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Vitamin D Deficiency Among Young Adults: Causes and Consequences

A Research Paper

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Abstract

Vitamin D deficiency has emerged as a significant, and frequently overlooked, nutritional concern among young adults aged eighteen to twenty-nine, a population commonly assumed to be at low risk of micronutrient insufficiency. This paper examines the physiological, behavioural, and environmental determinants that predispose young adults to suboptimal vitamin D status, together with the skeletal and extra-skeletal consequences that follow from prolonged deficiency. Drawing on epidemiological evidence from Australia, the Gulf region, South Asia, and the United States, the paper situates the problem within a broader pattern of lifestyle change: indoor-oriented education and early-career work, sun-protective practices, dietary patterns low in fortified foods, and rising rates of adiposity. Using a qualitative, literature-based methodology, the paper critically analyses the interaction between these causes and outcomes such as impaired bone mineralisation, immune dysregulation, mood disturbance, and metabolic risk. The analysis suggests that deficiency in young adulthood is not a transient inconvenience but a determinant of lifelong health trajectories, particularly for the accrual of peak bone mass. The paper concludes with recommendations for targeted screening, food fortification policy, and institutional and behavioural interventions suited to university and early-career populations, and calls for longitudinal research specific to this comparatively understudied age group.

Keywords

vitamin D deficiency; young adults; 25-hydroxyvitamin D; bone health; sun exposure; nutrition; public health; hypovitaminosis D

1. Introductoin

Vitamin D occupies an unusual position among micronutrients: unlike most vitamins, the majority of the body's supply is not obtained through diet but manufactured within the skin following exposure to ultraviolet B (UVB) radiation, then activated through hydroxylation in the liver and kidneys into calcitriol, its biologically active hormone form.[1]

This dual identity, part nutrient and part endocrine product, makes vitamin D status unusually sensitive to behaviour, geography, and season rather than to diet alone. For most of human history, outdoor labour and unshielded skin ensured adequate cutaneous synthesis; industrialisation, urban living, and, more recently, digitally organised academic and professional lifestyles have steadily narrowed that natural pathway.

Young adults, understood here as individuals between roughly eighteen and twenty-nine years of age, might appear the demographic least likely to experience nutritional deficiency. They are typically free of the mobility limitations of old age, the growth-related demands of childhood, and many of the chronic conditions that complicate nutrient absorption later in life. Yet a growing body of survey evidence contradicts this assumption. National data from Australia found that thirty-two percent of young adults aged eighteen to twenty-four were vitamin D deficient, nearly double the rate recorded among adolescents surveyed in the same study.[2]

[1]National Institutes of Health, Office of Dietary Supplements, "Vitamin D: Fact Sheet for Health Professionals," updated June 2025, https://ods.od.nih.gov/factsheets/VitaminD-HealthProfessional/.

[2]Kahlea Horton-French et al., "Prevalence and Predictors of Vitamin D Deficiency in a Nationally Representative Sample of Australian Adolescents and Young Adults," European Journal of Clinical Nutrition 75, no. 11 (2021): 1627–1636.

Even higher figures have been reported in Gulf and South Asian settings, where deficiency prevalence among university-aged cohorts has exceeded eighty percent in some samples.[3]

Regional pooled estimates for South Asia place adult deficiency prevalence at roughly two-thirds of the population studied.[4]

These figures suggest that vitamin D deficiency in young adulthood is neither a regional anomaly nor a statistical artefact, but a pattern that recurs across latitudes, climates, and cultures.

This paper undertakes a literature-based examination of why this pattern has emerged and what it means for the young adults affected. It treats the causes of deficiency not as a simple checklist of isolated risk factors but as an interacting system of behavioural, physiological, and structural forces: the routines of study and work, the aesthetics of sun avoidance, the availability of fortified food, and the biology of body composition. It treats the consequences, in turn, not merely as a matter of bone-density statistics but as a question of long-term health trajectory, since the years of young adulthood are also the years in which peak bone mass is consolidated and lifelong habits are formed. The paper moves from background and problem definition, through a review of existing literature, to a critical analysis connecting causal mechanisms to downstream outcomes, before closing with recommendations directed at clinicians, universities, and policymakers.

