# Seasonal Variation in Vitamin D Status of Auckland Intermediate (11-12 years) School Children

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**Abstract**

Vitamin D is an essential pro-hormone required for calcium homeostasis, bone mineralisation, and wider immune and metabolic functions. In New Zealand, vitamin D deficiency and insufficiency remain public-health concerns, particularly among children. Sun-safe behaviours, limited dietary sources and seasonal variation in ultraviolet beta (UVB) exposure are some of the factors that contribute to risk. This research investigated vitamin D status and UVB exposure from summer and winter in Auckland intermediate school children.

A longitudinal repeated-measures study (the DISC study) was conducted in 100 intermediate school children aged 11–12 years. Serum 25-hydroxy-vitamin D (s-25(OH)D) was assessed using dried-blood spots in March (after summer exposure) and September (after winter exposure). UVB exposure was measured using personal dosimeters worn for 10 days at each timepoint. Anthropometric data and self-reported behaviours were also collected. Statistical analyses examined seasonal differences in s-25(OH)D and UVB dose, and predictors of seasonal change.

Mean s-25(OH)D concentrations fell from 90 ± 22 nmol/L in summer to 65 ± 18 nmol/L in winter, a mean percentage change of -26±17% ($p < 0.0001$). The proportion of children classified as vitamin D deficient (< 50 nmol/L) rose from 3 % to 20 %, and those insufficient (< 75 nmol/L) from 21 % to 50 % from summer to winter. Mean UVB dose decreased from 9.2±5.0 Standard Erythemal Doses (SED) in summer to 3.1±1.1 SED in winter, a mean decrease of -6.1±28 SED ($p < 0.0001$). Greater seasonal reductions in s-25(OH)D occurred in children with higher summer concentrations (≥75 nmol/L), -30±17 vs -9.1±11 nmol/L ($p = <0.001$) and elevated body-fat percentage (>25% for boys and >30% for girls), -29±15 vs -24±19 nmol/L ($p = 0.04$). Both summer s-25(OH)D and body-fat percentage independently predicted winter decline ($R^2 = 0.45$, $p < 0.001$), suggesting impaired mobilisation of vitamin D from adipose stores rather than altered metabolism.

These findings suggest substantial seasonal variation in vitamin D status among Auckland school-aged children and highlight adiposity as a significant factor for insufficiency. Despite high exposure potential in summer, half of the cohort was insufficient by winter, indicating limited capacity to maintain optimal vitamin D status year-round. These results emphasise the need for evidence-based public-health guidance balancing sun-safety and vitamin sufficiency, consideration of targeted supplementation or fortification strategies, and

possible tailored guidance for specific at-risk groups. Understanding and addressing determinants of vitamin D insufficiency in childhood is essential to protect long-term musculoskeletal, immune, and metabolic health of this population group.

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## List of Abbreviations

1, 25(OH)D: 1, 25-hydroxyvitamin D (calcitriol)
$25(OH)_2D$: 25-hydroxyvitamin D (calcidiol)
7DHC: 7-dehydrocholesterol
ASD: Autism Spectrum Disorder
BF%: Body Fat Percentage
BIA: Bioelectrical Impedance Analysis
BMI: Body Mass Index
CVD: Cardiovascular Disease
$D_2$: Vitamin $D_2$ (ergocalciferol)
$D_3$: Vitamin $D_3$ (cholecalciferol)
IU: International Unit
MED: Minimal Erythemal Dose
MoH: Ministry of Health
NZ: New Zealand
PCOS: Polycystic Ovary Syndrome
PTH: Parathyroid Hormone
PreD: Pre-vitamin D
s-25(OH)D: Serum 25-hydroxyvitamin D
SED: Standard Erythemal Dose
SPF: Sun Protection Factor
UVB: Ultraviolet B
VDBP: Vitamin D Binding Protein
VDR: Vitamin D Receptor

# Chapter 1: Introduction

## *1.1 Background:*

Vitamin D is an essential pro-hormone which can either be produced endogenously from exposure to ultraviolet beta (UVB) radiation from sunlight or can be taken in through consumption of vitamin D containing foods (either naturally or through fortification) or supplements. It is known to play a vital role in calcium homeostasis and bone health, with deficiency linked to osteomalacia in adults and rickets in children (Chanchlani et al., 2020; Minisola et al., 2020). The vitamin D receptor, however, is expressed in most bodily tissues and low vitamin D status has also been related to various other conditions including respiratory disease, cancer, autoimmune conditions such as type 1 diabetes, cardiovascular disease, metabolic disorders such as type 2 diabetes, allergies and eczema, and autism spectrum disorder (Lucas et al., 2008; Mazahery et al., 2016; Shah et al., 2024; Zeng et al., 2023).

Rickets is a condition characterised by abnormal calcium and phosphate homeostasis and impaired bone mineralisation resulting in skeletal abnormalities and growth retardation (Chanchlani et al., 2020). Global consensus highlights vitamin D deficiency as a major contributing factor (Siafarikas et al., 2020). Rickets is considered a problem both globally and in New Zealand where incidence in children <15 years has been found to be 2.2/100,000, rising to 10.5/100,000 in those <3 years (Palacios & Gonzalez, 2014; Wheeler et al., 2015). Vitamin D status has also been linked to respiratory infections, with low status increasing the likelihood of disease (Bergman et al., 2013). Further, in infants, adverse respiratory outcomes, such as asthma and wheezing have been shown to be higher in those with mothers who had lower vitamin D status (Jensen et al., 2019). This is significant as respiratory diseases are a large contributor to mortality in the New Zealand childhood population (Te Rōpū Arotake Auau Mate o te Hunga Tamariki, 2021). Food allergies and eczema, conditions often prevalent in childhood, have also been associated with low vitamin D status (Heimbeck et al., 2013; Zeng et al., 2023). Vitamin D is known to play a role in gene modulation, with some of its target genes linked to the immune system and food allergy pathogenesis. Therefore, potentially through epigenetic pathways vitamin D status could affect the development of food allergies in those with a predisposition (Poole et al., 2018). Lastly, autism spectrum disorder, a condition with a rising prevalence among children, has been linked to vitamin D deficiency. This is thought to be through anti-inflammatory and neuro-protective effects of vitamin D in the brain, with supplement use being shown to reduce symptoms (Cannell, 2017; Mazahery et al., 2019).

Good dietary sources of vitamin D include oily fish (e.g. salmon or mackerel) and fish liver oils (e.g. cod liver oil). Other dietary sources are limited and less potent but include egg yolks, beef and some mushrooms (which have been in sunlight and synthesised their own vitamin D). It is however thought that vitamin D sufficiency would be impossible to meet through diet alone (Ministry of

Health, 2012a). There is no fortification programme of vitamin D in New Zealand, unlike in other countries such as Australia and Finland. These factors mean dietary vitamin D contributes only a small amount to vitamin D status making it important for both children and adults to gain adequate UVB exposure to decrease the likelihood of vitamin D insufficiency or worse deficiency in New Zealand populations.

UVB exposure is considered to provide most of a person's vitamin D (estimated at over 90%) (Ministry of Health, 2012b). However, exposure to UVB radiation is a risk factor for skin cancer and New Zealand experiences some of the highest skin cancer rates in the world, as a result there is strong sun safe public health messaging (Pondicherry et al., 2018). This messaging may be to the detriment of vitamin D status with research in athletes showing that this group is more concerned about staying sun safe than their vitamin D status (Walker et al., 2014). UVB is at its maximum when the sun is directly overhead, i.e. in the middle of the day, but current cancer prevention guidelines suggest avoiding the sun during the hours of 11am and 3pm, reducing capacity for optimal vitamin D production in the skin. There are also large seasonal variations in UVB radiation in New Zealand, with the daily winter intensity being as much as 95% lower than the summer dose (Livesey et al., 2007). Research examining the relationship between UVB dose on vitamin D status in children in New Zealand is lacking, however increasing this knowledge would help to provide more informed guidance around optimising vitamin D status.

Vitamin D status is measured through a blood test examining the concentration of 25-hydroxyvitamin D (s-25(OH)D) in the serum. Toxicity is considered >250 nmol/L, sufficiency is controversial but has been defined in NZ and Australia as >50 nmol/L, insufficiency as 30 - 50 nmol/L and deficiency as <30 nmol/L (Ministry of Health, 2012a; Siafarikas et al., 2020). Deficiency, however, is often asymptomatic, allowing potential for damage to health without awareness and there is debate as to whether a higher cutoff (>75 nmol/L) is needed to encompass requirements for non-skeletal functions (Siafarikas et al., 2020). A New Zealand Ministry of Health report looking at the vitamin D status of New Zealand adults found around 5% had vitamin D deficiency and 27% were below recommended concentrations (Ministry of Health, 2012b). In children (5–14 years) 31% were found to be insufficient and 4% deficient and this risk was increased during the winter (Rockell et al., 2005). Further research in Auckland primary school children found 1/3 of this population were insufficient (Delshad, 2019).

Vitamin D status is dependent on several factors including skin colour, ethnicity, amount of UVB exposure, seasonal changes in UVB concentrations, latitude of where a person lives, age, sex, body composition and behavioural factors such as amount of time spent outdoors and the use of sun protective clothing and sunscreen (Wacker & Holick, 2013). Global data show a higher prevalence of hypovitaminosis D (vitamin D deficiency) in those living in higher latitudes, the prevalence of deficiency was 1.7 times higher during winter through to spring, than during summer to autumn, females to be more vulnerable to deficiency and those aged under 18 years had a higher prevalence of having s-25(OH)D <50 nmol/L than those aged 45-64 years (Cui et al., 2023). A New Zealand Ministry of Health report found a mean difference in 25(OH)D concentrations from winter to summer of 15 nmol/L and vitamin D status to be 15 nmol/L lower for Pacific People and 7nmol/L lower for Māori than for New Zealand European children (Rockell et al., 2005). Deficiency was also found to be prevalent in 13% of children under 2 years and higher in Pacific People (Grant et al., 2009). New Zealand population specific research on vitamin D status and the factors that influence this, however, is still lacking and further study is needed, especially in childhood populations, to fully elucidate what is happening in New Zealand.

Research on knowledge, awareness and attitudes concerning vitamin D shows that there are gaps in individuals' knowledge, and this may further be influencing vitamin D status (Fitzgerald et al., 2023; Franklin, 2019). As hypovitaminosis D in childhood has been linked to so many adverse outcomes and is found in higher prevalence in those populations which have an increased vulnerability to poor health outcomes, it is important to increase our understanding of the landscape in New Zealand (Antonucci et al., 2018). By examining vitamin D status among school aged children in New Zealand and understanding some of the factors influencing this, we will have a better handle on what, if anything, needs to be done to reduce the likelihood of the potential detrimental effects that vitamin D deficiency may cause to the population's health.

## *1.2 Purpose of this study:*

To increase knowledge of vitamin D status in childhood and further understand the relationship between vitamin D status and its change through seasonality and UVB exposure. The findings of this research will allow more informed guidance about sun exposure, potential vitamin D supplementation and the need for food fortification in New Zealand (Bolland et al., 2021).

