frontiers in Endocrinology

TYPE Perspective
PUBLISHED 23 November 2026
DOI 10.3389/fendo.2026.2023205

Check for updates

OPEN ACCESS

EDITED BY
Rolf Jorde,
UiT the Arctic University of Norway, Norway

REVIEWED BY
Faraz Zandiyeh,
Shahid Beheshti Medical University, Iran

*CORRESPONDENCE
Dylan Thompson
d.thompson@bath.ac.uk

RECEIVED 09 October 2026
REVISED 19 October 2026
ACCEPTED 22 October 2026
PUBLISHED 23 November 2026

CITATION
Thompson D, Perkin O, Davies SE, Hewison M and Jones KS (2026) Physical activity plays a direct role in the regulation of vitamin D metabolism and status.
Front. Endocrinol. 17:2023205.
doi: 10.3389/fendo.2026.2023205

COPYRIGHT
© 2026 Thompson, Perkin, Davies, Hewison and Jones. This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY). The use, distribution or reproduction in other forums is permitted, provided the original author(s) and the copyright owner(s) are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms.

Physical activity plays a direct role in the regulation of vitamin D metabolism and status

Dylan Thompson[1]*, Oliver Perkin[1], Sophie E. Davies[2], Martin Hewison[3] and Kerry S. Jones[4]

[1]Centre for Nutrition, Exercise and Metabolism (CNEM), Department for Health, University of Bath, Bath, United Kingdom, [2]Cardiff School of Sport and Health Sciences, Cardiff Metropolitan University, Cardiff, United Kingdom, [3]Department of Metabolism and Systems Science, University of Birmingham, Birmingham, United Kingdom, [4]Nutritional Biomarker Laboratory, University of Cambridge, Institute of Metabolic Science - Epidemiology, Cambridge, United Kingdom

To date, efforts to address vitamin D insufficiency have focused primarily on increasing dietary vitamin D intake, particularly using supplements. In this perspectives article, we review recent evidence demonstrating that physical activity exerts direct positive effects on vitamin D metabolism, and we propose that interventions designed to increase physical activity may be a complementary strategy to improve individual and population vitamin D status. Recent evidence from carefully conducted randomised controlled trials indicates that physical activity has causal effects on vitamin D metabolism in humans. A single bout of exercise causes an acute increase in the concentration of circulating 25(OH)D and 1,25$(OH)_2D_3$ in the hours after each bout. Furthermore, in the longer term, regular exercise (without weight loss) ameliorates the normal seasonal decline in serum 25(OH)D during winter and completely prevents the decline in 1, 25$(OH)_2D_3$. Thus, recent evidence demonstrates that physical activity affects vitamin D metabolism and status over both hours and months, including increased concentrations of biologically active 1, 25$(OH)_2D_3$. These findings have important implications for individuals, researchers and policymakers. Physical activity interventions and policies could be used as an adjunct to target population vitamin D insufficiency and, furthermore, some of the benefits of physical activity could be mediated by vitamin D-related pathways.

KEYWORDS
25OHD, exercise, metabolism, physical activity, vitamin D

Introduction

Vitamin D is the collective term for a group of seco-steroids that play a role in the maintenance of bone health as well as the regulation of a wide range of physiological processes, including immune function, metabolic health, and skeletal muscle function (1). Following synthesis in the skin or from dietary intake, vitamin D_3 (and diet-derived vitamin D_2) undergoes hydroxylation in the liver to form 25-hydroxyvitamin D [25(OH)D], the major circulating form of vitamin D and primary status marker. This metabolite is subsequently converted in the kidneys into the biologically active form, 1, 25-dihydroxyvitamin D [1, 25$(OH)_2D_3$], which directly binds to the vitamin D receptor (VDR) to mediate the biological actions of vitamin D in a wide variety of target cells.

In humans, the primary source of vitamin D is cutaneous synthesis following exposure to ultraviolet B (UVB) radiation in sunlight (2). In countries at northern latitudes, such as the UK, seasonal reductions in UVB exposure contribute to a high prevalence of vitamin D insufficiency during winter, with almost half the population exhibiting serum 25(OH)D concentrations below 50 $nmol{\cdot}L^{-1}$ (3).

Low vitamin D status is conventionally tackled through strategies that focus on augmented dietary intake of vitamin D, including supplementation and/or food fortification. In the UK, the Scientific Advisory Committee on Nutrition (SACN) recommends a minimum reference nutrient intake of 10 µg per day for everyone (> 4 years) (4). In the US, the Department of Health and Human Services (DHHS) recommends a dietary intake of 15-20 µg/d for adults (5). Neither SACN nor DHHS consider a potential role for physical activity and exercise in the management of vitamin D insufficiency (4, 5). In this perspectives article, we discuss recent evidence from carefully designed Randomised Controlled Trials (RCTs) which demonstrate that physical activity exerts direct effects on vitamin D metabolism and causally impacts vitamin D status – and we propose that physical activity could be used as an adjunct in the management of individual and population vitamin D insufficiency.

