

Factors Affecting Uptake of Vitamin D In Gastro-Intestinal Tract

ABSTRACT:

Vitamin D deficiency affects about 50% [of](#) entire people. Vitamin D deficiency affects an estimated 100 million individuals worldwide, spanning all cultures and age groups. This catastrophic situation of vitamin D deficiency can mostly be attributed to lifestyle and [the](#) normal impact that cuts down exposure to rays from the sun. It is required for the epithelium to produce vitamin D with the help of ultraviolet B (UVB). In comparison to fair-skinned people, dark-skinned people absorb a larger quantity of UVB in their epidermal melanin. More solar exposition is required by fairer folks to get [thean](#) equal amount of the same.

The increased cases of vitamin D deficiency is a significant prevalent ailment caused by vitamin D deficiency. It is a self-governing, deadly cause of widespread mortality all across the world. New research supports hypovitaminosis D as a cause of neoplastic, cardiac, orthopaedic, immunological, NIDDM, and mental disorders. Vitamin D doses are usually compounded to a minimum of 1000 IU by physicians. According to a 2007 study, a high vitamin D intake is linked to a decreased risk of mortality. We've focused on vitamin D research and abstracted the mechanisms that have been linked to vitamin D and its medicinal implications.

Vitamin D is unique in that it may be produced from the epidermis in reaction to UV radiation. Vitamin D₂ is produced in sun-irradiated fungus after the ergosterol is [irradiationirradiated](#) to UV light. When UVB sunlight strikes the epidermis, vitamin D₃ is produced. It's the most "natural" recipe possible. Vitamin D₂ is produced by humans. Vitamin D₃ is produced mostly by oil-rich fish. Chylomicrons are generated when vitamin D is taken orally.

Keywords- Vitamin D, Absorption, GUT, micronutrients, physicochemical, fat-absorption. Obesity.

Vitamin D dietary supplements

A chief basis of vitamin D for homo sapiens is produced due to irradiation of epidermis via UVB light. Vitamin D synthesized in the epidermis lasts double in the circulation relative to dietary vitamin D.[1] If a grownup in ~~swim suit~~ swimsuit is irradiated via UVB with the least amount of dose leads to the production of vitamin D similar to dietary intake of 10,000 and 25,000 IU.[1] A wide range of aspects ~~lessen~~ lessens the epidermis capability of synthesizing vitamin D₃. [2] it includes amplified skin coloration, aging, and the on-skin use of a sunblock.[2]

levels in the gastrointestinal tract

~~Enteric~~ The enteric destiny of vitamin D is supervised by aspects used in chief lipid.[3] It includes emulsification, dissolution in micelles, distribution over the still water layer, and entry ~~in~~ into epithelial cell covering.[3] The enteric destiny of vitamin D is a complex mechanism with physical, chemical, and enzymatic ~~contribution~~ contributions. [3] acid pH of enteric secretion can alter its availability in blood.[4] It's understood that no reliable data is present on the vulnerability of chief nutritional versions of vitamin D in enteric pH circumstances.[4] Additionally, a premise is present which indicates peptide breaking tertiary proteins are aggressively used in vitamin D uptake due to the cutting property of vitamin D associating amino acid polymers in nutrition and ~~help~~ helping in its secretion. also, in 1st part of the intestine, the digestive enzyme remains the release of vitamin D via nutritional products.[5]

Vitamin D absorption mechanism

The process of intake of non-hydroxylated forms is believed to be facilitated via unsaturable non-ATP-based passage in the cell.[4-5] Current research on homo sapiens enteric cell line CaCO₂ and HEK transfected cells reveals the connection of enteric ~~cell~~ cells covering peptidyl polymers intaking the no hydroxylated version on edge of intestinal cells. intake of cholesterol and other lipid-loving compounds are mediated via SR-BI and CD 36 and NPC1L1.[4-5]

Considering the long term interventional and follow-up protocols, also taking into account the discomfort, one's routine gets hampered as basic chores are also challenging to perform, and this leads to an emotional setback for the affected individual and also an

added responsibility for their relatives and friends which eventually becomes burdening.[5] There is no leisure left in that family's life as the patient cannot perform his/her duties, and the family members have to take care of the patient, the patient's duties, and their routine.[6] In addition, social relationships are affected as all the parties are unable to cope emotionally.[6]