## *1.3 Aim:*

To determine seasonal variation, from summer to winter, for both vitamin D status and UVB dose in a sample of Auckland school children aged 11-12 years and whether this is influenced by other factors.

## *1.4 Objectives:*

- Assess serum 25-hydroxy-vitamin D (s-25(OH)D) status in Auckland school children aged 11-12 years at two time points, March (the beginning of autumn, after summer sun exposure) and September (the beginning of spring, after winter sun exposure) using a finger prick blood spot test.
- Compare the seasonal change in s-25(OH)D status in Auckland school children aged 11-12 years after the summer season and after the winter season and what factors influence this change.
- Assess UVB dose in Auckland school children aged 11-12 years during a 10-day period, at two time points, March (the beginning of autumn) and September (the beginning of spring) measured by Standard Erythemal Dose (SED) using data from dosimeters worn by the children during the day.
- Compare the seasonal change in UVB exposure for Auckland school children aged 11-12 years over a 10-day period using SED between spring and autumn and what factors influence this change.

## *1.5 Hypotheses:*

- Vitamin D status will be lower after winter compared to summer.
- UVB exposure will be less during autumn/winter than spring/summer.

## *1.6 Structure:*

Following on from this introductory chapter, chapter two is a literature review, examining relevant work in the field. It will firstly examine what vitamin D is, how humans acquire it and how it is metabolised in the body. Then vitamin D status will be explored including, how this is measured and

relevant cutoffs of sufficiency, insufficiency and deficiency. Followed by discussion of what we already know regarding status both globally and in New Zealand and the factors that affect status. The review will conclude by discussing the function of vitamin D in the body, why it is important to understand vitamin D status, especially in children and health issues related to vitamin D insufficiency and deficiency looking at both global and New Zealand related evidence.

Chapter three presents the findings of the research examining vitamin D status of Auckland school children aged 11-12 years in manuscript format.

Chapter four comprises the overall discussion and conclusions of the thesis, including assessment of the strengths and limitations and recommendations for future areas of research.

Appendices include the DISC (Vitamin **D** Status in **I**ntermediate **S**chool **C**hildren) study participant information sheet and demographic questionnaire.

References for the complete thesis are included at the end of the document.

## *1.7 Researcher Contribution:*

| Researcher | Contribution |
|---|---|
| Dr Cassie Slade | Thesis author, data analysis, manuscript author |
| Professor Cathryn Conlon | Supervision |
| Professor Pamela von Hurst | Supervision |
| Dr Hajar Mazahery | Statistical analysis |
| Owen Mugridge | Recruitment and data collection |

# Chapter 2: Literature Review

## *2.1 Literature Review Overview and Structure*

The literature review begins with an overview of vitamin D and how it is metabolised in the body. It looks at how the body acquires vitamin D both through UVB exposure and through diet and how this might contribute to status. Both UVB exposure and vitamin D status are discussed, including how these are determined. Definitions of vitamin D sufficiency, insufficiency, deficiency and toxicity are outlined and the current understanding of vitamin D status both globally and in New Zealand, focussing on school-aged children, are considered. The review moves on to look at the various factors that influence vitamin D status, especially those pertinent to young people. Finally discussing the bodily functions where vitamin D is known to play a role and the potential health issues that could occur, with a focus in school-aged children, with inadequate vitamin D status. The review closes by considering why the study of vitamin D is important, with an overview of its pertinence in young people and how this investigation contributes to this significant area of research.

Search strategies for this review included searches of Massey University's Discover, Web of Science, PubMed and Google Scholar databases. The keywords used included ("vitamin D" OR "25-hydroxyvitamin D" OR "25(OH)D") AND status AND (children OR adolescents OR youth OR child) AND ("New Zealand" OR Aotearoa OR NZ). Further searches included the keywords ("vitamin D" OR "25-hydroxyvitamin D" OR "25(OH)D") AND (guidelines OR "consensus statement") AND ("New Zealand" OR Aotearoa OR NZ). Additional review material was obtained through research referenced by articles highlighted in the searches and discussion with colleagues working in the field providing recommendations of research to read.

## *2.2 Introduction*

Vitamin D refers to a group of fat-soluble steroid prohormones, predominantly produced through sunlight on the skin and historically known to play a role in the metabolic pathways of both calcium and bone (Acar & Özkan, 2021; Saponaro et al., 2020). The association of inadequate sunlight exposure and the then common childhood bone-deforming disease rickets was first recognised in the early 1800s. The disease was eradicated as a major public health issue in the early 19$^{th}$ century, through both environmental improvements and supplementation (Rajakumar, 2003; Sniadecki & Sniadecki, 1939). However, a recent re-emergence has been seen in New Zealand children (Wheeler et al., 2015). Current research has shown the wider scope of vitamin D and its involvement in the metabolic processes of numerous non-skeletal tissues (Bouillon et al., 2018). As the research in this field expands there is increasing evidence that vitamin D deficiency is implicated in negative health outcomes, including those that cause a major risk to children's health such as respiratory diseases

which are a major cause of hospitalisation and mortality in children in NZ (Telfar Barnard & Zhang, 2021). In the past few years childhood hypovitaminosis D has been re-emerging as a public health issue (Creo et al., 2017). Understanding vitamin D status in differing populations, especially children and adolescence, is therefore crucial to inform public health policy and to implement preventative health measures to improve morbidity and mortality.

## *2.3 Vitamin D*

Vitamin D exists in two forms, vitamin D2 (Ergocalciferol) produced through the effects of UVB radiation on ergosterol in plants, yeasts and fungi, and vitamin D3 (Cholecalciferol) produced through the effects of UVB radiation on 7-dehydrocholesterol in the skin of animals (Jeon & Shin, 2018). These two molecules are very similar, differing only in their side-chain structures and are metabolised by the body to produce biologically active molecules (Procsal et al., 1976). Humans therefore obtain vitamin D in two ways, exogenously through dietary sources or endogenously through synthesis within their skin. However, adequate intakes of vitamin D are hard to achieve through diet alone (Ministry of Health, 2012a). As there is little vitamin D in breastmilk and putting infants in direct sunlight is not encouraged, supplementation is recommended for exclusively breastfed infants in New Zealand (Health New Zealand, 2024). Further, at risk groups such as people with naturally very dark skin, people who avoid sun exposure, experience little sun exposure or people with medical conditions that affect their vitamin D levels may also be recommended supplementation (Ministry of Health, 2012a).

## *2.4 Vitamin D metabolism*

Exogenous dietary sources contain both D2 and D3 forms and include oily fish, egg yolks, liver, red meat, mushrooms (left in the sunlight to produce their own vitamin D), fortified products e.g. plant-based milks and margarines, and supplements. These are thought to make up approximately 10-20% of the vitamin D sources in humans, although this may contribute less in children as these are foods may not be readily consumed by this population (Holick, 2002; Lamberg-Allardt, 2006). Dietary sources are absorbed in the small intestine both through passive diffusion and via intestinal membrane carrier proteins (Silva & Furlanetto, 2018). Vitamin D will be absorbed without the simultaneous presence of fat in the intestine, however this increases absorption (Silva & Furlanetto, 2018). It is transported in chylomicrons, a type of lipoprotein, via the lymphatic system to the hepatic portal circulation where the endogenous and exogenous vitamin D are metabolised along the same pathway.

The pathway of endogenous D3 synthesis starts with skin exposure to ultraviolet B (UVB) light (290-315nm wavelength range). UVB light causes a rearrangement of the 7-dehydrocholesterol (7DHC)

molecule present in the epidermis of the skin to form pre-vitamin D3 (PreD3) (Acar & Özkan, 2021). Thermal isomerisation then transforms PreD3 to vitamin D3. This pathway is controlled in periods of long UVB exposure, to prevent production of toxic concentrations of vitamin D, by PreD3 also being turned into the inactive metabolites lumisterol or tachysterol (Acar & Özkan, 2021). Vitamin D3 diffuses from the skin into circulation, binding to vitamin D binding protein (VDBP) which transports it to the liver. Vitamin D2 and D3 differ slightly in molecular structure and this difference means vitamin D2 has a lower affinity for VDBP, allowing it to be catabolised and removed from circulation more easily (Acar & Özkan, 2021).

In the liver vitamin D undergoes a hydroxylation reaction by vitamin-D-25-hydroxylase (CYP27A1) to form 25-hydroxyvitamin D (25(OH)D) or calcidiol. From there it is bound to VDBP and transported to other bodily tissues where it undergoes a second hydroxylation reaction by the enzyme 1-α-hydroxylase (CYP27B1) to become the active hormone 1,25-dihydroxy vitamin D ($1,25(OH)_2D$) or calcitriol. This process happens primarily in the kidney tubule cells but also at other sites such as the skin, lungs, colon and immune cells (Bikle et al., 2018). The active hormone then enters circulation primarily from the kidney, calcitriol made in extra-renal sites does not contribute significantly to circulating concentrations, and is able to influence specific cells by binding to and activating the Vitamin D Receptor (VDR) (Bikle et al., 2018; Jeon & Shin, 2018). Whether the active hormone has been derived from vitamin D2 or D3 may affect its efficacy due to their differing affinities for the VDR, with the metabolite derived from vitamin D3 potentially having a greater effect (Houghton & Vieth, 2006). When activated the VDR induces both genomic and non-genomic regulation of targets downstream. The genomic pathway involves calcitriol binding to cytosolic VDR causing phosphorylation of VDR and heterodimerisation with the retinoid-X receptor. This calcitriol-VDR-RXR complex is translocated to the nucleus where it binds to vitamin D response element (VDRE) and regulates mRNA expression of target genes (Jeon & Shin, 2018). The non-genomic pathway involves calcitriol binding to membrane bound VDR, also called 1,25D-membrane-associated rapid response steroid-binding protein (1,25D-MARRS). This binding causes changes to intracellular signalling molecules which affect phenotypic functions through protein to protein interactions (Jeon & Shin, 2018).

An alternative metabolic pathway for vitamin D is through the enzyme CYP11A1 (Slominski et al., 2005). The hydroxymetabolites of vitamin D generated from CYP11A1 and further catalysis by the enzymes CYP27A1 and CYP24A1 have been shown to influence anti-proliferation, differentiation and anti-inflammation in skin cells, improving defence against sun damage and oxidative stress (Slominski et al., 2017). As well as the VDR, retinoid-related orphan receptors (RORα and RORγ) act as receptors for the hydroxymetabolites of vitamin D generated from CYP11A1 and are involved in

gene suppression influencing both immune and metabolic pathways (Jeon & Shin, 2018). These RORs have been linked to non-communicable diseases such as cancer and metabolic syndrome (Cook et al., 2015).