Physical activity and exercise – a brief primer

Physical activity represents any movement (or force) exerted by skeletal muscle that leads to an increase in energy expenditure above rest (6). Exercise is usually described as a subcomponent of physical activity that is planned and/or structured (6). Incidental low-to-moderate intensity activities of daily life (e.g., occupation, chores, transportation) account for the vast majority of physical activity energy expenditure, even in people who do 3–4 substantial bouts of structured exercise a week (7, 8). Whilst incidental activity is more important for overall daily energy expenditure, some specific forms of structured exercise (e.g., higher intensity, or a specific mode – such as resistance training) can exert unique physiological changes and longer-term adaptations irrespective of relatively modest effects on energy expenditure (9). Historically, habitual physical activity has been measured using self-report (e.g., questionnaires or diaries), with a particular focus on physical activity only conducted during leisure time. Such subjective and fragmented approaches are increasingly being replaced with more robust and objective methods (e.g., wearable devices) due to their far superior accuracy, even in large scale studies (10).

Associations between habitual physical activity and vitamin D status

Dozens of cross-sectional studies have observed positive associations between higher levels of physical activity and systemic concentrations of 25(OH)D (e.g (11–18). Given the nature of these observations, it is often speculated that these relationships are the product of other factors rather than physical activity *per se* (e.g., greater UV exposure due to people with higher physical activity spending more time outside) (19). It is noteworthy that the relationship between objectively-determined physical activity and 25(OH)D is strongest during winter months when UV exposure is minimal (20). Thus, it is unlikely that this relationship is solely the product of differences in sunlight exposure. However, it is possible that the relationship between exercise and vitamin D is the product of another confounder (e.g., diet). Clearly, observational studies can only go so far to address this issue and more robust evidence from RCTs is required to understand the causal role of physical activity and exercise in vitamin D metabolism.

The causal effects of acute physical activity on vitamin D metabolism

The onset of physical activity triggers thousands of highly coordinated physiological responses across multiple tissues and systems (9). These include profound physiological responses – such as changes to the rate and distribution of blood flow, increases in metabolic fluxes and substrate utilisation, as well as changes to the concentration of circulating hormones and metabolites (9). At a cellular level, there are changes to the expression of hundreds of genes and proteins across many different tissue types (21). Some acute changes, such as heightened adipose tissue lipolysis, are durable responses that extend over 24h (22). Thus, even a single bout of physical activity triggers an integrated cascade of physiological changes throughout the entire body over minutes and hours – many of which have the potential to affect vitamin D metabolism.

Several studies have reported higher concentrations of circulating vitamin D metabolites after a bout of exercise using simple pre/post measurements (23–25). However, such observations cannot be considered causal because of the lack of a control resting condition to account for potential diurnal changes. Our recent crossover randomised controlled trial in men and women directly addressed this knowledge gap (26). We examined the effect of a single bout of laboratory-based moderate intensity exercise (*versus* rest) on vitamin D metabolites assessed using LC-MS/MS in 33 men and women. Exercise increased both 25(OH)D and 1, 25$(OH)_2D_3$ (Figures 1A, B). The increase in 1, 25$(OH)_2D_3$ was maintained for at least one hour after exercise. For context, the form of moderate intensity exercise used in this study is similar to brisk walking. Thus, a single bout of a relatively accessible form exercise increases both 25(OH)D and the biologically active form of vitamin D, 1, 25$(OH)_2D_3$. This robust causal evidence supports the notion that regular exercise produces transient windows of enhanced vitamin D biological action associated with each activity bout.

The causal effects of chronic exercise on vitamin D metabolism and status

Over weeks and months, participation in regular physical activity leads to extensive physiological changes and adaptations