The inferences of these proteins indicate a swing in intake via facilitated. It relies on the density of vitamin D. protein facilitated carriage at less density and non-energy dependent passage across increased densities.[7] also, varying vitamin D absorption in various parts of the intestine signifies the occurrence of some different transporter mainly voiced in the 2nd part of the intestine.[7] So, the uptake effectiveness of hydroxylated versions of vitamin D is much more compared to the no hydroxylated forms. No cases of dealing with the cellular intake of hydroxylated species of vitamin D have ever been seen.[8]

Vitamin D deficiency in the diet

It's thought that vitamin D, created in the epidermis, unaided would serve to encounter the day-to-day vitamin D prerequisite. Solid research shows solar irradiation fails to meet recommended dietary allowance of .vitamin D may be caused in response to varying solar contact reliant on weather factors, dress, epidermal colour, time of life, thinness, location on the globe, time of year, also day.[7-8] It necessitates one to meet recommended vitamin D via a dietary basis. One of the most important nutrition-based- vitamin D contains vitamin D₂ and vitamin D₃. [6-8]It is also obtained via pharmaceutical multivitamins, enriched foods, or foods via floral and faunal occurrence.[8] Nutritional intake and skin in vivo formation isare believed to be the method of intake of vitamin D that requires to be extensively managed to prevent avoid excess of vitamin D in the body.[9] due to the Insufficient ground report, it's painstaking to evaluate vitamin D daily consumption precisely as the diet intake -design differs through community financial position.[9] AThe research was done to venture into the day-to-day consumption of vitamin D through diet unaided and coalescing additions and diet.[5-9]

FibresFibers in the diet

Dietary ~~fibres~~fibers are presumed an important performer in determining the destiny in the enteric system.[9] It impacts the availability of vitamin D in blood by given mechanism:

- Hampers the micelle creation
- impacts the discharge of lipid loving ~~substances~~substances out of the fat drop
- Upsurges the viscosity of chyme resulting in the dissemination of lipid-loving dietary substance holding micelles to the cell of the enteric wall.

More than recommended ~~fibrefiber~~ consumption was believed to be the cause of decreased availability of vitamin D in the body. [10]also, greater instances of rickets and osteomalacia ~~isare~~ seen in Asiatic migrant's people.[10] "This presumption was backed by ~~a~~research on the comparative vanishing of D₃ in fit volunteers served high ~~fibrefiber~~ diet (20-g/day) or usual dietary intake.[11] It showed, average via usual dietary intake people was increased (27.5~~± ±~~ 2.1-day days) relative to that of increased ~~fibrefiber~~ intake people (19.2~~± ±~~ 1.7-day days)."[11] increased removal of 3H-25(OH)D₃ in increased ~~fibrefiber~~ intake people might be a result of meddling of the fiber products ~~in~~ blood i.e. association of 3H-25(OH)D₃ to dietary ~~fibrefiber~~. [11]

~~Availability~~The availability of insufficient data on vitamin D uptake in relation to ~~diet~~ included ~~fibres~~fibers, so making an opinion on vitamin D uptake will be a novice. In addition to it, several devoted ~~research~~types of research are needed to appreciate the outcome of fibers on vitamin ~~D's~~D's availability in the body.[11]

levels in the host

The overall vitamin D profile of a person depends on dietary and self-derived ~~vitamin D~~. [12] relation between ~~perorally~~personally taken and self-derived formation of vitamin ~~D~~ is hard to prove.[11-12] It is a result of the fat-loving nature of vitamin D which acts as a reservoir in fatty tissue and its regulated release. Increased consumption accompanied ~~with~~by increased daytime synthesis may cause a severe increase in concentration.[10-12] So, it is believed that increased intake ~~via~~ diet and more irradiation via UVB may lead to ~~its~~ increased levels in the body.[10-12] It leads to a decrease in dietary uptake as well as formation in the skin, but there are no studies that indicate this correlation. It is due to difficulty in ~~correlating~~correlating due to varying ~~amount~~amounts of UV radiation.[12]

Involvement of micronutrients

Vitamin E, as well as K, have a similar mechanism for absorption as vitamin D which may cause competitive inhibition in the gut. It was proved after research on the CaCO₂ cell line.[13] It approves involvement of vitamin E in hampering vitamin D uptake (decreased by 15% on intermediate and 17% at increased conc. of vitamin E) uptake in the GUT.[13] It was proved via an in vitro research on CaCO₂ enteric cell in which it was related to being a probable contender to hampering vitamin D₃ uptake by 16–36%. [12-13]