2. Background of the Study

Interest in vitamin D expanded considerably following the publication of Holick's influential 2006 review, which argued that vitamin D inadequacy constituted a substantially under-recognised

[3]Mohammed A. Jamali, Suad M. Abdeen, and Thazhumpal C. Mathew, "Prevalence of Vitamin D Deficiency among Healthy Young Adults at Kuwait University," Cureus 16, no. 12 (2024): e75911.

[4]Mahbubul H. Siddiqee et al., "High Prevalence of Vitamin D Deficiency among the South Asian Adults: A Systematic Review and Meta-Analysis," BMC Public Health 21 (2021): 1823.

epidemic affecting healthy populations across the age spectrum, not merely institutionalised or elderly patients.[5]

In the years since, national health surveys in several countries have incorporated serum 25-hydroxyvitamin D measurement as a routine biomarker, allowing researchers to move from isolated clinical case reports toward population-representative estimates.

What these surveys reveal is a status pattern specific to young adulthood. Adolescents, who are more likely to be involved in organised outdoor sport and school-based physical activity, and older adults, who are more frequently the target of clinical screening and supplementation advice because of osteoporosis risk, appear in some datasets to fare comparatively better than the age group sandwiched between them.[6]

Young adults occupy a transitional period in which the structured outdoor time of childhood has ended, but the medical surveillance associated with ageing has not yet begun. They are also the demographic most likely to be enrolled in higher education or entering demanding early-career employment, both of which are associated with long indoor hours, whether in lecture theatres, libraries, laboratories, or open-plan offices.

Global estimates converge on a shared conclusion, even as absolute figures vary by assay method and cut-off definition: a substantial share of otherwise healthy young adults carry insufficient circulating vitamin D. In a well-known United States cohort, close to two-thirds of healthy young adults were found to be vitamin D insufficient by the end of winter.[7]

[5]Michael F. Holick, "High Prevalence of Vitamin D Inadequacy and Implications for Health," Mayo Clinic Proceedings 81, no. 3 (2006): 353–373.

[6]Horton-French et al., "Prevalence and Predictors," 1630–1631.

[7]Medscape, "Vitamin D Deficiency and Related Disorders: Practice Essentials, Background, Pathophysiology," accessed August 2026, https://emedicine.medscape.com/article/128762-overview.

Regional analyses from South Asia have pooled prevalence estimates as high as sixty-eight percent among adults, with marked variation between Pakistan, Bangladesh, India, Nepal, and Sri Lanka.[8]

A single-institution study of Kuwaiti university students recorded deficiency in more than eighty-five percent of participants.[9]

These figures should not be read as directly comparable, since the underlying studies differ in assay methodology, deficiency thresholds, and season of sampling; nevertheless, the consistency of a high-prevalence finding across such geographically and culturally distinct settings supports the view that something structural, rather than merely local, is driving the pattern.

This paper is situated within that body of evidence. It does not report new primary data; rather, it synthesises existing findings into an integrated account of causes and consequences specific to the young-adult life stage, an age group that has historically received less dedicated research attention than either children or the elderly.

3. Statement of the Problem

Despite an expanding body of prevalence data, vitamin D deficiency in young adults remains comparatively under-addressed in both clinical practice and public health policy. Screening guidelines in many health systems restrict routine testing to recognised high-risk groups, such as pregnant women, older adults, or individuals with diagnosed bone disease, leaving apparently healthy young adults outside the scope of proactive assessment.[10]

[8]Siddiqee et al., "High Prevalence of Vitamin D Deficiency," 4–6.
[9]Jamali, Abdeen, and Mathew, "Prevalence of Vitamin D Deficiency," e75911.
[10]National Institutes of Health, Office of Dietary Supplements, "Vitamin D: Fact Sheet for Health Professionals."