Feedback from both the liver and the kidney control vitamin D concentrations in the body. Both calcidiol and calcitriol are regulated primarily by the inactivating enzyme 25(OH)D 24-hydroxylase (CYP24A1) through negative feedback loops. CYP24A1 causes a hydroxylation reaction rendering biologically inactive calcitroic acid which is excreted through the bile system (Jeon & Shin, 2018). Vitamin D metabolism is also regulated by both parathyroid hormone (PTH) and fibroblast growth factor-23 (FGF-23). PTH is secreted by the parathyroid gland when serum calcium is low, increasing calcitriol production which in turn enhances dietary calcium absorption, promotes reabsorption of calcium in the kidneys and the release of calcium stores from bone. High calcium concentrations negatively feedback to reduce PTH secretion and therefore calcitriol production. Both osteoblast and osteocytes secret FGF-23 in response to elevated calcitriol, inhibiting CYP27B1 expression and therefore reducing calcitriol production.

As it is a lipid-soluble molecule, vitamin D can be taken up by adipose and other tissues and stored for later use (Martinaityte et al., 2017). It is thought to be slowly released from tissues during times of limited vitamin D synthesis, for example during the winter months when UVB exposure may decrease (Martinaityte et al., 2017).

As discussed vitamin D metabolism involves several complex pathways with the final active metabolite able to influences numerous processes upstream. Furthermore, it can be stored potentially to mitigate periods of scarcity. This information points to the importance of this nutrient in the body. However, with only small amounts being obtained from diet and potentially from foods not palatable to children, and with sun safe behaviours encouraged for children, this population may be at risk of deficiency. Therefore, understanding bodily status, especially in children provides a tool to both assess and potentially influence health outcomes.

## *2.5 Measuring UVB exposure, vitamin D status, cutoffs and deficiency*

### *2.5.1 Measuring UVB exposure*

Personal exposure to UVB can be measured using dosimetry (Sun et al., 2014). The dosimeter is a device that can be worn by an individual so that it is exposed to the same amount of UVB radiation as that individual's skin. It has sensors with a spectral response that mimics the skin's tendency to turn red (erythema) when exposed to certain wavelengths of light, this is called the erythemal action spectrum ($S_{er}$) of the skin (Heepenstrick et al., 2022). An individual's sensitivity to UVB radiation can vary depending on several factors including the amount and type of melanin in their skin (Yardman-

Frank & Fisher, 2021). Further, children's skin is more sensitive than adults due to its thinner dermis and functional immaturity (Volkmer & Greinert, 2011). Minimal Erythemal Dose (MED) is defined as the smallest amount of UVB radiation needed to induce reddening in the skin of an individual's previously unexposed skin (Salvadori et al., 2019). Full body exposure to UVB of MED for approximately 20 minutes is considered able to produce up to 250µg vitamin D (10,000IU) (Mostafa & Hegazy, 2015). Standard Erythemal Dose (SED) is a parameter used to describe the UVB radiation dose an individual has experienced. It mitigates for individual differences as it is an erythemally-weighted dose equal for all skin sensitivities (McKenzie et al., 2014).

*2.5.2 Measuring Vitamin D*

Vitamin D is routinely measured through both serum and plasma concentrations of 25(OH)D, however serum 25(OH)D is considered superior (Amrein et al., 2020). This is the main vitamin D transport metabolite and reflects vitamin D obtained both exogenously and endogenously (Institute of Medicine Committee to Review Dietary Reference Intakes for Vitamin D and Calcium, 2011). S-25(OH)D has a half-life of approximately 15 days and circulates between concentrations of 25-200nmol/L (Jones, 2008). This makes it more useful as a biomarker than the active hormone $1,25(OH)_2D$ which only has a half-life of approximately 15 hours, is tightly regulated by concentrations of PTH, calcium and phosphate, and often only changes in severe vitamin D deficiency (Jones, 2008). Although useful as a biomarker of vitamin D status, s-25 (OH)D does have limitations, firstly that single samples values can show large variations, depending on the assay and laboratory measuring the sample (Harvey et al., 2024). Further, there are limitations in its ability to show biological effects, and observational research routinely only studies correlations with s-25(OH)D concentrations which does not allow for conclusions on causation.

*2.5.3 Vitamin D status, cutoffs and deficiency*

There is no global consensus for vitamin D deficiency and sufficiency ranges (Dai et al., 2021). In New Zealand the consensus at present suggest vitamin D sufficiency as s-25(OH)D concentrations of >50 nmol/L, insufficiency as 25 - 50 nmol/L and deficiency as < 25 nmol/L (Ministry of Health, 2012a). These cutoffs have been reached through the consideration of vitamin D status' effects on biological processes like calcium absorption, PTH concentrations and bone mineralisation (Ministry of Health, 2012a). Vitamin D concentrations <50nmol/L are likely to cause hypocalcemia and hyperparathyroidism leading to unfavourable skeletal outcomes (Amrein et al., 2020; Holick, 2010). Concentrations below <30 nmol/L greatly increase mortality, infection and disease risk (Amrein et al., 2020). Deficiency however can be asymptomatic, allowing for potential damage to health without the individual being aware. The cutoffs are therefore open to debate, and some have suggested they may be too low. Research has suggested PTH is only stable when s-25(OH)D

concentrations are between 70-80nmol/L and PTH concentrations start increasing when s-25(OH)D falls below 78nmol/L , indicating sufficiency cutoff needing to be higher than the current New Zealand consensus (Chapuy et al., 1997; Dawson-Hughes et al., 2005). There are also no separate cutoffs for children which may be pertinent as their needs may be different due to their immature immune system and susceptibility to respiratory tract infections (Paul et al., 2014). Cutoff concentrations may be revised as science discovers more about the varying roles of vitamin D and how blood concentrations may affect these. Establishing universally accepted cutoffs would help with public health measures for screening and prevention of vitamin D deficiency (Antonucci et al., 2018).

*2.5.4 Global research on vitamin D status*

Vitamin D status as measured by s-25(OH)D concentrations vary greatly around the world (see Table 1) and also within countries due to the changing effects of different influential factors (Harvey et al., 2024). Different laboratory methods for measuring vitamin D often give substantially different 25(OH)D results, leading to inconsistencies in how deficiency and sufficiency are classified across countries. Historically, there has been little standardisation between assays, interference from other vitamin D metabolites, and varying cut-offs for deficiency, all of which have contributed to global differences in vitamin D status that may reflect methodological variation rather than true biological differences. However, these inconsistencies are now being rectified.

**Table One: To show prevalence of different vitamin D thresholds in varying global populations**

| Region | Country | Population Studied | Cut-off used (s-25(OH)D) | Prevalence | Notes / Trends | Reference |
|---|---|---|---|---|---|---|
| **Africa** | 28 countries | – | – | No data | Lack of serum vitamin D status data | Cashman et al., 2019 |
| | 23 countries (incl. Egypt, Nigeria, South Africa) | Mixed | <30 nmol/L | 17.3% | Higher deficiency in N. Africa, S. Africa, urban areas, women | Mogire et al., 2020 |
| | | | <50 nmol/L | 34.2% | | |
| **Central Asia** | Kazakhstan | General population | <25 nmol/L | 27.5% | Higher in females & Asians vs. Caucasians | Gromova et al., 2020 |
| | | | 25–50 nmol/L | 42.4% | | |
| **Middle East** | General | Mixed | <30 nmol/L | 30–90% | Very variable across countries | Cashman et al., 2019 |
| | West Bank & Gaza | Women of childbearing age | <27.5 nmol/L | Up to 70% | High risk group | Cashman et al., 2019 |
| | Yemen | Girls | <30 nmol/L | 61% | | Cashman et al., 2019 |
| | Syria | Adults | <25 nmol/L | 58.2% | | Cashman et al., 2019 |
| | Iran | General population | <25 nmol/L | 26.6% (summer), 63.0% (winter) | Strong seasonality | Nikooyeh et al., 2016; 2021 |

| Region | Country | Population Studied | Cut-off used (s-25(OH)D) | Prevalence | Notes / Trends | Reference |
|---|---|---|---|---|---|---|
| **Middle East (cont)** | Iran | Children | <25 nmol/L | 56% (winter) | | Nikooyeh et al., 2021 |
| **East Asia** | Mongolia | Pregnant women | <50 nmol/L | 75.4% | National Nutrition Survey 2016–17 | Health, 2017 |
| | | Infants | <50 nmol/L | 61% | | |
| | South Korea | Females | <50 nmol/L | 68.2% → 82.5% (2008–2014) | Deteriorating status | Park et al., 2018 |
| | | Males | <50 nmol/L | 51.8% → 75.2% (2008–2014) | | |
| | China | Children 6–17 yrs | <50 nmol/L | 53.2% | National Nutrition & Health Survey | Hu et al., 2017 |
| | | Children 6–11 yrs | <25 nmol/L | 5.6% | | |
| | | Children 12–14 yrs | <25 nmol/L | 8.8% | | |
| **South Asia** | Pakistan, Afghanistan, India | General population | – | High prevalence ("hot spots") | | Cashman, 2022 |
| | Nepal | Infants | <30 nmol/L | 0.6% | | Cashman, 2022 |
| | | Women (pregnant/lactating + children) | <30 nmol/L | 14% | | |
| | Bangladesh | Infants | <30 nmol/L | 6% | | Cashman, 2022 |
| | | Non-pregnant women | <30 nmol/L | 12–38.9% | | |

| Region | Country | Population Studied | Cut-off used (s-25(OH)D) | Prevalence | Notes / Trends | Reference |
|---|---|---|---|---|---|---|
| **South Asia (cont)** | Sri Lanka, Indonesia, Thailand, Cambodia, Philippines | General population | – | Low prevalence | | Cashman et al., 2019 |
| | Vietnam | Children | – | 11.2–20.6% | | Cashman et al., 2019 |
| | Malaysia | Children | – | 47.5% | | Poh et al., 2013 |
| **Americas** | USA | General population | <30 nmol/L | 5% | Higher risk in non-white (3–5×) | Cashman, 2022 |
| | | Adolescents 12–19 yrs | <30 nmol/L | ~5% | | |
| | Canada | General population | <30 nmol/L | 8.8% | Higher risk in non-white (3–5×) | Cashman, 2022 |
| | Mexico | School children 5–11 yrs | <50 nmol/L | 36.6% | | Cashman, 2022 |
| | Central America (other) | Children | <50 nmol/L | 0–11.5% | Data only from capital cities, not national | Robinson et al., 2017 |
| | Argentina | General population | 25–50 nmol/L | 15–55% | No national data | Cashman, 2022 |
| | Brazil | Children | 25–50 nmol/L | 9–10% | | Cashman, 2022 |
| | | Adults | 25–50 nmol/L | 2–16% | | |
| | Ecuador | General population | <40 nmol/L | 9–19% | | Cashman, 2022 |
| | Chile | Adults | <30 nmol/L | 16.4% | | Cashman, 2022 |
| | | School children | <30 nmol/L | 2.4% | | |

| Region | Country | Population Studied | Cut-off used (s-25(OH)D) | Prevalence | Notes / Trends | Reference |
|---|---|---|---|---|---|---|
| **Americas (cont)** | Chile | Adolescents | <30 nmol/L | 3% | | Cashman, 2022 |
| **Europe** | UK, Ireland, Germany (pooled) | General population | <30 nmol/L | 13% | Higher risk in adolescents (15–18 yrs) & dark-skinned ethnic groups. No sex differences. | Cashman, 2022 |
| | | | <50 nmol/L | 40.4% | Variation: some countries as low as 1% <30 nmol/L | |
| **Oceania** | Australia | General population | <30 nmol/L | 4.5% (~0.8M people) | | Cashman, 2022 |
| | | | <50 nmol/L | 20.1% (~3.3M people) | | |
| | | Aboriginal & Torres Strait Islanders | <30 nmol/L | 4.7% | Higher prevalence than general population | Cashman, 2022 |
| | | | <50 nmol/L | 27% | | |
| | | Adolescents (12–17 yrs) | <50 nmol/L | 17% | | Cashman, 2022 |
| | Fiji | Women of childbearing age | <50 nmol/L | 11% | National Nutrition Survey 2004 | Heere, 2010 |
| | Other Oceania countries | – | – | No data | | – |

As has been highlighted in this summary of global vitamin D status, there is large variation in status, however, data is lacking for many countries across the world and in turn across many ethnicities (Harvey et al., 2024). Therefore, although insightful and helpful in understanding the global landscape of vitamin D these findings should be interpreted with caution. It does seem however that many children globally are experiencing insufficiency or deficiency in vitamin D which may be having an impact on the health of this population.