Relative abundance was seen to be similar uptake. Additionally, it also distinguishes diet in taken D₃ and self-produced D₃ in its use i.e.[12] hindering the use of diet in taken vitamin D₃, but not to self-derived vitamin D₃. In recent times a study showed that increased presence of vitamin A decreases its uptake in body 30%. [14] Whatever be the way, vitamin A way of hampering the vitamin D uptake, still is not found.[14]

Vitamin D absorption enhancers and inhibitors

Literary articles regarding varied mediators that possess the ability to initiate or diminish vitamin D uptake via the intestine. Such mediators may be available in dietary products or can be supplemented to facilitate greater uptake of vitamin D. context from such literary articles are presented here-. [15]

Fat-absorption inhibitors

~~Individual~~ The individual struggling with obesity takes various obesity-reducing pills and fat alternates to decrease the fat amount. Pills and fat alternates decrease the uptake of triglycerides. [12] As ~~vitamin~~ Vitamin D trails the same league of triglyceride in the GUT. It is believed that fat ~~reducing~~ reduction can hinder vitamin D availability in the body causing a decrease in its uptake. [13] “The uptake of vitamin D is hindered if vitamin D was given with a fat alternative was given to 102 fit men and women. also, the cholesterol derived via plant source which is taken to decrease cholesterol uptake, affects the availability of vitamin D in the body.” [13-15] it was established by numerous ~~researches~~ researchers. Reduction in vitamin D conc. in serum and in the liver was found in rats after they were administered stanol ester for 3 months. [13-15] It was done for numerous phytosterols in ~~rat~~ rats and in vitro and inferred that ~~phytocholesterols~~ Phyto cholesterol are a reason for decreased micelles production and passage in enteric cells causing availability decrease in the body. Newer reports on patients don't recommend this theory by opposing the outcome of phytosterol on

[the](#) availability [of](#) vitamin D in [the](#) body. Such inferences are generally disproved as assessment was done on the [basebasis](#) of the serum level of 25(OH)D.[13-15] it might be changed by endogenous vitamin D production which relies [-on](#) the various elements i.e.[14] “UVB irradiation [-and](#) weather. Additionally, it was once more backed by findings of two clinical trials where serum level of 25(OH)D was monitored in diverse people.”[15] They were given plant sterol ester improved meal. the fat decreasing mediators decrease fat uptake hampering [the](#) uptake of vitamin D. the precise quantity of lipid desired for maximum uptake of vitamin D is not adjusted. The patients are burning with anxiety and questions for the oncologists.[15] If a psycho-oncologist can take care of this professionally, both the patients and the treating clinician will be at ease with each other, and there will be a better understanding from both parties.[15] The patients and families, amidst the chaos, can have someone to counsel them.[15] Even if the patient succumbs, the psycho-oncologist must counsel the family members as their spectrum is not limited to one individual.[15] Even the family trusts the psycho-oncologists more as they have a one-on-one interaction for a long time.[15]

Vitamin D enhancers

vitamin D transport in dedicated preparation is believed to have improved [the](#) availability of vitamin D in [bodythe body's](#) enteric canal.[16][17] It can cause increased availability of micro/nano covered vitamin D compared to its enriched dietary products. [PresenceThe presence](#) of Inadequate works [-on](#) these factors causes [the](#) inability of [a](#) conclusion about [the](#) effects of such forms of vitamin D on its availability in [the](#) body.[17]

GIT secretion physiochemical interaction

Uptake of vitamin D via gut wall is thought to be highest inside a bracket of salt ionic concentration and pH past which its absorption might be hindered. This supposition was found [-in](#) numerous researches.[16] Absorption of vitamin D₃ was found to be hindered with varying salt conc. Within normal.[15] the Gradual decrease in vitamin D₃ uptake was found as the sodium taurocholate salt levels were changed beyond the 5 mM (10 or 15 mM).[15]

Factors affecting the host

articles reflect the participation of numerous host-related aspects that might be related factors in determining its availability in the body. numerous researches that were done leading to enhanced suggested dietary recommendation due to result of aspects (age and disease obesity).[18]