The result is a population in which deficiency frequently goes undiagnosed until symptoms such as fatigue, musculoskeletal pain, or, in more severe cases, stress fractures prompt investigation.

The problem is compounded by the fact that the causes of deficiency in this age group are not isolated behaviours but interlocking features of ordinary young-adult life: extended indoor study and screen time, sun-protective skincare practices, urban residence, dietary patterns that rarely include the small number of naturally vitamin D-rich foods, and, for a subset of the population, higher body-fat percentage, which is independently associated with lower circulating 25-hydroxyvitamin D.[11]

Because these causes are embedded in ordinary routine rather than in unusual circumstance, they are easy to overlook as risk factors and difficult to address through advice alone.

At the same time, this life stage carries particular biological stakes. Peak bone mass, the maximum bone density an individual will ever attain, is largely consolidated by the late twenties; deficiency during this window may therefore have consequences that persist for decades, well beyond the period of deficiency itself. The problem this paper addresses, then, is the mismatch between the scale and structural nature of the causes on one hand, and the low visibility of the condition in clinical and institutional practice on the other.

4. Research Questions

1. What behavioural, environmental, and physiological factors most consistently contribute to vitamin D deficiency among young adults?

[11] Horton-French et al., "Prevalence and Predictors," 1633.

2. What skeletal and extra-skeletal health consequences are associated with vitamin D deficiency during young adulthood, and how do these differ from consequences documented in children or older adults?

3. What screening, dietary, and policy interventions are best suited to the specific circumstances of young-adult populations, such as university students and early-career workers?

5. Research Objectives

• To synthesise existing epidemiological evidence on the prevalence of vitamin D deficiency among young adults across diverse geographic and cultural settings.

• To critically examine the causal pathways, behavioural, dietary, and physiological, through which modern young-adult lifestyles reduce vitamin D status.

• To evaluate the documented health consequences of deficiency in this age group, distinguishing well-established outcomes, such as bone health, from associations that remain under active investigation, such as depression.

• To propose feasible, evidence-informed recommendations for screening, dietary policy, and institutional practice appropriate to young-adult settings such as universities and workplaces.

6. Significance of the Study

This paper contributes to the literature in three respects. First, it consolidates evidence that is currently scattered across country-specific and often clinically narrow studies into a single, age-group-focused synthesis, making the scale of the issue easier to appreciate than it is in any single national report. Second, by treating causes and consequences as interacting rather than as separate lists, the paper offers a more integrated account of why deficiency in young adulthood matters, not

simply as a laboratory abnormality but as a determinant of trajectories in bone health, immune function, and possibly mental health, that extend well beyond the years in which the deficiency itself occurs. Third, the paper is oriented toward practical application: universities, employers, and primary care providers who interact with young adults are offered recommendations calibrated to the realities of this life stage, rather than generic advice designed for the population as a whole. For students of public health and nutrition, the paper also illustrates how a nutrient once associated primarily with childhood rickets and elderly osteoporosis has become a relevant concern across the full life course, reflecting broader shifts in how modern populations spend their daylight hours.

7. Literature Review

7.1 Physiological Basis of Vitamin D Status

Vitamin D3 (cholecalciferol) is synthesised in the epidermis when 7-dehydrocholesterol reacts with UVB radiation of wavelengths of approximately 290 to 315 nanometres; the resulting previtamin D3 undergoes thermal isomerisation before being transported to the liver, where it is hydroxylated to 25-hydroxyvitamin D, the primary circulating form used to assess status, and subsequently to the kidney, where it is converted to the active hormone 1,25-dihydroxyvitamin D.[12]

Because synthesis depends on UVB availability, which itself depends on latitude, season, time of day, cloud cover, and skin pigmentation, vitamin D status is inherently more variable across populations than that of nutrients obtained solely through diet.[13]

[12]National Institutes of Health, Office of Dietary Supplements, "Vitamin D: Fact Sheet for Consumers," https://ods.od.nih.gov/factsheets/VitaminD-Consumer/.