*2.5.5 Vitamin D Status in the New Zealand adults and children*

Recent New Zealand population data for vitamin D status is limited. The Adult Nutrition Survey taken in 2008/09 showed 4.9% of the population were considered deficient with a blood concentration of <25nmol/L, of which 0.2% were classified as severely deficient (Ministry of Health, 2012b). Another 27.1% were considered insufficient with a blood concentration of between 25-50nmol/L which is below recommendations. Vitamin D deficiency was also more prevalent in Māori and Pacific People at 6% and 10% respectively (Ministry of Health, 2012b). Vitamin D status in the South Asian population is also known to be low with one New Zealand study showing only 16% of participants being sufficient (>50nmol/L) and another finding 72% of participants had a status considered insufficient (Scragg, 2020; von Hurst et al., 2010).

The 2002 National Children's Nutrition Survey showed that 31% of children aged 5–14 years had 25(OH)D concentrations below 37.5 nmol/L and 4% had 25(OH)D concentrations less than 17.5 nmol/L (Rockell et al., 2005). Further, risk of deficiency was seven times higher if vitamin D status had been collected in the winter rather than the summer. Prevalence of vitamin D deficiency also varied with ethnicity and was found to be 24% for Pacific, 11% for Māori, 3% for European and 16% for other ethnic groups (Rockell et al., 2005). A study in newborns found only 27% had 25(OH)D concentrations ≥75 nmol/L which is correlated with optimal health (Camargo et al., 2010). The strongest determinants of low vitamin D status for this population were being born in the winter and being of non-European ethnicity. In young children <2yrs vitamin D deficiency was found to be 13% and more prevalent in Pacific ethnicity (Grant et al., 2009). In children (N=1329) aged 2-4 years looking at vitamin D status assessed from late winter to early spring, 7% had vitamin D < 25 nmol/L and 48% had vitamin D < 50 nmol/L , only 11% had 25(OH)D concentrations of ≥ 75nmol/L (Cairncross et al., 2017). Further research on primary school-aged children (N=507) in Auckland found 28% of this population had 25(OH)D < 50 nmol/L (Delshad et al., 2019). Further, annual incidence of rickets in children aged <15 years due to vitamin D deficiency was found to be 2.2/100,000 suggesting deficiency of vitamin D is a persistent issue for some New Zealand children (Wheeler et al., 2015). There is, however, still a lack of research on vitamin D status, the effect of

seasonal variation and in at-risk populations in New Zealand school children to really understand how prevalent this issue is.

## *2.6 Influences on vitamin D production in the skin*

There are numerous factors that affect the production of vitamin D in the skin and influence a person's risk of deficiency, for example latitude, season, hemisphere, time of day, ethnicity, age, sex, body composition, socioeconomic status, cultural beliefs and behaviours all impact the UVB radiation a person may be both exposed to and be able to utilise to endogenously produce vitamin D.

### *2.6.1 Latitude*

As a person moves further away from the equator and the latitude increases their UVB dose decreases (Godar, 2005). At 70$^{o}$ latitude cutaneous vitamin D synthesis can be missing for 5 months of the year and even at a latitude of 51$^{o}$ the UVB radiation may not be strong enough to allow vitamin D production in the skin at certain times of year (Engelsen et al., 2005). New Zealand lies in the southern hemisphere and spans latitudes of 34$^{o}$S to 47$^{o}$S (Macara, 2018). Research exploring the effect of latitude on vitamin D in New Zealand has been mixed. Rickets has been found to be most prevalent in the South Island which has the highest latitude and therefore has the least potential UVB exposure (Wheeler et al., 2015). The most recent Adult Nutrition Survey found vitamin D status as measured by annual mean concentration of 25(OH)D was significantly lower for those living in both southern (60.5nmol/L) and central regions (62.6nmol/L) compared to those in the north of the country (65.1nmol/L) (Ministry of Health, 2012b). There was also a higher concentration of vitamin D deficiency seen for those in southern regions over the winter months (Ministry of Health, 2012b). Studies examining vitamin D status of children in differing parts of the country have found in Dunedin (45$^{o}$S) a mean 25(OH)D concentration of 52.3nmol/L, whereas in Auckland (36$^{o}$S) it was 55nmol/L (Grant et al., 2009; Houghton, 2010). However, data from the 2002 National Children's Nutrition Survey and more recent research by Cairncross and colleagues found no difference in vitamin D status of those living in different latitudes (Cairncross et al., 2017; Rockell et al., 2005).

### *2.6.2 Season, hemisphere and time of day*

The solar zenith (the angle of the sun or how high it is in the sky) has a large impact on the amount of UVB radiation reaching the surface of the earth (Godar, 2005). This angle changes depending on the season, hemisphere and the time of day, the smaller the angle, the higher the sun is in the sky, the more UVB reaches the earth. The UVB is most intense between 11am and 1pm and during the summer months in the southern hemisphere, when the sun is closer to the earth than it is in the northern hemisphere in the same season (Godar, 2005). At mid-latitudes (~45$^{o}$) daily UVB dose in

summer can be similar to the dose in the tropics, however in winter the dose is less than 10% of the summer dose (McKenzie et al., 2009). Daily vitamin D production has been found to vary from summer to winter by 10 times in Auckland and 20 times in Invercargill (Grant et al., 2009). The UVB radiation change from summer to winter is also affected by ozone and pollution concentrations, these can interfere with UVB radiation moving through the atmosphere and decrease its transmission as they increase. The southern hemisphere has both lower ozone and pollution concentrations than the northern hemisphere allowing more UVB to penetrate especially in the summer, in winter increased cloud cover inhibits UVB (McKenzie et al., 2009). Several studies have shown vitamin D status is lower in both adults and children during the winter months compared to the summer (Houghton, 2010; Rockell et al., 2005)

*2.6.3 Ethnicity and skin colour*

Global data shows ethnicity to be strongly associated with vitamin D status, for example a study in otherwise healthy adolescents in Boston showed those of African American descent to have the highest prevalence of deficiency, followed by Hispanic ethnicity, then Asian ethnicity and those of White ethnicity were least likely (Gordon et al., 2004). A further study in Sweden found children of non-Swedish ethnicity including Indian, Middle Eastern and African, also had a higher prevalence of vitamin D deficiency than those of Swedish ethnicity (Trollfors, 2022).

New Zealand is home to a mix of ethnicities with the 2023 census showing the ethnic mix to be predominantly European ethnicities (67.8%), followed by Māori (17.8%), Asian (17.3%), Pacific Peoples (8.9%), Middle Eastern, Latin and African (1.9%) (Statistics New Zealand, 2023). The relationship between ethnicity and vitamin D status has also been demonstrated within the New Zealand population with the New Zealand National Nutrition Survey (2008/09) data showing increased prevalence of deficiency in Pacific People, Māori and South Asian women (Ministry of Health, 2012b; von Hurst et al., 2010).

Ethnicity is thought to impact vitamin D status through a number of means, including ethnic differences in genetics influencing vitamin D absorption, chylomicron production, vitamin D metabolism and the quantity and dominant isoform of VDBP in circulation (Abdul-jabbar et al., 2024). Ethnicity also influences phenotype including differences in skin colour with melanin, the pigment that gives colour to skin, found to interfere with cutaneous vitamin D production and a systematic review concluding having pigmented skin decreases both the effectiveness of vitamin D production and concentration of s-25(OH)D (Gozdzik et al., 2008; Xiang et al., 2015). Individuals with dark skin may need to spend 5-10 times longer in the sun to synthesise the equivalent vitamin D of an individual with light skin (Chen et al., 2007). Furthermore, behaviour including dietary intake of

vitamin D containing food and cultural practices regarding skin exposure (which will be discussed in section 2.6.5) are also linked to ethnicity (Abdul-jabbar et al., 2024).

*2.6.4 Age, sex and body composition*

Vitamin D status is affected by changing factors across lifespan. Newborn vitamin D status is dependent on that of the mother, with low status during pregnancy resulting in low newborn status (Antonucci et al., 2018). Further, exclusive breastfeeding without adequate sun exposure can exacerbate low vitamin D status due to breastmilk's low vitamin D content (Dawodu & Tsang, 2012). Studies in children and adolescents indicate that younger children may have better vitamin D status than both older children and adolescents (Mansbach et al., 2009; Papandreou et al., 2010). This may be due to several reasons including younger children having less skin pigmentation (as skin darkens over time) making vitamin D synthesis easier or younger children having a smaller surface area and therefore needing less UVB exposure, or that older children and adolescents are spending more time indoors than their younger counterparts (Mansbach et al., 2009; Munns et al., 2006; Stein et al., 2006).

At the other end of life there is an age-related decline in vitamin D status due to several factors. The concentration of 7-dehydrocholesterol decreases in the skin and there is a reduction in response to UVB light, with research showing up to a 50% decrease in the skin's ability to produce previtamin D3 in participants aged 75yrs+ (MacLaughlin & Holick, 1985). Further, declining renal function with age impacts the kidney's ability to convert 25OHD to 1,25(OH)D (Gallagher, 2013). Lastly, substrate deficiency of 25OHD is a common yet preventable problem in older adults, with many not getting enough due to poor diet and limited time in sunlight (Gallagher, 2013).

There are potential sex-related differences in vitamin D status and this may be due to the ability of sex hormones (androgens and oestrogens) to influence vitamin D metabolism (Wierzbicka & Oczkowicz, 2022). However, the findings are mixed with some studies showing females to have lower status than males and some the opposite (AlQuaiz et al., 2018; Rockell et al., 2005; Sanghera et al., 2017; Wierzbicka & Oczkowicz, 2022). A study in children of different ethnic backgrounds found girls to have lower vitamin D status compared to boys but only in those children originating from countries where females are encouraged to cover skin for religious, cultural or aesthetic reasons, e.g. Pakistan and the Middle East. There was no status difference between boys and girls from European or Latin American ethnicities (Trollfors, 2022). A further study in healthy adolescents in America found no significant difference in prevalence of vitamin D deficiency between boys and girls (Gordon et al., 2004). A greater body fat percentage in women, allowing them to potentially store more vitamin D in adipose tissue may keep vitamin D status more stable and explain a larger

variability in vitamin D status in men over the course of a year (Wierzbicka & Oczkowicz, 2022). Sex-related disparities are most marked in people who are overweight or obese, especially children, with boys who are overweight and obese most burdened with low vitamin D status (de Oliveira et al., 2020; Turer et al., 2013).