Host's age

Biological changes in body functioning isare observed with advancing age. ~~Its~~ It is presumed that the age-related physical deviations might affect vitamin D availability in the body.[19] Age-related discrepancies in lipoprotein breakdown waswere supposed for the decrease of uptake and post food intake carriage of vitamin D.[19] Numerous researches have led to the finding which ~~indicate towards~~ indicates decreased vitamin D grade in aged persons compared to younger progenies.[19] Initial research was done with 20 aged females, they presented with decreased levels of [3H]cholecalciferol compared to ~~of~~ young femalefemales. [19] It suggested that Gut of aged females was competent as compared to their younger progenies.[20] Similar results were not replicated in mice-. [20] Convincing reports concerning variations in serum 25OHD levels in aged compared to younger people might be because of low self-produced vitamin D epidermis, lesser solar irradiation, and its less amount in food intake.[20]

Obesity

it's usually destructively associated ~~to~~ with a lack of vitamin D.[21] it was backed by Liel et al. (1988) research accd. To which they found- better uptake and increased removal of vitamin D by overweight patients compared to regular. Contrariwise vitamin D's storage in fatty tissue ~~doesn't~~ doesn't cause its release during requirement causing its increased availability of vitamin D.[21] in a research conducted on aged subjects concluded that on supplementing.[21] Decreased concentration in overweight people might be a result of Dec. conc. of vitamin in their big fatty build. Findings The findings here isare that vitamin D accumulated in fatty structures isn't released easily.[21] Overweight people might need a greater dosage to establish a serum 25OHD concentration similar to proper weight beings. Increased serum 25(OH)D concentration in the period of fat loss in overweight people proves this speculation.[21]

Diseases and surgeries involving the digestive tube

Vitamin D is readily absorbed if ingested with fat-containing meals, according to many research and investigations. First researches revealed that those with a clogged GI tract, had much lower vitamin D uptake.[3-6] Roux-en-Y gastric bypass surgery resulted in a 30% decrease in blood vitamin D3 levels compared to previously.[3-6][7] Moreover, people with cystic fibrosis were found to have worse vitamin D intake efficiency than their healthy counterparts.[7] “Vitamin D is also believed to have a favourable/favorable effect on the intestine and CD8+ cells, which might also help to maintain the stability of the Gut mucosal lining by modulating intercellular connections, reducing mucosal leakage, and raising CD8+ cell numbers. Furthermore, certain clinical investigations have shown that vitamin D has a role in cancer prevention, particularly GI cancer, by modifying tumorigenic indicators, VDR polymorphism, and other VDR regulations. However, these findings support the role of vitamin D in prevention of illness, there is a lack of information absorption in the gastrointestinal tract is affected by these conditions” [7] medical stage, treatment protocols as mentioned above, and the patient's prognosis in coordination with the medical oncologist.[3-5] If the patient has a poor prognostic outcome, the psycho-oncologist should be ready with cognitive therapy for the patient in such situations.[3-5] The patient's past and current course of medical illness are noted. Risk factors for cancer, including environmental, genetic, and behavioral, are looked for, and the current medications are studied to rule out if the behavioral changes result from the adverse reactions.[8-15]

Variations in genetics

Vitamin D intake is controlled by a fat-digesting enzyme, bile secretion, and a vitamin D converting liver enzyme.[11] Modifying the gene sequence can affect the production and function of these ~~protein~~proteins, resulting in a partial or full loss in performance.[12] Moreover, changes in the genetic sequence of ~~neighbouring~~neighboring genes may disrupt transcription factor engagement, resulting in the lack of these protein transporters. There is currently no data in the literature that addresses this topic.[12] In the same way, genetic variations in fat-digesting enzyme and vitamin D protein complex might alter vitamin D uptake. Entities like support groups and psychotherapy are familiar for cancer patients in developed countries.[16-22] With this review, the authors wish to emphasize making psychological care obligatory for cancer patients, in the form of regular counseling,

support groups, inclusion on psycho-oncological consultation in the treatment protocols, feedback of the psycho-oncologists to the treating clinician.[23-30]