[13]Holick, "High Prevalence of Vitamin D Inadequacy," 355–357.

Dietary sources, including oily fish, egg yolks, fortified milk and cereals, and UV-exposed mushrooms, supply a comparatively modest share of most people's vitamin D and were never intended, within dietary reference frameworks, to fully substitute for cutaneous synthesis.[14]

7.2 Prevalence Evidence Specific to Young Adults

Multiple national and institutional surveys converge on the finding that young adults are not protected from deficiency by their relative youth and general health. In Australia, deficiency prevalence among eighteen-to-twenty-four-year-olds was found to be roughly double that observed in adolescents from the same nationally representative survey, and was independently associated with season, region of birth, and physical activity level.[15]

In the Gulf region, more than eighty-five percent of a sample of medical and dental students at Kuwait University were found to be vitamin D deficient, with academic year, a proxy for indoor clinical versus outdoor pre-clinical study patterns, significantly associated with status.[16]

Pooled estimates for South Asian adults place regional deficiency prevalence at roughly two-thirds, with the highest rates recorded in Pakistan and the lowest, though still substantial, in Sri Lanka.[17]

United States data drawing on national survey cohorts similarly document a considerable combined prevalence of deficiency and insufficiency among adults, with severe deficiency concentrated in specific demographic subgroups.[18]

[14]National Institutes of Health, Office of Dietary Supplements, "Vitamin D: Fact Sheet for Health Professionals."
[15]Horton-French et al., "Prevalence and Predictors," 1629–1630.
[16]Jamali, Abdeen, and Mathew, "Prevalence of Vitamin D Deficiency."
[17]Siddiqee et al., "High Prevalence of Vitamin D Deficiency," 2.
[18]Ting-Yi Wang, Hung-Wei Wang, and Ming-Yan Jiang, "Prevalence of Vitamin D Deficiency and Associated Risk of All-Cause and Cause-Specific Mortality among Middle-Aged and Older Adults in the United States," Frontiers in Nutrition 10 (2023): 1163737.

Although these studies differ in the serum cut-offs used to define deficiency and in assay technique, which limits precise cross-study comparison, the consistent pattern of high prevalence among ostensibly healthy young cohorts, across markedly different latitudes and cultural contexts, is difficult to attribute to any single local factor.[19]

7.3 Behavioural and Lifestyle Determinants

The literature identifies several recurring correlates of low vitamin D status in this age group: low levels of outdoor physical activity, urban residence, use of sun-protective clothing or sunscreen, higher body mass index, and, in migrant populations, movement from regions of higher UVB availability to regions of lower availability.[20]

Body composition appears to interact with vitamin D status through more than one pathway. Because vitamin D is fat-soluble, it is sequestered within adipose tissue, and larger fat mass appears to dilute circulating concentrations even when total-body vitamin D content is not necessarily reduced, alongside possible reductions in outdoor activity among individuals with higher body fat.[21]

7.4 Consequences Literature

The skeletal consequences of vitamin D deficiency are the most firmly established in the literature, given the nutrient's role in intestinal calcium absorption and bone mineralisation; sustained deficiency is linked to reduced bone mineral density and, in severe or prolonged cases, osteomalacia.[22]

[19] Medscape, "Vitamin D Deficiency and Related Disorders."
[20] Horton-French et al., "Prevalence and Predictors," 1632–1633.
[21] Horton-French et al., "Prevalence and Predictors," 1633.
[22] Holick, "High Prevalence of Vitamin D Inadequacy," 358–360.