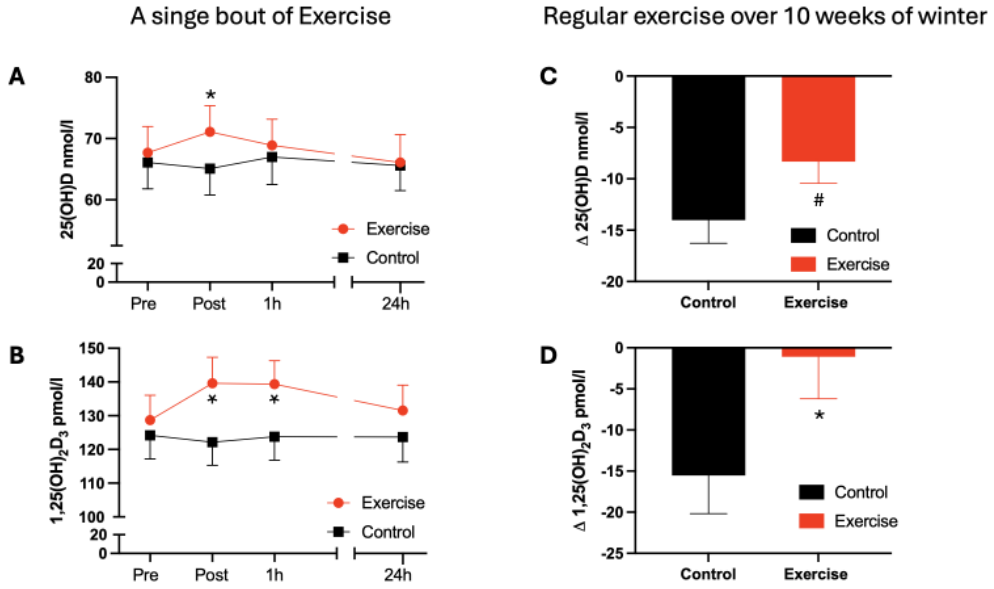

FIGURE 1
Effect of short- and long-term exercise on vitamin D metabolites in humans. Time course of responses for 25(OH)D [panel **(A)**] and 1, 25$(OH)_2D_3$ [panel **(B)**] to a single bout of moderate intensity exercise (60 min) or control (rest) [data redrawn from Davies et al. (26)]. Changes in 25(OH)D [panel **(C)**] and 1, 25$(OH)_2D_3$ [panel **(D)**] over 10-weeks of winter in men and women allocated to an Exercise Intervention or Control (lifestyle maintenance) group (Data redrawn from Perkin et al. (27)). Data represent means±SE. *significantly different to corresponding control condition ($P < 0.05$), #($P = 0.07$).

including changes to tissue structures and composition, altered metabolic fluxes and basal substrate utilisation, and hundreds of other coordinated sustained physiological and molecular changes (9). These changes include altered basal concentrations of many hormones and metabolites (9). Khan and colleagues attempted to determine if vitamin D is also one of the things affected by regular exercise through a systematic review and meta-analysis of exercise and physical activity interventions published before 2022 that included measures of 25(OH)D (28). The main conclusion was that the evidence available from trials at this time was weak. The available trials were not explicitly designed to address this question, were poorly controlled, and generally examined only 25(OH)D (often measured using ELISA, rather than the current gold-standard Liquid Chromatography- Tandem Mass Spectrometry (LC-MS/MS) analyses). In 2025, we published the VitaDEx Randomised Controlled Trial which directly addressed this knowledge gap (27).

VitaDEx examined the causal effect of regular exercise on systemic vitamin D concentrations in sedentary overweight men and women. The primary outcome was 25(OH)D, but we also determined 1, 25$(OH)_2D_3$ and various other metabolites using LC-MS/MS. To remove any confounding associated with sunlight exposure, the intervention data collection period was restricted to three consecutive winters in the UK. The prescribed and monitored exercise (4 times per week over 10 weeks) was primarily at a moderate intensity which is associated with high rates of adipose tissue lipolysis (approximately equivalent to brisk walking), with the assumption that lipophilic vitamin D sequestered in adipose tissue would be mobilised alongside the mobilisation of stored lipid to support augmented energy expenditure (see "Mechanisms" section). Crucially, we compensated dietary intake to match increased energy expenditure to avoid exercise-induced weight loss as a potential confounder (29). We also assessed circulating 25(OH)D half-life using stable isotope-labelled 25$(OH)D_3$ and, in adipose tissue, we measured concentrations of 25(OH)D and vitamin D_3 using LC-MS/MS, and the expression of genes associated with vitamin D metabolism. Thus, VitaDEx was a highly controlled trial specifically designed to examine the effects of moderate intensity exercise on systemic vitamin D metabolites in sedentary overweight men and women.

Exercise adherence in VitaDEx was high, significantly increasing daily physical activity, energy expenditure, and maximal oxygen uptake in the exercise *vs* control groups. There was a complete prevention of the winter decline in the active vitamin D metabolite 1, 25$(OH)_2D_3$ in the exercise group compared to controls, with an ameliorated decline in circulating 25(OH)D (Figures 1C, D). Both groups displayed pronounced reductions in subcutaneous adipose tissue vitamin D metabolite concentrations over winter with no between group differences. Exercise also impacted relative concentrations of vitamin D metabolites in the circulation and adipose tissue, and how these changed over the winter. These novel data indicate that exercise has a direct causal effect on vitamin D metabolism and basal vitamin D concentrations (and status) during winter months.