Conclusion

In various areas of the globe, a vast variety of food items are accessible; each meal product's matrix varies owing to differences in nutritive value, lipid content, dietary ~~fibres~~fibers, and other factors. All of these factors make estimating the absorption of vitamin D in a specific diet challenging. The method of vitamin D uptake is not well understood in the current research. Even though some factors governing vitamin D fate in the GI tract have been well documented, others, such as genetic differences, dietary ~~fibrefiber~~. More ~~specialised~~specialized studies using ~~labelled~~labeled vitamin D are necessary for understanding absorption, targeting research directions potential. Biological changes in body functioning ~~isare~~ are observed with advancing age. ~~Its-It's~~ It's presumed that the age-encouraged physical deviations might affect vitamin D availability in ~~the~~ body. Age-related discrepancies in lipoprotein breakdown ~~waswere~~ were supposed for ~~the~~ decrease of uptake and post food intake carriage of vitamin D. Numerous researches have led to the finding which ~~indiate~~indicates towards- decreased vitamin D grade in aged persons compared to younger progenies. Initial research was done with 20 aged females, they presented with decreased levels of [3H]cholecalciferol compared to ~~of~~-young ~~female~~females. It suggested that Gut of aged females was competent as compared to their younger progenies. Similar results were not replicated in mice-. Convincing reports concerning variations in serum 25OHD levels in aged compared ~~to~~ younger people might be because of low self-produced -vitamin D epidermis, lesser solar irradiation, and its less amount in food intake.

References

1. Holick MF. Vitamin D deficiency. *N Engl J Med.* 2007;357:266–81.
2. Gordon CM, DePeter KC, Feldman HA, Grace E, Emans SJ. Prevalence of vitamin D deficiency among healthy adolescents. *Arch Pediatr Adolesc Med.* 2004;158:531–7.
3. Lips P, Hosking D, Lippuner K, Norquist JM, Wehren L, Maalouf G, et al. The prevalence of vitamin D inadequacy amongst women with osteoporosis: An international epidemiological investigation. *J Intern Med.* 2006;260:245–54.

4. Rostand SG. Ultraviolet light may contribute to geographic and racial blood pressure differences. *Hypertension*. 1997;30:150–6.
5. Melamed ML, Michos ED, Post W, Astor B. 25-hydroxyvitamin D levels and the risk of mortality in the general population. *Arch Intern Med*. 2008;168:1629–37.
6. Harvard School of Public Health Nutrition Source. *Vitamin D and health*. [Last accessed on 2010 Aug 30].
7. Autier P, Gandini S. Vitamin D supplementation and total mortality: A meta-analysis of randomized controlled trials. *Arch Intern Med*. 2007;167:1730–7.
8. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes Food and Nutrition Board, Institute of Medicine. Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. Chapter 7. Vitamin D. [Last accessed on 2010 Aug 02].
9. NIH Office of Dietary Supplements. *Dietary supplement fact sheet: Vitamin D*. [Last accessed on 2010 Aug 04].
10. Nair S. Symptoms of low vitamin D levels. [Last accessed on 2010 Sep 02].
11. MedlinePlus. 25-hydroxy vitamin D test. [Last accessed on 2010 Aug 04].
12. Moyad MA. Vitamin D: A rapid review: Side effects and toxicity. [Last accessed on 2010 Sep 02].
13. Lappe JM, Travers-Gustafson D, Davies KM, Recker RR, Heaney RP. Vitamin D and calcium supplementation reduces cancer risk: Results of a randomized trial. *Am J Clin Nutr*. 2007;85:1586–91.
14. Chlebowski RT, Johnson KC, Kooperberg C, Pettinger M, Wactawski-Wende J, Rohan T, et al. *J Natl Cancer Inst*. 2008;100:1581–91.
15. Stolzenberg-Solomon RZ, Vieth R, Azad A, Pietinen P, Taylor PR, Virtamo J, et al. A prospective nested case-control study of vitamin D status and pancreatic cancer risk in male smokers. *Cancer Res*. 2006;66:10213–9.
16. Stolzenberg-Solomon RZ, Hayes RB, Horst RL, Anderson KE, Hollis BW, Silverman DT. Serum vitamin D and risk of pancreatic cancer in the Prostate, Lung, Colorectal, and Ovarian Screening Trial. *Cancer Res*. 2009;69:1439–47.