Evidence for extra-skeletal consequences is more heterogeneous. Several studies report associations between low vitamin D status and depressive symptoms among adolescents and young adults, including a cohort study that found low vitamin D levels associated with depressive symptoms specifically in young adult males.[23]

The direction of causation, however, remains unresolved: research involving adults recovering from traumatic brain injury cautions that conditions which reduce outdoor activity could plausibly lower vitamin D status rather than the reverse, since the injury itself, rather than vitamin D deficiency, may better explain both reduced sunlight exposure and depressive symptoms observed in that population.[24]

Cohort data linking vitamin D deficiency to increased all-cause mortality risk exist primarily for middle-aged and older adults, and should not be extrapolated uncritically to a younger population with a markedly different baseline risk profile.[25]

Taken together, the literature therefore supports a two-tier reading of consequences: strong, mechanistically well-understood skeletal effects, and weaker, associational but biologically plausible extra-skeletal effects that merit further age-specific research.

8. Research Methodology

This paper adopts a qualitative, literature-based (desk) research design rather than primary data collection. It is best characterised as a narrative-analytical review: sources were purposively selected to represent geographically diverse epidemiological studies of vitamin D status in young-

[23]Lucinda J. Black et al., "Low Vitamin D Levels Are Associated with Symptoms of Depression in Young Adult Males," Australian & New Zealand Journal of Psychiatry 48, no. 5 (2014): 464–471.
[24]Omer A. Jamal et al., "Prevalence and Correlates of Vitamin D Deficiency in Adults after Traumatic Brain Injury," Clinical Endocrinology 85, no. 4 (2016): 636–644.
[25]Wang, Wang, and Jiang, "Prevalence of Vitamin D Deficiency and Associated Risk of Mortality."

adult populations, official nutrition-reference documents, and studies addressing the physiological mechanisms and health consequences of deficiency. Selection prioritised peer-reviewed journal articles and data from recognised national health authorities over informal or unreviewed sources, in order to maintain a reasonable standard of evidentiary reliability given the absence of primary data collection in this paper.

Because the paper synthesises existing findings rather than generating new measurements, its methodology is interpretive rather than statistical: reported prevalence figures, associations, and mechanisms drawn from the reviewed literature are compared, contextualised, and, where relevant, critically qualified, for instance by noting differences in deficiency thresholds between studies, or by distinguishing correlational from causal claims. This approach is appropriate to the paper's aim, which is not to establish new empirical facts about vitamin D status but to construct an integrated account of causes and consequences specific to the young-adult life stage from evidence that currently exists in a fragmented, country-specific form.

The principal limitation of this methodology is that it inherits the limitations of its source material: cross-sectional study designs that cannot themselves establish causation, variation in serum 25-hydroxyvitamin D assay methods and deficiency cut-offs across studies, and the concentration of much available research in specific countries and institutional settings, notably university student cohorts, which may not generalise to young adults outside education, such as those in manual or outdoor occupations, whose vitamin D status may differ considerably. Where such limitations bear directly on the interpretation of a claim, they are flagged in the critical analysis below rather than treated as a single uniform caveat applied once and then set aside.

9. Critical Analysis

Rather than treating causes and consequences of vitamin D deficiency as two separate inventories, this analysis argues that they are better understood as a feedback structure specific to the young-

adult life stage, one in which the causes are not incidental but built into the ordinary architecture of study, work, appearance, and body composition that characterises this age group, and in which the consequences, in turn, tend to reinforce or compound the original causes.

Consider first the relationship between indoor lifestyle and academic structure. University education and many entry-level professional roles concentrate waking hours indoors, under artificial light that does not emit UVB. This is not a lapse in individual judgement so much as a structural feature of how education and early careers are organised; a medical or engineering student attending lectures and laboratories for much of the day has comparatively little discretionary daylight left, particularly at higher latitudes during winter months or in climates where daytime heat discourages outdoor activity. The finding that Kuwaiti students in clinical academic years, spent largely indoors in hospital settings, showed different vitamin D patterns from those in earlier, less clinically intensive years is instructive here: it suggests that the causal factor is less "being a student" in the abstract than the specific configuration of indoor time that a given stage of study or career imposes.[26]

This reframes deficiency not as a matter of insufficient health literacy, since these are typically well-educated cohorts, but as a structural by-product of how time is organised during this life stage.