Physical activity – two for the price of one

The combined acute and long-term effects of exercise of vitamin D may 'double up' the overall net accumulated physiological changes and associated health benefit (Figure 2). Our recent trials indicate that there are both short-term transient effects in and around each exercise bout – as well as longer term effects on basal

levels. The short and long-term effects on biologically active 1, 25 $(OH)_2D_3$ are particularly striking. Whether the mechanisms involved in shorter- and longer-term changes are similar or separate is not yet clear, but it is possible that the biological consequences and 'benefits' could be distinct. Exercise could have a unique role to play as an adjunct to vitamin D supplementation which does not appear to have similar effects on 1, $25(OH)_2D_3$ concentrations in either the short or longer term (30, 31).

Exercise has well-documented effects on many of the physiological systems also known to be affected by vitamin D (e.g., bone, skeletal muscle, immune system etc.). Thus, short- and longer-term changes to vitamin D with regular exercise may play a role in facilitating and supporting short- and longer-term biological adaptations associated with higher levels of physical activity.

Potential mechanisms underpinning changes to vitamin D status

We previously proposed that one mechanism contributing to improved vitamin D status with regular exercise could involve the mobilisation of vitamin D from adipose tissue (32). Adipose accumulates large amounts of lipophilic vitamin D (mostly vitamin D_3), equivalent to >4 months of daily intake at 10 µg/day in a typical overweight individual not using supplements (32, 33). During most forms of physical activity, increases in the concentrations of hormones such as adrenaline and atrial natriuretic peptide, and decreases in insulin, lead to the hydrolysis of triacylglycerol from the lipid droplet of adipocytes by adipose triglyceride lipase and hormone-sensitive lipase (34). The net effect during light to moderate intensity physical activity is a 2 to 3-fold increase in adipose tissue lipolysis (34). When triacylglycerol is hydrolysed, vitamin D within the lipid droplet may also be released (32, 35). A heightened turnover of adipose triacylglycerol may be sufficient to mobilise vitamin D from adipose but there is also the potential for a reduction in adipose mass which would additionally affect systemic vitamin D concentrations (29).

An alternative and potentially complementary explanation could involve skeletal muscle (36, 37). Vitamin D binding protein (DBP), which transports 25(OH)D in the circulation, has a high-affinity for actin and has long been known to bind to actin within myocytes (38). With DBP having a half-life of 1–4 days, it is proposed that DBP is quickly proteolyzed within muscle releasing 25(OH)D which then binds to intracellular DBP or diffuses from the cell to bind to extracellular DBP (36, 37). This cycling of 25(OH)D from bound to unbound state is thought to be necessary for the comparatively longer half-life of 25(OH)D (at least 15 days) compared to DBP (39), and may help conserve vitamin D status during winter (37). Low physical activity and poor muscle health may disturb the uptake and release of 25(OH)D from muscle cells (36, 37). It is also important to recognise the role of DBP in transporting 25(OH)D to proximal tubule cells in the kidney, where there is abundant expression of the 25-hydroxyvitamin D-1α-hydroxylase enzyme (CYP27B1) that facilitates synthesis of 1, $25(OH)_2D_3$ (40). Proximal tubule acquisition of 25(OH)D from glomerular filtrate requires megalin-mediated endocytic uptake of DBP and its 25(OH)D cargo (41). Thus, mobilisation of muscle DBP may indirectly impact CYP27B1-mediated synthesis of 1, $25(OH)_2D_3$ by increasing the renal availability of 25(OH)D bound to the DBP. With a half-life in serum of only 5–9 hours, concentrations of biologically active 1, $25(OH)_2D_3$ are tightly regulated by parathyroid hormone, calcium, acidosis, insulin, and 1, $25(OH)_2D_3$ itself (42). Increases in 25(OH)D concentrations with exercise may provide more substrate for renal conversion to 1, $25(OH)_2D_3$, and exercise may also directly perturb the various other factors that have the potential to directly influence 1, $25(OH)_2D_3$ concentrations (9). The fact that the acute increase in 1, $25(OH)_2D_3$ concentrations outlasts the increase in 25(OH)D (26) may be indicative of increased activation of the vitamin D endocrine axis and VDR-mediated gene regulation, particularly in pathways related to calcium-phosphate homeostasis, bone metabolism, and immune regulation.

Thus, there are a variety of biologically plausible mechanisms through which physical activity could influence vitamin D metabolism and downstream physiological processes.

Implications and future directions

Future research should examine the temporality and dose-response following single bouts of exercise. Much larger increases in 25(OH)D have been reported after more demanding forms of