Obesity is associated with lower vitamin D status (Karampela et al., 2021). In New Zealand the National Children Survey found children aged 5 to 14 years who were obese had decreased s-25(OH)D (Rockell et al., 2005). This may be due to numerous factors including individuals with obesity having lower sunlight exposure due to more sedentary lifestyles and vitamin D being less bioavailable due to it being sequestered and stored in the increased amount of adipose of tissue (Blum et al., 2008; Pourshahidi, 2015; Wortsman et al., 2000). Adipose tissue has also been connected to vitamin D metabolism and obesity associated with decreased expression of genes coding for the enzymes used in the metabolic pathway (Elkhwanky et al., 2020; Wamberg et al., 2013). Interestingly, there is little evidence to suggest weight loss improves vitamin D status (Karampela et al., 2021)

### *2.6.5 Socioeconomic status, cultural beliefs and behaviours*

Socioeconomic factors are associated with vitamin D status, this may be due to access and opportunity to be outside in the sunshine. The association is seen to vary between countries with children of lower socioeconomic status from Jordan seen to have higher vitamin D status than those in higher brackets, thought to be due to them having increased amounts of time outside to escape cramped surroundings indoors (Gharaibeh & Stoecker, 2009). In New Zealand the opposite is found with the National Nutrition Survey 2008/2009 finding those in the most deprived households, often in urban areas with little space for outdoor play, having three times the risk of vitamin D deficiency than those in the least (Ministry of Health, 2012b).

Cultural and behavioural differences are also seen to influence vitamin D status with a cross-sectional study in Saudi Arabia indicating women have a lower exposure to the sun than men as women cover more of their skin (Alharbi et al., 2018). However, they are also found to take vitamin D supplementation more often than men (Geddawy et al., 2020). A review found vitamin D deficiency to be more prevalent in women in Middle Eastern countries and in women from Middle Eastern countries living in Western countries and was associated with factors such as clothing choices, sedentary life-style and less education, as well as lower socioeconomic status (Bedewy et al., 2022).

Sun safe behaviours such as the use of suncream, coverage of the skin and staying out of the sun further influence vitamin D status. This messaging is especially targeted at children and encouraged in the school environment. New Zealand has some of the highest melanoma rates in the world, with melanoma ranked as the third most common cancer among the population and consequently there is strong sun safe messaging (Wen et al., 2022). Suncream with sun protection factor (SPF) of 30 and applied appropriately is thought to reduce their skin's ability to synthesise vitamin D by as much as 95-99% (Hossein-nezhad & Holick, 2013). Although, a recent review reported there was little evidence to suggest that suncream use decreases vitamin D status, however most of the research used only moderate SPF, around 16, and not the high protection sun block that is often recommended in New Zealand (Neale et al., 2019). Further, application of suncream such as amount used and how often it is applied may have influenced findings (Delshad, 2019).

## *2.7 Vitamin D and Health*

### *2.7.1 Functions of vitamin D in the body*

It has long been known that vitamin D plays a crucial role in bone health through its effects on calcium and phosphorus metabolism, two important molecules for bone mineralisation (Acar & Özkan, 2021). If calcium concentrations decrease in the blood, vitamin D works in three ways to increase them. Firstly, it increases intestinal calcium absorption through modulation of the proteins involved in active transportation of calcium from dietary intake (DeLuca, 2004; Fong & Khan, 2012). It also stimulates active absorption of phosphate in the intestine. Secondly, with PTH it stimulates osteoclasts to perform bone resorption, mobilising calcium from the bone into circulation (DeLuca, 2004). Lastly, and again with PTH, vitamin D stimulates increased reabsorption of calcium in the kidneys (DeLuca, 2004). Vitamin D is tightly linked to the parathyroid gland and plays a function in preventing hyperproliferation of parathyroid gland cells to maintain normal parathyroid status (Darwish & DeLuca, 1999).

In addition to skeletal health vitamin D has been found to play a role in many other bodily functions. Firstly, it acts to modulate the immune system, especially T-cell mediated immunity, with both deficiency and excess shown to impair immune function (Shouli Yang et al., 1993; SL Yang et al., 1993). It is also involved in teeth mineralisation through its effects on calcium, phosphate and magnesium, and deficiency can lead to significant alterations in dental-oral-craniofacial structures (Foster & Hujoel, 2018). Vitamin D is involved in skin homeostasis and both vitamin D and the VDR play a role in regulation of keratinocyte proliferation and differentiation (Bikle, 2018; Demay, 2018). Mutation of the VDR results in alopecia totalis (Demay, 2018). Vitamin D further serves to promote a

healthy vascular system and is involved in anti-inflammatory pathways as well as modulation of the renin-angiotensin system, cholesterol deposition and endothelial nitric oxide release (Riek et al., 2018). Vitamin D also acts in the brain where it has been found to cross the blood-brain barrier and influence glial cells. The VDR is found in central nervous system cells, with vitamin D considered a neurosteroid able to modulate the synthesis of both neurotransmitters and neurotrophic factors (Anjum et al., 2018). Lastly, it is thought to play a role in the regulation of skeletal muscle being one of a number of endocrine molecules which influence muscle differentiation, metabolism and contractile function (Girgis, 2018).

### *2.7.2 Vitamin D and health outcomes*

Hypovitaminosis D can cause numerous acute biochemical effects and clinical symptoms which are known to affect both skeletal and extra-skeletal parts of the body. Hypovitaminosis D is further associated with several more chronic effects and disorders. This section will give a brief overview of the health implications of sub-optimal vitamin D status.

Due to the close link between vitamin D and calcium metabolism, hypovitaminosis D is known to cause hypocalcaemia, where serum calcium decreases below 2.12mmol/L (Fong & Khan, 2012; Khazai et al., 2008). Symptoms include muscle spasms and cramps, cognitive impairment, arrhythmia, and ultimately it can lead to seizures and heart failure. Hypophosphatemia (serum phosphate <0.8mmol/L) can also occur due to the influence of vitamin D on phosphorus absorption in the small intestine and vitamin D deficiency causing an increase in parathyroid hormone (PTH) concentrations which in turn reduces phosphate reabsorption in the kidneys (Sarathi et al., 2024). Chronically these both cause hypomineralisation of bone and myopathy, i.e. bone and muscle pain and weakness. Skeletal abnormalities due to disruption of bone metabolism include weakening and softening of bone resulting in the disease rickets in children and adolescents and osteomalacia in adults. Vitamin D deficiency is also associated with other bone and muscular diseases such as osteoporosis and sarcopenia in older adults, and abnormalities including bowed legs, small stature, delayed fontanelle closure, frontal bossing and craniotabes in children (Acar & Özkan, 2021; Kupisz-Urbańska et al., 2021).

Vitamin D is seen to play a role in the fight against respiratory tract infections caused by airborne pathogens. These are especially pertinent to children being a major cause of hospitalisation and mortality in this population group (Telfar Barnard & Zhang, 2021). Respiratory tract epithelium are the first line of defence, these cells are known to express the VDR on their surface and produce the enzyme 1-α-hydroxylase which converts vitamin D into its active form (Raju et al., 2022). Vitamin D

produced in these cells causes cell proliferation and inhibits cell apoptosis during periods of inflammation, it also induces gene expression of protein receptors on the cell surface that recognise pathogens and stimulates antimicrobial peptides in immune cells (Bikle, 2022; White, 2022; Youssef et al., 2011). It is postulated that the increase in respiratory infections seen during the winter months may be due to a reduction in UVB exposure and a subsequent decrease in vitamin D production in the skin (McNally et al., 2009). Vitamin D supplementation has been found to reduce risk, although the reduction was minimal, of respiratory infection (OR=0·92, 95% CI 0·86–0·99) (Jolliffe et al., 2021). However, the protective effects of daily or weekly supplementation were higher in those with a low 25(OH)D baseline (<25nmol/l) (Martineau, 2019)

Both the VDR and 1-α-hydroxylase enzyme are present in several immune cells and vitamin D influences both the innate and adaptive immune systems (Sîrbe et al., 2022). It has been shown to cause gene expression driving antibacterial and antiviral responses from the innate immune system and is involved in growth and differentiation of T and B lymphocytes, macrophages and dendritic cells (Fletcher et al., 2022; Sîrbe et al., 2022). This influence on the immune system has led to the idea that vitamin D deficiency may cause dysregulation in both the innate and adaptive immunity pathways and potentially influence autoimmune diseases such as inflammatory bowel disease, rheumatoid arthritis, multiple sclerosis and type 1 diabetes. This may be particularly relevant to children who have an immature immune system and are potentially at increased risk for some autoimmune conditions such as type one diabetes (Diaz-Valencia et al., 2015). Several reviews have concluded there is an association between vitamin D and the onset and progression of autoimmune conditions and that low serum vitamin D is associated with increased prevalence and severity of autoimmune diseases but it is not yet known whether deficiency brings about the disease or is a symptom of it (Ao et al., 2021; Fletcher et al., 2022; Sîrbe et al., 2022).

The link between vitamin D and brain health is through its neuromodulatory effects. It is thought to reduce dementia risk through regulating pathways that protect against neurodegeneration such as plaque deposition, neurofibrillary degeneration, excess calcium influx in neurons and oxidative stress (Panza et al., 2021). It may also play a role in Parkinson's disease, with vitamin D status associated with disease progression and severity and motor-symptoms such as fall risk, however there is little evidence regarding the benefits of supplementation (Pignolo et al., 2022). Although perhaps not obviously relevant in children, if vitamin D affects brain conditions such as Parkinsons, it could be postulated that a child's maturing brain may also be affected by low vitamin D and insufficiency may affect a child reaching their full potential. Vitamin D is further found to influence mental health and disorders such as depression and bipolar disorder, which are thought to be brought about by neuroinflammation, through inflammation reduction both in the brain directly through

neuromodulatory, anti-inflammatory, antioxidant and proneurogenic affects but also in the intestine through modulation of gut bacteria to reduce activation of inflammatory molecules (Grant et al., 2022; Luthold et al., 2017). These disorders are increasing in childhood populations (Baranne & Falissard, 2018; Zubrick et al., 2000). A systematic review in childhood populations concluding that vitamin D status should be maintained at sufficient concentrations to prevent and alleviate mental health issues (Głąbska et al., 2021)

Several associations between low vitamin D status and issues during pregnancy and early infant life have been found. These include preeclampsia, gestational diabetes, preterm birth, caesarean delivery, low birth weight, foetal skeletal growth, infant respiratory issues and brain development (Grant et al., 2022; Zhang et al., 2022). Low vitamin D status in early life may lead to a vicious cycle of becoming vitamin D deficient mothers, leading to further infants who are deficient and never able to fulfil their potential. Vitamin D supplementation has been seen to improve outcomes for both mothers and infants and this has led to advice on vitamin D supplementation especially for those at risk of deficiency and for exclusively breastfed babies (Health New Zealand, 2024; Tan et al., 2020).

