells and tissues, including pancreatic beta cells, and thus appears to play a role in glucose homeostasis [2,5,6]. Observational studies have shown an association between low serum vitamin D levels and the presence of type 2 diabetes. Although some intervention studies have suggested that vitamin D supplementation may exert a potential beneficial effect on blood sugar control and the degree of insulin resistance, large-scale works and some meta-analyses of randomised clinical trials have reported conflicting data [7]. For instance, in the randomised client trial D2d, which enrolled approximately 2,400 adult subjects with prediabetes, regardless of their basal vitamin status, oral supplementation with vitamin D_3 for 24 months did not reduce the risk of developing diabetes compared to placebo [8]. In contrast, a recent meta-analysis of 4.190 participants, which included individual data from three large randomised clinical trials (including the D2d trial), showed that vitamin D supplementation in subjects with prediabetes (in particular, in subjects who maintained circulating 25(OH)D values ≥ 125 nmol/L [≥ 50 ng/ mL] during the trial compared to those with 25(OH)D values between 50 and 74 nmol/L) was effective in reducing the risk of developing type 2 diabetes by approximately 15% over ~3 years of treatment [9]. However, this observation is not necessarily translatable to the general adult population with normal fasting blood sugar. In particular, there are currently few epidemiological studies in the literature conducted in the general adult population that have assessed the risk of developing type 2 diabetes mellitus within the entire spectrum of carbohydrate tolerance (i.e. in the presence of normal blood sugar levels and forms of prediabetes, which include impaired fasting blood sugar levels and reduced carbohydrate tolerance). Furthermore, it is still not entirely clear whether genetic variants of the vitamin D receptor (VDR), which is expressed in multiple tissues, are able to modulate the association between vitamin D status and long-term risk of developing diabetes.

A recent prospective cohort study, which was published in April 2024 in the *Journal of Clinical Endocrinology & Metabolism* by Fu et al. [10], tried to answer these questions. To do this, the authors used data from a

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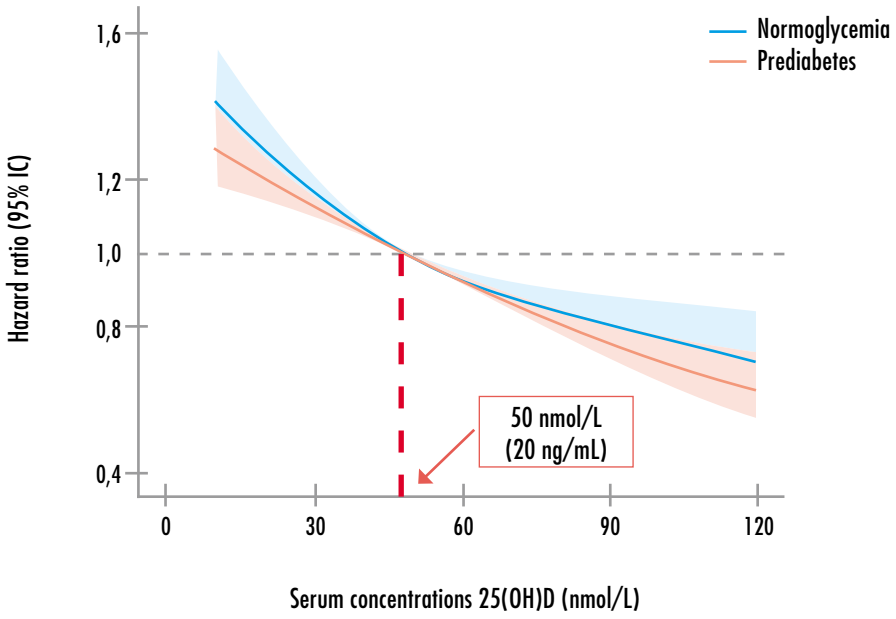

FIGURE 1.
Dose-response type relationship between circulating 25(OH)D levels and risk of developing type 2 diabetes mellitus during follow-up (median of approximately 14 years) in subjects with normal glucose tolerance and subjects with prediabetes at baseline. In both subject groups, the risk of diabetes was progressively reduced in subjects who had 25(OH)D values ≥ 50 nmol/L (≥ 20 ng/mL) at baseline. On the y-axis, data are expressed as hazard ratios and 95% confidence intervals (95% CI, represented as shaded areas in blue and red) after statistical adjustment for possible confounding variables (taken from Fu et al., 2024, mod.) [10].