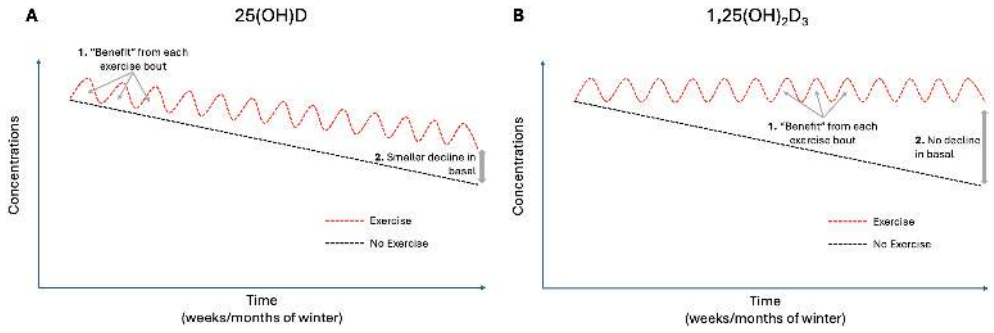

FIGURE 2
A conceptual depiction of the dual effect of exercise on systemic vitamin D metabolites. Exercise causes both acute changes in and around each exercise bout, as well as ameliorating or preserving the normal seasonal decline in basal levels during winter. Panel **(A)** denotes 25(OH)D and Panel **(B)** depicts 1, $25(OH)_2D_3$.

acute exercise (e.g., ultra-marathons), and these seem to be maintained for up to 24h (24, 43). Whilst this might indicate evidence for a dose-response, the lack of a corresponding control condition limits this interpretation. As far as we are aware, our recent crossover RCT is the first study to demonstrate a statistically significant increase in the biologically active form of vitamin D (1, 25$(OH)_2D_3$). The concentration of 1, 25$(OH)_2D_3$ was increased by ~13 pmol/L an hour after exercise, but the effect could have been much longer (and greater) since no samples were collected beyond 1h post-exercise until the 24h timepoint. Thus, we do not know if the effect of acute moderate intensity exercise on 1, 25$(OH)_2D_3$ lasts one hour or many hours. Indeed, 3 hours after 60 minutes of more intense exercise (70% $V\dot{O}_2$max), athletes had numerically greater increase from baseline in 1, 25$(OH)_2D_3$ (19 pmol/L), albeit this was not statistically significant in this smaller study (44). We also do not know the minimum amount of exercise or physical activity that would stimulate an increase in 25(OH)D or 1, 25$(OH)_2D_3$, and whether the acute effects of exercise are different in different populations (e.g., older people).

The VitaDEx 10-week RCT provides causal evidence for the effect of moderate intensity physical activity on vitamin D metabolism in sedentary overweight men and women in the context of the normal seasonal decline in vitamin D status. Further research is needed to determine if other populations show the same response. More work is also needed to examine different forms of exercise and to elucidate the key mechanisms. Studies to address these mechanisms will need to consider seasonal impacts on vitamin D status. Whilst winter may represent the most important time to intervene from a vitamin D status perspective (when physical activity and vitamin D levels are lowest), it is likely that the interaction between physical activity and vitamin D has important physiological ramifications all year round. Thus, additional work is required to examine the generalisability of these findings across different populations and seasons, as well as to establish the key mechanisms.

Given the effects of physical activity on vitamin D metabolism, future vitamin D research should include robust measures of physical activity. Differences in physical activity could explain previous surprising associations – such as the finding that some people in northern latitudes do not become vitamin D deficient in winter (45). Similarly, differences in physical activity could explain why some groups of people respond more effectively to vitamin D supplementation than others (46). Thus, vitamin D researchers should include robust measures of physical activity when phenotyping participants.

Conclusion

Recent carefully designed randomised controlled trials demonstrate that physical activity has both short- and long-term effects on vitamin D metabolism and status. In many ways, this should not really come as a surprise. Our physiology evolved to 'expect' high levels of physical activity (47, 48). Low physical activity leads to a deficiency in anticipated biological responses and increased risk of non-communicable diseases (47). Thus, the low levels of vitamin D observed today in many populations could be partly a product of low levels of physical activity in these populations (49), especially in winter when physical activity tends to fall still further (50). By extension, interventions designed to increase physical activity may be a complementary strategy to improve individual and population vitamin D status. Thus, the beneficial effects of physical activity on vitamin D metabolism can be added to the already-long list of good reasons to be physically active. Furthermore, policymakers and health agencies can add physical activity to their toolbox of strategies to improve population vitamin D status.

Data availability statement

Publicly available datasets were analyzed in this study. These data can be found here: https://researchportal.bath.ac.uk/en/datasets/dataset-for-the-effect-of-an-acute-bout-of-exercise-on-circulatin https://researchportal.bath.ac.uk/en/datasets/dataset-for-exercise-prevents-seasonal-decline-in-vitamin-d-metab/.

Author contributions

DT: Conceptualization, Writing – review & editing, Funding acquisition, Writing – original draft. OP: Writing – review & editing, Writing – original draft. SD: Writing – original draft, Writing – review & editing. MH: Writing – original draft, Writing – review & editing. KJ: Writing – review & editing, Writing – original draft.

Funding

The author(s) declared that financial support was received for this work and/or its publication. DT, OP, SD, MH and KJ were supported by a grant from the Biotechnology and Biological Sciences Research Council (BBSRC, BB/R018928/1). KJ was supported by the NIHR Cambridge Biomedical Research Centre (NIHR203312). The views expressed are those of the authors and not necessarily those of the NIHR or the Department of Health and Social Care.