Vitamin D is associated with many cardiovascular disease (CVD) risk factors through influences on inflammatory pathways, including the production of reactive oxygen species, increased blood pressure through the renin-angiotensin system and the development of insulin resistance leading to metabolic syndrome (Kheiri et al., 2018; Rammos et al., 2008). It can also directly affect calcification and proliferation of smooth muscle influencing cardiovascular health and improved vitamin D status is associated with both improved lipid profile and reduction in metabolic syndrome occurrence and low status with increased incidence of CVD (Heston, 2010; Kendrick et al., 2009; Skaaby et al., 2012). Although CVD usually affects older adults, contributing factors start long before adulthood and build towards the disease state. The research on using supplementation to improve CVD and ameliorate risk is contradictory and more research is needed to elucidate whether it is of benefit (Kheiri et al., 2018). Dyslipidaemia and insulin resistance are also common symptoms of polycystic ovary syndrome (PCOS), a disorder on the rise in adolescent populations, and several studies have found an association between low vitamin D status and onset and severity of PCOS, as well as symptom alleviation with supplementation (Franks, 2008; Mohan et al., 2023).

Vitamin D status has also been linked to skin disorders such as eczema, psoriasis and vitiligo (Formisano et al., 2023; Palmer, 2015; Varikasuvu et al., 2021). Eczema is often a problem for children and can be detrimental to quality of life. This is thought to be due to the influence of vitamin D on both the immune system and skin cells such as keratinocytes and fibroblasts (Rebelos et al., 2023; Zeng et al., 2023). Further, lower vitamin D status in mothers and infants has been found to increase the incidence of food allergies, a disorder on the rise in childhood populations

(Devdas et al., 2018; Psaroulaki et al., 2023). This may be due to vitamin D's role in immune system function and it helping maintain integrity of the mucosal barrier in the intestine reducing allergen exposure (Di & Chen, 2021).

Vitamin D research has found antiproliferative effects on cancer cells through gene regulation, as well as inhibition of angiogenesis, induction of apoptosis, reduction of inflammation, repair of DNA and a decrease in metastasis (Bouillon et al., 2018). There is further a suggestion that vitamin D metabolism may be dysregulated in certain cancers (Jeon & Shin, 2018). The relationship is thought to be complicated with more evidence needed for many types of cancers, however a systemic review found good evidence for an inverse relationship between vitamin D status and colorectal and breast cancers (van der Rhee et al., 2013). There is evidence to suggest a frequency of low vitamin D status in child cancer patients and that higher vitamin D status is associated with better cancer outcomes, however the evidence for its association with reduced cancer risk is more inconsistent (Atkinson, 2008; Mondul et al., 2017).

Much of the research that has found associations between vitamin D and various diseases have been observational with randomised controlled trials (RCTs) having more mixed results. However, this may be due to several reasons including RCTs not always having vitamin D deficient participants, low doses of supplementation being used, evaluation of outcomes being by vitamin D dosage rather than serum vitamin D (animal models show vitamin D status is affected by metabolism, therefore s-25(OH)D does not simply mirror intake) and genetic factors that might influence supplement use in the body not being taken into consideration (Grant et al., 2022; Saponaro et al., 2020). The influence of vitamin D on so many diseases and disorders that affect not only adults but also children or have the potential to affect their long-term outcomes, make it an important area to research. Understanding the fundamentals of what status looks like in this population and what factors are affecting this is vital, so that status can be optimised and the likelihood of disease lessened.

## *2.8 Conclusion*

As discussed in this literature review, the role of vitamin D in the body, although still not fully elucidated, is considerable with it seemingly able to influence potentially every bodily function. Further, the importance of this issue to the health of children is evident. As we have seen an individual's status could have significant effects on both their acute and chronic health outcomes. Understanding status in the New Zealand childhood population, will give more insight into status throughout the lifespan and will help paint a picture of where the New Zealand population currently stands. This will help to inform policy makers if more work is needed to improve vitamin D status in childhood populations as a preventative measure against future health issues.

# Chapter 3: Researching Vitamin D Status and UVB Exposure in Auckland school children aged 11-12 years

**ABSTRACT**

Background: Vitamin D is primarily synthesised in the skin through UVB exposure, and concentrations fluctuate with seasonal changes in sunlight. New Zealand children may be particularly vulnerable to low vitamin D status in winter due to strong sun-safety practices, limited dietary sources, and marked seasonal variation in UVB availability.

Aims: The study aimed to determine seasonal variation in both serum 25-hydroxy-vitamin D (S-25(OH)D) status and UVB exposure measured in Standard Erythemal Dose (SED) in Auckland school children aged 11-12 years.

Method: Auckland intermediate school children (N=100), mean age 11±0.8 years, majority female (58%) and of European ethnicity (65%) were tested at two different seasonal timepoints, March (the beginning of autumn, after summer sun exposure) and September (the beginning of spring, after winter sun exposure). Two assessments were made, S-25(OH)D status using finger prick blood spots and UVB exposure measured over a 10-day period using dosimeters worn by the children.

Results: S-25(OH)D reduced from a mean of 90±22 nmol/L in summer to 65±18 nmol/L in winter, a mean percentage change of -26±17% ($P<0.00001$). Mean change was greater for those with a summer s-25(OH)D ≥75nmol/L or an elevated BF%. In summer 3% of participants had vitamin D deficiency and 21% were insufficient in vitamin D, whereas in winter, 20% were deficient and 50% insufficient. Participants with winter concentrations <50 nmol/L had an increased chance of having concentrations <75 nmol/L in summer, OR=3.6 (95%, CI 1.1-11). The mean UVB dose over a 10-day period for the children in summer was 9.2±5.0 SED decreasing to 3.1±1.1 SED in winter, this is a significant mean reduction of -6.1±28 SED ($P<0.0001$).

Conclusion: Vitamin D status in children significantly decreases from summer to winter in parallel with their exposure to UVB light across the change in seasons. The greatest percentage change was seen in those who had replete vitamin D in summer or had elevated body fat percentage. The results suggest vitamin D insufficiency is a potential problem for children in New Zealand. As low vitamin D status is associated with undesirable health outcomes, including respiratory diseases (a major contributor to childhood mortality) understanding status and influencing factors are very important to inform health policy.

**INTRODUCTION**

Vitamin D is an essential prohormone, primarily acquired through endogenous production in the skin. Its function in calcium homeostasis and bone health is well documented, with studies showing an association between vitamin D insufficiency and deficiency, and poor skeletal health (Chanchlani et al., 2020; Minisola et al., 2020; Moon et al., 2014). Rickets, a disease in children causing skeletal abnormalities and growth disturbances, and considered a problem both globally and in New Zealand, is known to have vitamin D deficiency as a major contributing factor (Chanchlani et al., 2020; Palacios & Gonzalez, 2014; Siafarikas et al., 2020; Wheeler et al., 2015). Further, the vitamin D receptor is expressed in most bodily tissues, indicating this nutrient is involved in numerous bodily processes in addition to bone health. Associations between low vitamin D status and several childhood disorders have been noted, including respiratory disease, autoimmune conditions such as type one diabetes, allergies, eczema, and autism spectrum disorder (Lucas et al., 2008; Mazahery et al., 2016; Shah et al., 2024; Zeng et al., 2023). These conditions are an issue for New Zealand youth with many increasing in prevalence among childhood and adolescent populations and respiratory diseases in particular being a large contributor to childhood mortality (Gomber, 2022; Te Rōpū Arotake Auau Mate o te Hunga Tamariki, 2021).

Considering the importance of vitamin D in health and especially children's health, there are still gaps in our understanding of the prevalence of insufficiency and deficiency in the New Zealand young population. Studies looking at young children have found deficiency (<30nmol/L) to be at around 13% for children under two years old and 7% for children between two and four years old (Cairncross et al., 2017; Grant et al., 2009). Insufficiency has been found to be even more prevalent at 48% in two to four year olds and 28% in children five to eleven years (Cairncross et al., 2017; Delshad, 2019). Studies in vitamin D status in school-aged children are lacking, however annual incidence of rickets in children aged below fifteen years due to vitamin D deficiency was found to be 2.2/100,000 suggesting deficiency of vitamin D is a persistent issue for some New Zealand children (Wheeler et al., 2015). Further, the 2002 National Children's Nutrition Survey showed over a third of five to fourteen year olds were vitamin D insufficient or deficient (Rockell et al., 2005). Understanding status in the New Zealand adolescent population would help determine whether this is a major problem in public health for this age group.

Numerous factors influence vitamin D status including ethnicity, where Māori, Pacific People and South Asian populations have been found to be more at risk of deficiency (Ministry of Health, 2012b; Scragg, 2020; von Hurst et al., 2010). Season is another major influence on status with risk of

deficiency increasing seven-fold in the winter compared to summer (Rockell et al., 2005). Sex has been shown as a factor with mixed results, some studies show females have a lower status than males and some the reverse (AlQuaiz et al., 2018; Rockell et al., 2005; Sanghera et al., 2017; Wierzbicka & Oczkowicz, 2022). Older children have been found to be more deficient than younger (Mansbach et al., 2009; Papandreou et al., 2010). Skin colour can affect endogenous vitamin D metabolism and therefore affect status (Chen et al., 2007; Gozdzik et al., 2008; Xiang et al., 2015). Body composition may further have an effect with reduced concentrations of 25-hydroxyvitamin D found in children with obesity (Rockell et al., 2005). Lastly, sun protective behaviours and supplementation affect status (Brett, 2018; Delshad, 2019). Knowledge on how these factors are influencing the school-aged population would allow for more targeted approaches to interventions for this age group to improve vitamin D status should this be needed.

The study aimed to determine seasonal variation in both serum 25-hydroxy-vitamin D (S-25(OH)D) status and UVB exposure measured in Standard Erythemal Dose (SED) in Auckland school children aged 11-12 years. Further, it explored factors that might be influencing status.

**METHODS**

The DISC (Vitamin **D** Status in **I**ntermediate **S**chool **C**hildren) research used a longitudinal study with repeated measures design to examine 25-hydroxy-vitamin D (25(OH)D) status and UVB exposure in intermediate school children (aged 11-12 years) at two time points. The first timepoint was in March (the beginning of autumn, after summer sun exposure) and the second timepoint was September (the beginning of spring, after winter sun exposure). Data was collected from 109 students in year 7 and year 8 at Birkdale Intermediate School, Auckland, New Zealand. Participants were recruited using convenience sampling from a single school in Birkdale through an existing school contact. All students meeting the predefined inclusion criteria (e.g., age range and enrolment at the school) were invited to participate. Participation was voluntary, and students chose whether to take part. Exclusion criteria included children with a bleeding disorder. Individuals with metal in their body were exclude from the BIA assessment only. 100 students' data was analysed due to seven students having missing data from both seasons and two students having missing data from one season. Demographic data collection included sex, self-reported skin colour and use of suncream and hats. Anthropometric data collection included height, weight and body composition. This was measured through bioelectrical impedance analysis (BIA) using the InBody 230 analyser machine from Biospace Co. Ltd., Seoul. BIA has been validated against both air displacement plethysmography and dual-energy X-ray absorptiometry (von Hurst et al., 2016). 25(OH)D status was measured in all

participants using finger prick blood samples and dried blood spot analysis. A lancet was used to produce blood from the finger, drops of blood were then applied to the filter paper card, the blood was allowed to absorb and dry and was sent to Canterbury Health Laboratory for analysis. Finally, individual sun exposure was measure by UVB dosimetry. UVB dosimeters were worn on the wrist by 58 participants (restricted to the number of available dosimeters) during the school day for a period of 10-days during both time points.