large observational cohort study, the *UK Biobank database*, which recruited over 500,000 British adult subjects aged 40-69 years between 2006 and 2010. Subjects who had diabetes at baseline (based on their clinical history and/or HbA1c levels) and those without serum 25(OH)D dosage and measurement of four specific VDR genetic polymorphisms (rs7975232 *ApaI*; rs1544410 *BsmI*; rs2228570 *FokI*; rs731236 *TaqI*) were excluded from the study. Information regarding the diagnosis of diabetes during the follow-up period was obtained from the analysis of hospital admission records and death records.
In the study by Fu et. [10] Thus, a total of 379,699 adult individuals without diabetes at baseline (average age 56 years, 54% women) were included; 86% of these subjects had normal glucose tolerance (defined as HbA1c < 5.7%), while the remaining 14% (n = 53,886) had prediabetes at baseline (defined as HbA1c between 5.7% and 6.5%). Participants with normal glucose tolerance at baseline had a mean of 25(OH)D of 48 nmol/L (IQR: 33.5-63.4 nmol/L), while those with pre-diabetes had a mean of 25(OH)D of 45 nmol/L (IQR: 30.9-60.3 nmol/L). Overall, in the entire study cohort 53.4% of the subjects had circulating 25(OH)D values < 50 nmol/L. During the follow-up of the study (median 14 years), 6,315 (1.9%) normal blood sugar level subjects and 9,085 (16.9%) subjects with prediabetes developed type 2 diabetes mellitus.
When study participants were divided according to their circulating 25(OH)D values at baseline in accordance with the cutoffs proposed by the *Endocrine Society* [25(OH)D < 25, 25-49.9, 50-74.9 and ≥ 75 nmol/L], the authors observed a significant association between higher circulating levels of 25(OH)D and reduced risk of developing type 2 diabetes. In particular, compared to subjects who had 25(OH)D levels < 25 nmol/L, subjects with normal blood sugar levels and 25(OH)D values ≥ 75 nmol/L at baseline had a significantly reduced risk of developing type 2 diabetes (hazard ratio: 0.62, 95% CI: 0.56-0.70); similarly, compared with subjects who had 25(OH)D levels < 25 nmol/L, subjects with prediabetes and 25(OH)D values ≥ 75 nmol/L at baseline had a significantly reduced risk of developing diabetes (hazard ratio: 0.64, 95% CI: 0.58-0.70). These data remained significant even after statistical adjustment for gender, age, race, obesity, physical activity, economic status, use of medication for dyslipidemia and hypertension, use of vitamin D supplements, and multiple other possible confounding factors. The results remained significant even when cases of diabetes occurring in the first two years of follow-up of the study were excluded from the statistical analysis. The authors observed that there was a reverse, linear relationship between levels of 25(OH)D and risk of developing diabetes in subjects with prediabetes, whereas this relationship was significant but not linear (but inverse polynomial) in subjects with normal HbA_{1c} values at baseline. For each increment of 10 nmol/L in the circulating values of 25(OH)D at baseline, there was a 7% decrease in the risk of developing diabetes. Furthermore, both in subjects with normal glucose tolerance and in those with prediabetes at baseline the risk of developing diabetes during follow-up was progressively reduced in subjects who had 25(OH)D values ≥ 50 nmol/L (Fig. 1). The authors also reported a statistically significant interaction between 25(OH)D levels and the presence of genetic polymorphisms of the VDR in subjects with prediabetes (but not in those with normal blood sugar levels at baseline); in these subjects, the protective effect of elevated 25(OH)D levels on the risk of developing diabetes was greater in subjects carrying the T allele (rs1544410) of the *BsmI* gene (TT allele carriers: hazard ratio: 0.53, 95% CI: 0.38-0.73; CT alleles: hazard ratio: 0.65, 95% CI 0.55-0.77; CC alleles: hazard ratio: 0.75, 95% CI: 0.61-0.91). Finally, in a statistical mediation analysis, the authors also demonstrated that plasma lipids, in particular plasma triglyceride levels, mediate a significant part of the association between 25(OH)D levels and risk of incident diabetes, both in subjects with normal glucose tolerance (26 per cent) and in those with prediabetes (34 %) at baseline. In particular, if an individual had both low 25(OH)D levels and high circulating levels of triglycerides, his risk of developing di-

abetes during follow-up was much higher than in subjects who only had an isolated alteration [10].

The main strengths of this cohort study are its prospective design, the large number of samples examined (about 380,000 subjects), the length of follow-up (about 14 years), the statistical adjustment for common risk factors and multiple confounding factors. The main limitations of the study include the observational design of the study (in fact, it should be remembered that this is not a study of supplementation/pharmacological intervention with vitamin D and, therefore, the presence of a significant association between 25(OH)D and the risk of diabetes does not automatically mean that there is causality!), the lack of measurement of circulating 25(OH)D levels, the inclusion of British subjects aged between 40 and 69 years and predominantly Caucasian, the lack of measurement of fasting blood glucose at baseline (having available only the HbA1C values) and the fact that the diagnosis of incident diabetes during the follow-up period was based on the analysis of medical records of hospital admissions and death records [10].

Therefore, the results of this British population study (with subjects aged between 40 and 69 years) document that high circulating 25(OH)D levels at baseline are significantly associated with a reduced risk of developing type 2 diabetes over an average follow-up period of approximately 14 years, both in subjects with normal glucose tolerance and in those with prediabetes at baseline. In this cohort of subjects, the serum vitamin D level where possible protective effects on the risk of developing type 2 diabetes began to be observed was ≥ 50 nmol/L (≥ 20 ng/mL). In subjects with prediabetes, the association between high 25(OH)D levels and reduced risk of diabetes was also modified by the presence of genetic variants of the VDR (rs1544410) of the *BsmI* gene. From the data of this study, it can finally be hypothesised that the improvement of the lipid profile (in particular the reduction of plasma triglyceride levels) may help to explain at least part of the protective effect of 25(OH)D levels on the risk of developing type 2 diabetes mellitus [10].

In conclusion, the results of this large prospective cohort study (using the *UK Biobank database*) provide further significant support for the possibility that adequate circulating levels of vitamin D may have beneficial effects on the risk of developing type 2 diabetes mellitus in the general adult population.

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