Acknowledgments

For the purpose of Open Access, the authors have applied a Creative Commons Attribution (CC BY) licence to any Author Accepted Manuscript version arising.

Conflict of interest

The author(s) declared that this work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Generative AI statement

The author(s) declared that generative AI was not used in the creation of this manuscript.

Any alternative text (alt text) provided alongside figures in this article has been generated by Frontiers with the support of artificial intelligence and reasonable efforts have been made to ensure accuracy, including review by the authors wherever possible. If you identify any issues, please contact us.

Publisher's note

All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.

References

1. Bikle DD. Extraskeletal actions of vitamin D. *Ann N Y Acad Sci*. (2016) 1376:29–52. doi: 10.1111/nyas.13219

2. Wacker M, Holick MF. Sunlight and vitamin D. *Dermato-Endocrinology*. (2013) 5:51–108. doi: 10.4161/derm.24494

3. Lin LY, Smeeth L, Langan S, Warren-Gash C. Distribution of vitamin D status in the UK: a cross-sectional analysis of UK Biobank. *BMJ Open*. (2021) 11:e038503. doi: 10.1136/bmjopen-2020-038503

4. SACN. *Vitamin D and Health*. Public Health England, Crown Copyright (2016).

5. US Department of Health and Human Services. Vitamin D (2025). Available online at: https://ods.od.nih.gov/factsheets/VitaminD-HealthProfessional/ (Accessed June 23, 2026).

6. Caspersen CJ, Powell KE, Christenson GM. Physical-activity, exercise, and physical-fitness - definitions and distinctions for health-related research. *Public Health Rep*. (1985) 100:126–31.

7. Thompson D, Peacock O, Western M, Batterham AM. Multidimensional physical activity: an opportunity, not a problem. *Exercise Sport Sci Rev*. (2015) 43:67–74. doi: 10.1249/JES.0000000000000039

8. Turner JE, Markovitch D, Betts JA, Thompson D. Nonprescribed physical activity energy expenditure is maintained with structured exercise and implicates a compensatory increase in energy intake. *Am J Clin Nutr*. (2010) 92:1009–16. doi: 10.3945/ajcn.2010.29471

9. Thompson D, Perkin OJ. Physiology of physical activity and sedentary behaviour. In: Armstrong MEG, editor. *The Routledge Handbook on Physical Activity and Health*. Routledge, New York, US (2027).

10. Wang Z, Cao Z, Min J, Duan T, Xu C. Associations between device-measured and self-reported physical activity and common mental disorders: findings from a large-scale prospective cohort study. *BMJ Evid Based Med*. (2025) 30:45–54. doi: 10.1136/bmjebm-2024-112933

11. Chomistek AK, Chiuve SE, Jensen MK, Cook NR, Rimm EB. Vigorous physical activity, mediating biomarkers, and risk of myocardial infarction. *Med Sci Sports Exerc*. (2011) 43:1884–90. doi: 10.1249/mss.0b013e31821b4d0a

12. Gerdhem P, Ringsberg KA, Obrant KJ, Akesson K. Association between 25-hydroxy vitamin D levels, physical activity, muscle strength and fractures in the prospective population-based OPRA study of elderly women. *Osteoporos Int*. (2005) 16:1425–31. doi: 10.1007/s00198-005-1860-1

13. Scott D, Blizzard L, Fell J, Ding C, Winzenberg T, Jones G, et al. A prospective study of the associations between 25-hydroxy-vitamin D, sarcopenia progression and physical activity in older adults. *Clin Endocrinol (Oxf)*. (2010) 73:581–7. doi: 10.1111/j.1365-2265.2010.03858.x

14. van den Heuvel EG, van Schoor N, de Jongh RT, Visser M, Lips P. Cross-sectional study on different characteristics of physical activity as determinants of vitamin D status; inadequate in half of the population. *Eur J Clin Nutr*. (2013) 67:360–5. doi: 10.1038/ejcn.2013.22

15. Kim J, Park J, So W-Y. Association between blood vitamin D levels and regular physical activity in Korean adolescents. *Healthcare*. (2022) 10:1277. doi: 10.3390/healthcare10071277

16. Og G, Chang M, Jonsson Pv Thorsdottir I, Ramel A. Associations of physical activity with vitamin D status depends on obesity status in old adults. *Clin Nutr ESPEN*. (2020) 39:222–6. doi: 10.1016/j.clnesp.2020.06.009

17. Miettinen ME, Kinnunen L, Leiviskä J, Keinänen-Kiukaanniemi S, Korpi-Hyövälti E, et al. Association of serum 25-hydroxyvitamin D with lifestyle factors and metabolic and cardiovascular disease markers: population-based cross-sectional study (FIN-D2D). *PloS One*. (2014) 9:e100235. doi: 10.1371/journal.pone.0100235