A second thread concerns body composition, and here the analysis must resist a simple linear story. It would be easy to characterise the association between higher body-fat percentage and lower vitamin D status as evidence that deficiency and adiposity share a common behavioural root, namely reduced outdoor activity, but the literature suggests a more layered mechanism, involving fat-soluble sequestration of vitamin D within a larger adipose compartment even where total synthesis or intake has not changed.[27]

[26] Jamali, Abdeen, and Mathew, "Prevalence of Vitamin D Deficiency."
[27] Horton-French et al., "Prevalence and Predictors," 1633.

This matters analytically because it implies that dietary or supplement-based correction may need to account for body composition rather than applying a uniform dose across all young adults, and it cautions against treating higher body fat purely as a downstream consequence of the same sun-avoidant behaviour that causes deficiency, when it may also independently dilute vitamin D concentrations through a separate physiological pathway.

A third and more contested thread is the relationship between vitamin D status and mood. The pattern of association between low vitamin D and depressive symptoms recurs across several studies focused on young people, which is notable given that this age range coincides with peak onset for many mood disorders.[28]

Yet this analysis treats the relationship cautiously rather than as an established causal chain, for a reason the literature itself identifies: depression, or the condition underlying it, can plausibly reduce time spent outdoors, appetite, and general activity, each of which would independently lower vitamin D status, meaning the association could run in either direction, or in both directions simultaneously, forming a self-reinforcing cycle rather than a one-way cause.[29]

This distinction is not merely academic. If deficiency were confirmed as a meaningful causal contributor to mood disturbance in a subset of young adults, then supplementation would carry a plausible role in mental health support for this age group; if the relationship is predominantly reverse-causal, then supplementation alone would be unlikely to resolve the underlying mood disturbance, though correcting deficiency would still carry independent skeletal benefit. Precision on this point matters for how clinicians and universities should prioritise resources, and the current evidence base does not yet permit that precision.

[28] Black et al., "Low Vitamin D Levels Are Associated with Symptoms of Depression."
[29] Jamall et al., "Prevalence and Correlates of Vitamin D Deficiency," 641.

The skeletal consequences, by contrast, rest on firmer mechanistic ground, and this is where the young-adult life stage carries disproportionate long-term stakes. Peak bone mass, the ceiling of bone density an individual will reach, is substantially determined by the third decade of life, after which bone density more typically plateaus and then gradually declines with age. A young adult who experiences a period of deficiency during this consolidation window is not simply at temporary risk; they may be setting a lower ceiling for bone density that persists for the rest of their life, altering the trajectory toward osteoporosis in later decades regardless of whether their vitamin D status is subsequently corrected. This is arguably the most consequential, and least appreciated, feature of vitamin D deficiency in young adulthood: because the immediate skeletal effects are usually subclinical at this age, since young bones rarely fracture from moderate deficiency alone, the condition can proceed unnoticed even as it forecloses long-term bone-health options that cannot later be fully recovered through correction alone.

Taken together, this analysis suggests that vitamin D deficiency in young adults should not be read primarily as a dietary oversight correctable through advice, but as an emergent property of how this life stage is structured, its indoor institutions, its aesthetic and dermatological norms around sun exposure, and its body-composition trends, interacting with a biological window, peak bone mass accrual, that makes the timing of deficiency, and not only its severity, clinically significant.

10. Suggestions and Recommendations

Given that the causes identified above are structural rather than purely individual, recommendations are most likely to be effective when they target institutions and systems that shape young-adult routines, rather than relying solely on individual behaviour change.