Statistical analyses were performed using IBM SPSS software version 27 (IBM Corporation, New York, USA). The data was assessed for normality using the Kolmogrov-Smirnov test and visual inspection of histograms for normal distribution. Descriptive statistics were used to summarise participants' demographic, lifestyle and anthropometric characteristics. Continuous variables are presented as mean ± standard deviation (SD) if normally distributed, or as median and interquartile range (25th, 75th percentiles) if not normally distributed. Categorical variables are presented as counts (n) and percentages (%). Body fat percentage was categorised relative to sex-specific cut-offs (25% for boys and 30% for girls) (Williams et al., 1992). Statistical significance was set at $p<0.05$ for all analyses.

Seasonal differences in serum 25-hydroxyvitamin D [s-25(OH)D] concentrations and UVB dose after summer exposure and winter exposure were assessed using paired samples t-test. Results are presented as mean ± standard deviation (SD) with 95% confidence intervals (CI). Statistical significance was set at $p<0.05$.

The risk of winter vitamin D deficiency (s-25(OH)D <50 nmol/L) was assessed using logistic regression, with summer s-25(OH)D (<75 vs. ≥75 nmol/L) as the primary predictor and body fat percentage, sex, ethnicity, and skin colour included as covariates. Results are reported as adjusted odds ratios (OR) with 95% confidence intervals (CI).

To examine predictors of seasonal change in serum 25-hydroxyvitamin D [s-25(OH)D] concentration, a multiple linear regression analysis was performed. The dependent variable was the change in s-25(OH)D from summer to winter. Independent variables included self-reported skin colour, sex, reported use of sunscreen during winter months, summer s-25(OH)D concentration, and body fat percentage (BF%). All predictors were entered into the model simultaneously to assess the independent contribution of each variable while controlling for the others.

**RESULTS**

S-25(OH)D concentration for both seasons, summer and winter, was available for 100 children out of the 109 initial recruits (both seasons missing 7, one season missing 2). Participant characteristics are

presented in **Table 2**. A larger proportion of children were New Zealand European (66%), had fair/medium skin colour (70%), reported not using any dietary supplements (88%), and had BF% within the normal range (82%). While a large proportion of children reported using sunscreen during summer months (66%), only 5% used sunscreen during winter months.

**Table 2:** Participant characteristics

| Participant characteristics | **N=100** |
|---|---|
| Age, years, mean±SD | 11±0.8 |
| Sex, n (%) | |
| Girls | 58 (58) |
| Boys | 42 (42) |
| Ethnicity, n (%) | |
| European | 65 (66) |
| Māori | 9 (9) |
| Pacific | 9 (9) |
| Asians | 8 (8) |
| Others[†] | 7 (8) |
| Skin colour, n (%) | |
| Fair | 38 (38) |
| Medium | 32 (32) |
| Olive | 19 (19) |
| Dark/Brown | 8 (11) |
| Using sunscreen, n (%) | |
| During summer | 66 (67) |
| During winter | 5 (5) |
| Taking vitamin D containing supplement, n (%) | 4 (4) |
| Anthropometrics/body composition | |
| Weight, kg, mean±SD | 151±8.4 |
| Height, cm, mean±SD | 43±10 |
| BMI, kg/m$^2$, median (25$^{th}$, 75$^{th}$ percentiles) | 18 (17, 20) |
| Body fat percentage, %, median (25$^{th}$, 75$^{th}$ percentiles)[*] | 19 (14, 24) |
| <sex-specific cutoff | 82 (82) |
| >sex-specific cutoff | 17 (17) |

BMI, body mass index; SD, standard deviation
[†]Others included the following ethnicities: Latin Americans (3), New Zealander (n=1), Indian African (n=1) African (n=2).
[*]A cut-off of 25% for boys and 30% for girls

The Mean±SD s-25(OH)D concentration in summer was 90±22 nmol/L, which decreased to 65±18 nmol/L in winter (a mean change of -25±18 nmol/L and mean percentage change of -26±17%, P<0.00001) (**Figure 1**). In summer, 3% of children had vitamin D deficiency (25(OH)D <50 nmol/L), 21 % had insufficiency (25(OH)D <75 nmol/L) and 76% were sufficient (25(OH)D ≥75 nmol/L). Vitamin D deficiency, insufficiency and sufficiency were seen in 20%, 50%, and 30% of children in winter,

respectively. The odds of having winter s-25(OH)D concentration <50 nmol/L were increased for children with summer s-25(OH)D <75 vs. ≥75 nmol/L, 33% vs. 16%, OR=3.6 (adjusted for potential confounders), 95% CI 1.1–11.

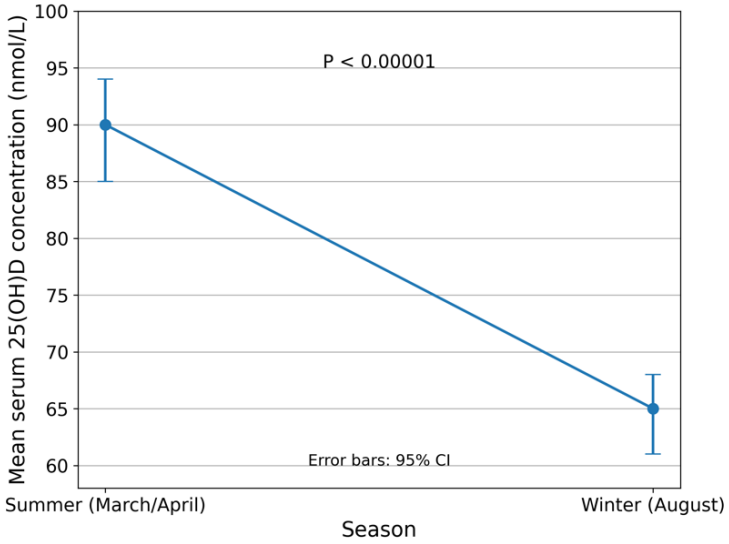



**Figure 1:** Seasonal change in s-25(OH)D concentration.

The mean change in s-25(OHD concentration (from summer to winter) did not differ between girls and boys, ethnic groups, and children with different skin colours. However, the mean change in s-25(OH)D concentration was greater in children with summer s-25(OH)D ≥75 (n=76) than <75 nmol/L (n=24) [-30±17 vs. -9.1±11 nmol/L, P<0.001], and in children with elevated BF% (n=17) than normal BF% (n=82) [-29±15 vs. -24±19 nmol/L, P=0.04 , adjusted for summer s-25(OH)D]. When all potential factors were held constant, summer s-25(OH)D and BF% were the only predictors of seasonal change. These variables accounted for 45% of the variance in the seasonal change in s-25(OH)D concentrations (**Table 3**).

**Table 3** Predictors of seasonal change in s-25(OH)D (from summer to winter)†

| Seasonal change in s-25(OH)D Model | Coefficient *(B)* | Standard Error *(B)* | 95% CI *B* | Standardised *B* | $R^2$ | *P*-value |
|---|---|---|---|---|---|---|
| **Model** | | | | | 0.45* | <0.001 |
| Intercept | 6.6 | 17 | -28, 41, 38 | | | |
| Summer s-25(OH)D** | -0.6 | 0.1 | -0.7, -0.4 | -0.7 | | <0.001 |
| BF%*** | -9.5 | 4.1 | -18, -1.5 | -0.2 | | 0.02 |

†Enter technique. The following variables were included in the model: skin colour, sex, using sunscreen during winter months, summer serum-25(OH)D, and body fat percentage (BF%).
Valid number of participants: 89
*F (5, 84)=14, P<0.001
**For each increase of one nmol/L in summer s-25(OH)D concentration, winter s-25(OH)D concentration is expected to decrease by 0.6 nmol/L.

***BF% was coded as 1=normal and 2=elevated; having elevated BF% was associated with a greater decrease of 9.5 nmol/L in winter s-25(OH)D concentration.
Regression equation: percentage change in s-25(OH)D (nmol/L)=6.6 – 0.6 x summer s-25(OH)D (nmol/L) – 9.5 x BF%

**Seasonal change in UVB dose**

The UVB dose for both seasons, summer and winter, was available for 44 children out of 58 who consented to wear UVB dosimeter (summer and winter UVB dose data were missing for six and eight children, respectively). During a 10-day period, the average UVB dose received by children in summer was 9.2±5.0 (average daily dose of 0.9±0.5 SED), which decreased to 3.1±1.1 (average daily dose of 0.3±0.1 SED) in winter (a mean decrease of -6.1±28 SED, P<0.0001) (**Figure 2**).

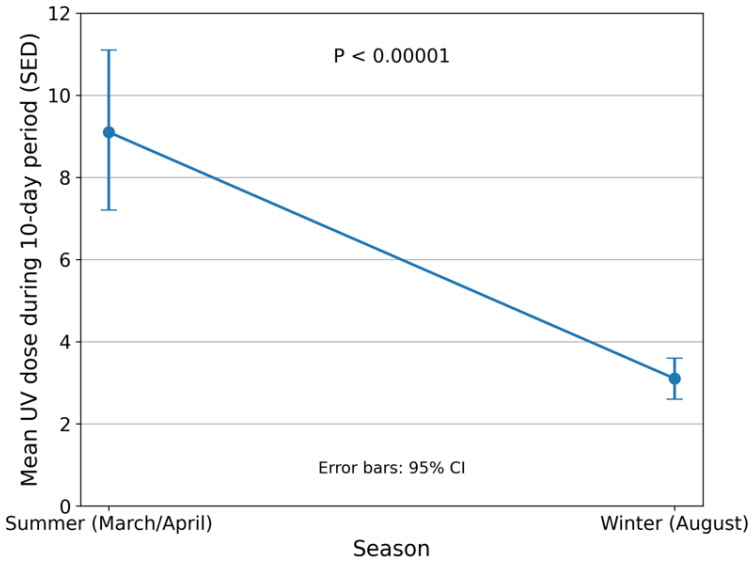



**Figure 2**: Seasonal change in UVB dose.

**DISCUSSION**

This study showed a significant seasonal change in s-25(OH)D concentration (mean decrease of 26% from summer to winter) in school-aged children in Auckland, New Zealand. Although smaller, the seasonal change in s-25(OH)D concentration reflects the seasonal change in UVB dose (more than 60% decrease from summer to winter). In addition, the number of children with vitamin insufficiency (s-25(OH)D concentration 50-75 nmol/L) increased from 21% in summer to 50% in winter. Greater seasonal (summer to winter) decrease in s-25(OH)D was seen in children with higher summer s-25(OH)D concentration and higher BF%.