18. Larose TL, Chen Y, Camargo CA Jr, Langhammer A, Romundstad P, Mai XM. Factors associated with vitamin D deficiency in a Norwegian population: the HUNT study. *J Epidemiol Community Health*. (2014) 68:165–70. doi: 10.1136/jech-2013-202587

19. Kluczynski MA, Lamonte MJ, Mares JA, Wactawski-Wende J, Smith AW, Engelman CD, et al. Duration of physical activity and serum 25-hydroxyvitamin D status of postmenopausal women. *Ann Epidemiol*. (2011) 21:440–9. doi: 10.1016/j.annepidem.2010.11.011

20. Klenk J, Rapp K, Denkinger M, Nagel G, Nikolaus T, Peter R, et al. Objectively measured physical activity and vitamin D status in older people from Germany. *J Epidemiol Community Health*. (2015) 69:388–92. doi: 10.1136/jech-2014-204632

21. Booth F, Chakravarthy MV, Spangenburg EE. Exercise and gene expression: physiological regulation of the human genome through physical activity. *J Physiol*. (2002) 543.2:399–411. doi: 10.1113/jphysiol.2002.019265

22. Magkos F, Mohammed BS, Patterson BW, Mittendorfer B. Free fatty acid kinetics in the late phase of postexercise recovery: importance of resting fatty acid metabolism and exercise-induced energy deficit. *Metab Clin Exp*. (2009) 58:1248–55. doi: 10.1016/j.metabol.2009.03.023

23. Sun X, Cao ZB, Taniguchi H, Tanisawa K, Higuchi M. Effect of an acute bout of endurance exercise on serum 25(OH)D concentrations in young adults. *J Clin Endocrinol Metab*. (2017) 102:3937–44. doi: 10.1210/jc.2017-00146

24. O'Leary TJ, Coombs CV, Edwards VC, Blacker SD, Knight RL, Koivula FN, et al. The effect of sex and protein supplementation on bone metabolism during a 36-h military field exercise in energy deficit. *J Appl Physiol (1985)*. (2023) 134:1481–95. doi: 10.1152/japplphysiol.00106.2023

25. Żychowska M, Rola R, Borkowska A, Tomczyk M, Kortas J, Anczykowska K, et al. Fasting and exercise induce changes in serum vitamin D metabolites in healthy men. *Nutrients*. (2021) 13. doi: 10.3390/nu13061963

26. Davies SE, Perkin OJ, Betts JA, Gonzalez JT, Hewison M, Jenkinson C, et al. The effect of an acute bout of exercise on circulating vitamin D metabolite concentrations: a randomised crossover study in healthy adults. *J Physiol*. (2024) 602:4157–70. doi: 10.1113/jp286395

27. Perkin OJ, Davies SE, Hewison M, Jones KS, Gonzalez JT, Betts JA, et al. Exercise without weight loss prevents seasonal decline in vitamin D metabolites: the VitaDEx randomized controlled trial. *Adv Sci*. (2025) 12:2416312. doi: 10.1002/advs.202416312

28. Khan SR, Claeson M, Khan A, Neale RE. The effect of physical activity on vitamin D: a systematic review and meta-analysis of intervention studies in humans. *Public Health Pract (Oxf)*. (2024) 7:100495. doi: 10.1016/j.puhip.2024.100495

29. Gangloff A, Bergeron J, Lemieux I, Despres JP. Changes in circulating vitamin D levels with loss of adipose tissue. *Curr Opin Clin Nutr Metab Care*. (2016) 19(6):464–70. doi: 10.1097/mco.0000000000000315

30. Gazerani P, Clarke N, Reitz RE, Travison TG, Holick MF. A randomized, double-blinded, placebo-controlled, parallel trial of vitamin D(3) supplementation in adult patients with migraine. *Curr Med Res Opin*. (2019) 35:715–23. doi: 10.1080/03007995.2018.1519503

31. Biancuzzo RM, Perkin O, Gonzalez JT, Betts JA, Hewison M, Manolopoulos KN, et al. Serum concentrations of 1,25-dihydroxyvitamin D2 and 1,25-dihydroxyvitamin D3 in response to vitamin D2 and vitamin D3 supplementation. *J Clin Endocrinol Metab*. (2013) 98:973–9. doi: 10.1210/jc.2012-2114

32. Hengist A, Burild A, Jakobsen J, Fuskevåg OM, Jorde R. Mobilising vitamin D from adipose tissue: the potential impact of exercise. *Nutr Bull*. (2019) 44:25–35. doi: 10.1111/nbu.12369

33. Didriksen A, Karpe F, Lafontan M, Frayn KN. Vitamin D3 increases in abdominal subcutaneous fat tissue after supplementation with vitamin D3. *Eur J Endocrinol*. (2015) 172:235–41. doi: 10.1530/eje-14-0870