17. Wang TJ, Pencina MJ, Booth SL, Jacques PF, Ingelsson E, Lanier K, et al. Vitamin D deficiency and risk of cardiovascular disease. *Circulation*. 2008;117:503–11.
18. Hollis BW. Circulating 25-hydroxyvitamin D levels indicative of vitamin D sufficiency: Implications for establishing a new effective dietary intake recommendation for vitamin D. *J Nutr*. 2005;135:317–22.
19. Holick MF, Chen TC. Vitamin D deficiency: A worldwide problem with health consequences. *Am J Clin Nutr*. 2008;87:1080S–6S.
20. Matsuoka LY, Ide L, Wortsman J, MacLaughlin JA, Holick MF. Sunscreens suppress cutaneous vitamin D₃ synthesis. *J Clin Endocrinol Metab*. 1987;64:1165–8.
21. Clemens TL, Henderson SL, Adams JS, Holick MF. Increased skin pigment reduces the capacity of skin to synthesise vitamin D₃. *Lancet*. 1982;1:74–6.
22. Sahu, Preeti Rajendra, Kishor Madhukar Hiwale, Sunita Jayant Vagha, and Samarth Shukla. “Spectrum of Lesions on Upper Gastrointestinal Endoscopy and Its Correlation with Histopathological Evaluation.” *JOURNAL OF EVOLUTION OF MEDICAL AND DENTAL SCIENCES-JEMDS* 9, no. 32 (August 10, 2020): 2301–6. <https://doi.org/10.14260/jemds/2020/498>.
23. Kirnake, Vijendra, Anil Arora, Praveen Sharma, Mohan Goyal, Romesh Chawlani, Jay Toshniwal, and Ashish Kumar. “Non-Invasive Aspartate Aminotransferase to Platelet Ratio Index Correlates Well with Invasive Hepatic Venous Pressure Gradient in Cirrhosis.” *INDIAN JOURNAL OF GASTROENTEROLOGY* 37, no. 4 (July 2018): 335–41. <https://doi.org/10.1007/s12664-018-0879-0>.
24. Sahu PR, Hiwale KM, Vagha SJ. Study of Various Gastrointestinal Tract Lesions by Endoscopic Biopsies in a Tertiary Care Centre of Rural District of Maharashtra. *JOURNAL OF EVOLUTION OF MEDICAL AND DENTAL SCIENCES-JEMDS*. 2021 Apr 19;10(16):1135–9.
25. Abbafati, Cristiana, Kaja M. Abbas, Mohammad Abbasi, Mitra Abbasifard, Mohsen Abbasi-Kangevari, Hedayat Abbastabar, Foad Abd-Allah, et al. “Five Insights from the Global Burden of Disease Study 2019.” *LANCET* 396, no. 10258 (October 17, 2020): 1135–59.
26. Abbafati, Cristiana, Kaja M. Abbas, Mohammad Abbasi, Mitra Abbasifard, Mohsen Abbasi-Kangevari, Hedayat Abbastabar, Foad Abd-Allah, et al. “Global Burden of 369 Diseases and Injuries in 204 Countries and Territories, 1990-2019: A Systematic Analysis for the Global Burden of Disease Study 2019.” *LANCET* 396, no. 10258 (October 17, 2020): 1204–22.
27. James, Spencer L., Chris D. Castle, Zachary Dingels V, Jack T. Fox, Erin B. Hamilton, Zichen Liu, Nicholas L. S. Roberts, et al. “Estimating Global Injuries Morbidity and Mortality: Methods and Data Used in the Global Burden of Disease

- 2017 Study.” INJURY PREVENTION 26, no. SUPP_1, 1 (October 2020): 125–53.
<https://doi.org/10.1136/injuryprev-2019-043531>.
28. James, Spencer L., Chris D. Castle, Zachary Dingels V, Jack T. Fox, Erin B. Hamilton, Zichen Liu, Nicholas L. S. Roberts, et al. “Global Injury Morbidity and Mortality from 1990 to 2017: Results from the Global Burden of Disease Study 2017.” INJURY PREVENTION 26, no. SUPP_1, 1 (October 2020): 96–114.
<https://doi.org/10.1136/injuryprev-2019-043494>.
29. James, Spencer L., Chris D. Castle, Zachary Dingels V, Jack T. Fox, Erin B. Hamilton, Zichen Liu, Nicholas L. S. Roberts, et al. “Estimating Global Injuries Morbidity and Mortality: Methods and Data Used in the Global Burden of Disease 2017 Study.” INJURY PREVENTION 26, no. SUPP_1, 1 (October 2020): 125–53.
<https://doi.org/10.1136/injuryprev-2019-043531>.
30. James, Spencer L., Chris D. Castle, Zachary Dingels V, Jack T. Fox, Erin B. Hamilton, Zichen Liu, Nicholas L. S. Roberts, et al. “Global Injury Morbidity and Mortality from 1990 to 2017: Results from the Global Burden of Disease Study 2017.” INJURY PREVENTION 26, no. SUPP_1, 1 (October 2020): 96–114.
<https://doi.org/10.1136/injuryprev-2019-043494>.