10.1 For Universities and Employers

Institutions that concentrate young adults indoors for extended periods, such as universities and early-career workplaces, could incorporate brief outdoor scheduling, for example positioning breaks or informal seminars outdoors where climate permits, not as a health directive but as a low-cost structural adjustment to the daily indoor concentration identified in this analysis. Student health services could extend baseline blood-screening panels offered to at-risk groups, such as students in extended indoor clinical placements, to include 25-hydroxyvitamin D where local prevalence data suggest elevated risk.

10.2 For Dietary and Food Policy

Because a comparatively small number of foods, namely oily fish, egg yolks, and fortified dairy and cereal products, provide meaningful dietary vitamin D, food fortification policy has particular relevance for young adults whose diets often prioritise convenience and cost over nutrient density.[30]

Expanding fortification of commonly consumed staples, a strategy already used with other nutrients in several countries, offers a population-level correction that does not depend on individual awareness or purchasing power in the way that supplement use does.

10.3 For Clinical Practice

Given the evidence that peak bone mass consolidation occurs substantially within this age window, clinicians treating young adults, particularly those presenting with unexplained fatigue, musculoskeletal pain, or recurrent stress injury, should consider vitamin D status as part of a routine differential rather than reserving testing for pregnancy, elderly, or diagnosed bone-disease populations, as many current guidelines effectively do.[31]

[30]National Institutes of Health, Office of Dietary Supplements, "Vitamin D: Fact Sheet for Consumers."
[31]National Institutes of Health, Office of Dietary Supplements, "Vitamin D: Fact Sheet for Health Professionals."

10.4 For Future Research

Given the methodological limitations identified above, including variable assay cut-offs, concentration of existing research in student cohorts, and unresolved directionality in the depression literature, future research would benefit from longitudinal cohort designs following young adults through this decade specifically, from expansion of sampling beyond university populations to include young adults in manual and outdoor occupations, and from studies designed explicitly to disentangle the direction of the vitamin D-mood association through repeated measures rather than single cross-sectional snapshots.

10.5 For Individuals

Young adults themselves can be encouraged toward modest, non-alarmist behavioural adjustments: brief unprotected sun exposure during safe periods of the day, inclusion of vitamin D-containing foods where accessible, and awareness of personal risk factors such as darker skin pigmentation, which reduces cutaneous synthesis efficiency, or higher body fat, which may require higher intake to achieve equivalent circulating status.[32] These individual measures, however, are best framed as a complement to structural change rather than a substitute for it.[33]

11. Conclusion

Vitamin D deficiency among young adults is neither a marginal clinical curiosity nor a simple failure of personal health habits. The evidence surveyed in this paper, spanning Australia, the Gulf, South Asia, and the United States, points to a consistent pattern of elevated deficiency prevalence in this age group, driven by causes that are structurally embedded in how young adults spend their time: extended indoor education and early-career work, sun-protective norms, and shifting body

[32]Holick, "High Prevalence of Vitamin D Inadequacy," 356.
[33]Horton-French et al., "Prevalence and Predictors," 1633.

composition. The consequences of this deficiency are not uniform in certainty: the skeletal effects rest on well-established mechanisms and carry particular weight in this life stage because peak bone mass is substantially set during these years, while extra-skeletal associations, including the relationship with mood disturbance, remain real but not yet fully disentangled from reverse causation.

What this paper has attempted to show is that causes and consequences in this context are not independent categories but part of an interacting system specific to young adulthood, one in which the timing of deficiency, not merely its severity, carries long-term significance. Addressing the problem effectively will likely require institutional and policy-level responses, including targeted screening practices, food fortification, and structural adjustments to indoor-oriented routines, rather than relying on individual awareness alone. As research on this age group continues to mature, closing the gap between the scale of documented prevalence and the comparatively limited clinical and policy attention it currently receives should be treated as a public health priority rather than an afterthought to paediatric and geriatric vitamin D research.

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