34. Thompson D, Abboud M, Puglisi DA, Gordon-Thomson C, Brennan-Speranza TC, Mason RS, et al. Physical activity and exercise in the regulation of human adipose tissue physiology. *Physiol Rev*. (2012) 92:157–91. doi: 10.1152/physrev.00012.2011

35. Di Nisio A, et al. Impaired release of vitamin D in dysfunctional adipose tissue: new cues on vitamin D supplementation in obesity. *J Clin Endocrinol Metab*. (2017) 102 (7):2564–74. doi: 10.1210/jc.2016-3591

36. Rybchyn MS, Abboud M, Puglisi DA, Gordon-Thomson C, Brennan-Speranza TC, Mason RS, et al. Skeletal muscle and the maintenance of vitamin D status. *Nutrients*. (2020) 12. doi: 10.3390/nu12113270

37. Mason RS, Puglisi DA, Davies BN, Rybchyn M, Whitehead NP, Brock KE, et al. The role of skeletal muscle in maintaining vitamin D status in winter. *Curr Dev Nutr*. (2019) 3:nzz087. doi: 10.1093/cdn/nzz087

38. Haddad JG. Human serum binding protein for vitamin D and its metabolites (DBP): evidence that actin is the DBP binding component in human skeletal muscle. *Arch Biochem Biophys*. (1982) 213:538–44. doi: 10.1016/0003-9861(82)90581-1

39. Abboud M, Puglisi DA, Davies BN, Rybchyn M, Whitehead NP, Brock KE, et al. Evidence for a specific uptake and retention mechanism for 25-hydroxyvitamin D (25OHD) in skeletal muscle cells. *Endocrinology*. (2013) 154:3022–30. doi: 10.1210/en.2012-2245

40. Zehnder D, Bland R, Walker EA, Bradwell AR, Howie AJ, Hewison M, et al. Expression of 25-hydroxyvitamin D3-1alpha-hydroxylase in the human kidney. *J Am Soc Nephrol*. (1999) 10:2465–73. doi: 10.1681/asn.v10122465

41. Nykjaer A, Dragun D, Walther D, Vorum H, Jacobsen C, Herz J, et al. An endocytic pathway essential for renal uptake and activation of the steroid 25-(OH) vitamin D3. *Cell*. (1999) 96:507–15. doi: 10.1016/s0092-8674(00)80655-8

42. Brown AJ, Dusso A, Slatopolsky E. Vitamin D. *Am J Physiol Renal Physiol*. (1999) 277:F157–75. doi: 10.1093/ndt/14.1.11

43. Mieszkowski J, Stankiewicz B, Kochanowicz A, Niespodziński B, Kowalik T, Żmijewski MA, et al. Ultra-marathon-induced increase in serum levels of vitamin D metabolites: a double-blind randomized controlled trial. *Nutrients*. (2020) 12:3629. doi: 10.3390/nu12123629

44. Zittermann A, Sabatschus O, Jantzen S, Platen P, Danz A, Stehle P. Evidence for an acute rise of intestinal calcium absorption in response to aerobic exercise. *Eur J Nutr*. (2002) 41:189–96. doi: 10.1007/s00394-002-0375-1

45. Ramnemark A, Norberg M, Pettersson-Kymmer U, Eliasson M. Adequate vitamin D levels in a Swedish population living above latitude 63°N: the 2009 Northern Sweden MONICA study. *Int J Circumpolar Health*. (2015) 74:27963. doi: 10.3402/ijch.v74.27963

46. Tobias DK, Luttmann-Gibson H, Mora S, Danik J, Bubes V, Copeland T, et al. Association of body weight with response to vitamin D supplementation and metabolism. *JAMA Netw Open*. (2023) 6:e2250681–e2250681. doi: 10.1001/jamanetworkopen.2022.50681

47. Booth FW, Roberts CK, Thyfault JP, Ruegsegger GN, Toedebusch RG, et al. Role of inactivity in chronic diseases: evolutionary insight and pathophysiological mechanisms. *Physiol Rev*. (2017) 97:1351–402. doi: 10.1152/physrev.00019.2016

48. Eaton SB. An evolutionary perspective on human physical activity: implications for health. *Comp Biochem Physiol a-Molecular Integr Physiol*. (2003) 136:153–9. doi: 10.1016/s1095-6433(03)00208-3

49. Booth FW, Hawley JA. The erosion of physical activity in Western societies: an economic death march. *Diabetologia*. (2015) 58(8):1730–4. doi: 10.1007/s00125-015-3617-5

50. Patten T, Preble EA, Master H, Adjemian J, Ramirez A, McClain J, et al. The All of Us Research Program's wearables dataset. *Nat Med*. (2026) 32:2302–10. doi: 10.1038/s41591-026-04352-3