The present study shows a relatively high prevalence of winter vitamin D insufficiency in this population of school aged children, a finding confirmed by other researchers from New Zealand (Delshad, 2019; Maryam Delshad, 2019). Given that 50% of children in the present study had 25(OH)D concentrations between 50 and 75 nmol/L, the cut-off used to define insufficiency will play a major role in determining what proportion of the children is considered to be insufficient. Although using the current cut-off of 50 nmol/L decreases the number of vitamin D insufficient children in winter (20%), our data suggest that there are some children who have insufficient sun exposure to maintain optimal 25(OH)D concentrations all year around (all seasons).

This study also showed a significant difference in the mean s-25(OH)D concentrations between two seasons; high in summer and low in winter, a finding confirmed by others. Studies from New Zealand reported similar significant seasonal differences of 24-35 nmol/L in infants (Grant et al., 2009; Houghton, 2010), 15 nmol/L in school age children (Rockell et al., 2005), and 10-20 nmol/L in adults (Bolland, 2012; Mazahery, 2015). Also, studies from Canada (7-15 nmol/L) (Yousef, 2021), Denmark (27 nmol/L) (Hansen, 2018), Russia (18-23 nmol/L) (Kondratyeva, 2020), Korea (20 nmol/L) (Kim, 2013) and Turkey (14-16 nmol/L) (Karagüzel, 2014) have shown significant seasonal variation in school age children. To the best of our knowledge, this study is the first to investigate the seasonal change in 25(OH)D concentration in New Zealand children.

The seasonal changes in 25(OH)D concentrations could be explained by three major factors; the decrease in the average daily hours of sunlight exposure, the average intensity of UVB exposure, and the percentage of total body surface area exposed outdoors. In line with these explanations, the present study showed a significant decrease of more than 60% in UVB dose in winter months. One might also postulate that children might engage in more physical activity during summer months (however this was not measured in this study), and this could result in an increase in the mobilisation of vitamin D from body's adipose tissue (Hengist, 2019). Lower concentrations of physical activity and elevated sedentary time has been reported during autumn and winter months compared to summer months in children, particularly due to weather conditions and day length (Atkin, 2016; Harrison, 2017).

This study also showed that greater change in 25(OH)D concentration was associated with higher summer 25(OH)D concentration. Such findings could be explained by differences in children's outdoor lifestyle or sun exposure protective behaviours. These were not measured in this study, however,it is plausible to suggest that while children with higher summer 25(OH)D concentration might have more outdoor lifestyle/physical activity and less sun exposure protective behaviours, children with lower summer 25(OH)D concentration might be opposite. Therefore, while seasonal

change in UVB dose and sun exposure could have a more pronounced effect on seasonal change in 25(OH)D concentration in the former group, it might be less effective in the latter group of children.

Children's BF% was also associated with seasonal change in 25(OH)D concentration, with higher BF being associated with a greater decrease in winter 25(OH)D concentrations. The role of obesity and higher BF% in having lower vitamin D status has been well documented (vitamin D deficiency/insufficiency is highly prevalent in individuals with obesity), however its role in seasonal change has not been previously reported (Golzarand et al., 2018; Savastano et al., 2017).

Vitamin D is fat-soluble and found in adipose tissue. Although the underlying mechanism is not clearly understood, it appears that vitamin D can be trapped in adipose tissue, potentially due to insufficient lipolytic stimulation and/or due to tissue dysfunction/adaptation resulting from adipose expansion. Evidence suggests that vitamin D mobilisation is accompanied by lipolysis process (Di Nisio, 2017). In vitro and in vivo studies have shown that the obese individuals' lipolytic response of adipose tissue to exercise is impaired and is less than that of individuals without obesity (Hellström, 2000; Large, 1999; Reynisdottir, 1994; Stich, 2000). This may contribute to sequestration of vitamin D in adipose tissue and consequently to lower vitamin D status (Di Nisio, 2017; Pramono, 2019). Our study showed that BF% was not associated with summer 25(OH)D concentration but was associated with winter and seasonal change in 25(OH)D concentration. Our findings may provide further evidence for the notion that vitamin D metabolism and uptake is not affected but its mobilisation from adipose tissue is impaired in obesity (Hengist, 2019).

One of the strengths of the study was the within-person seasonal assessment of vitamin D status together with the near-complete follow up. Ninety-eight percent of children sampled in summer were also sampled during the following winter. By measuring vitamin D status of the same children in summer and winter, the estimated change in mean 25(OH)D concentration is not affected by between-subject variation. There are, on the other hand, some limitations to this study; firstly, the findings cannot be generalised due to this study having a small sample size of 100 children who were all from one school, and although the UVB radiation in Auckland during winter is sufficient in the middle of the day to make vitamin D, this is not applicable to areas further south. Secondly, the association between UVB dose and 25(OH)D concentration could not be determined as not all the same children who provided blood samples wore the UV dosimeters, due to the limited number of dosimeters . Finally, other factors such as diet, time spent in or outdoors, amount of physical activity or bare skin exposure were not assessed and controlled for in this piece of research. However, it worth mentioning that food sources of vitamin D are very limited and their contribution to vitamin D status is minimal.

**CONCLUSION**

The results suggest that vitamin D insufficiency is potentially a problem for many children in New Zealand, as approximately one fifth of the children showed vitamin D insufficiency in summer, rising to half the children in winter. Further, this is not only affected by season and exposure to UVB light, it may also be influenced an individual's body fat percentage. As low vitamin D has the potential to cause harm to children's health, understanding vitamin D status in school-aged children and the contributing factors will help better inform potentially both health and education policy to combat these deficits.

# Chapter 4: Discussion

## *4.1 Overview*

Vitamin D is vital for calcium balance and bone health. Deficiency can cause rickets in children and is linked to respiratory, immune, metabolic, and neurodevelopmental disorders. In New Zealand, strong sun safety messages, limited dietary sources and varying amounts of UVB dose reaching the Earth's surface are among several factors that could be contributing to vitamin D insufficiency or deficiency, especially in winter and among Māori and Pacific children. With no national fortification programme, vitamin D status depends largely on UVB exposure, season, and behaviour. At present insight is lacking into vitamin D status of the childhood population in New Zealand.

This research set out to increase knowledge of vitamin D status in New Zealand children and further understand the relationship between vitamin D status and its change through seasonality and UVB exposure. The study aim was to determine seasonal variation, from summer to winter, for both vitamin D status and UVB dose in a sample of Auckland school children aged 11-12 years and whether this is influenced by other relevant factors. This was achieved by conducting a longitudinal study with repeated measures design, testing s-25(OH)D status in intermediate school children (aged 11-12 years) at two seasonally different timepoints, March (the beginning of autumn, after summer sun exposure) and September (the beginning of spring, after winter sun exposure).

## *4.2 Main Findings*

The research showed a significant seasonal change in s-25(OH)D status of the children, decreasing from summer to winter. This direction of change was mirrored by the UVB dose data, with a decrease in the dose the children were exposed to from summer to winter. This indicates as would be expected that reduced UVB exposure during winter months was impacting their vitamin D status. The change in vitamin D status across the seasons may be clinically relevant, as the number of children meeting a vitamin D insufficiency cutoff of <75 nmol/L also increased from summer to winter. Crossing this threshold may contribute to the increase in illnesses seen in this population during winter months including respiratory illnesses which are of particular concern in childhood populations. A larger decrease in s-25(OH)D across the seasons was observed in those with a higher concentration in summer, this could be due to those with lower summer concentrations engaging in more sun protective behaviours in both seasons meaning any change would be less marked. Those with a higher body fat percentage also experienced a larger decrease in s-25(OH)D from summer to winter, this was not unexpected as the association between low vitamin D status and higher body fat percentage has been previously documented (Karampela et al., 2021). The fact that body fat percentage was associated with change in S-25(OH)D across the seasons, rather than with summer

or winter status alone, suggests that a plausible mechanism is impaired mobilisation of vitamin D stores from adipose tissue, rather than an effect on vitamin D metabolism itself

## *4.3 Research Impact*

Research on vitamin D status in young people is lacking and particularly within a New Zealand context. As hypovitaminosis D has many potential negative health outcomes in children and young people, understanding where current status sits and what constitute the major contributing factors to status is important. This research will enhance the body of work in this field helping produce a clearer picture for public health policy development to improve the health of the population moving forward. These findings add to this knowledge area by showing children are exposed to less UVB during winter months and this is impacting their vitamin D status. The large proportion of children shown to be insufficient in vitamin D, even in summer, suggests those in charge of health and education policies may feel it pertinent to consider guidelines around sun exposure at school, balancing sun safe messaging with adequate exposure for vitamin D sufficiency, especially during winter months, or look to encourage vitamin D supplementation or fortification of foods. It may further help inform discussion of whether current cut offs for insufficiency and deficiency are set too low, whether these need to be raised or indeed whether there needs to be specific higher cut offs for children, considering the function of vitamin D for optimal neurodevelopment and immune health. The relationship identified between a higher body fat percentage and a greater seasonal drop in vitamin D status may help to inform clinical recommendations of vitamin d supplementation in certain populations. Lastly, the observation that higher body fat may be associated with reduced mobilisation of stored vitamin D, rather than altered metabolic conversion, highlights an important area for future investigation.

## *4.4 Strengths and Limitations*

The study using a repeated measures design was a strength as it meant vitamin D status was measured in the same children across the two seasons meaning the results were not affected by between-subject variation. Further, looking at seasonal data gives a more representative picture of vitamin D status across the year. This reduces the risk of under or over estimating prevalence of insufficiency and not taking data from a single time point means the results are less likely to be affected by uncontrollable variables such as unusual weather conditions or air pollution. Previous studies have considered the difference in s-25(OH)D concentrations across seasons, avoiding the bias of single-season sampling. However, this research also looked at percentage change across seasons giving further insight into the effect of season on vitamin D status. The findings help to quantify seasonal vitamin D fluctuations which will add to our understanding of how this is affected

by differences in UVB availability across the year and with further data collection this could be extrapolated to different regions.

Measuring UVB exposure through dosimetry rather than assuming exposure by using season as a proxy, allows for a stronger basis to interpret causal links between sunlight exposure and vitamin D status. By collecting UVB dose, this helps to distinguish between variation due to actual environmental UVB intensity and variation due to other behaviours such as sunscreen use. Further, assessing both modifiable (e.g. sunscreen use) and non-modifiable predictors of vitamin D status (e.g. age, skin colour) increases understanding of determinants of vitamin D status, which could be used to inform targeted public health recommendations. By looking at a variety of predictors of vitamin D status, for example environmental data through e.g. UVB exposure, behavioural data through e.g. sunscreen use and individual data through various anthropometric measures allow for the examination of interaction effects helping to elucidate what puts a person more at risk of insufficiency or deficiency.

The study is however limited as data were collected from a limited sample size (especially the dosimeter data) and in only one school population in Auckland. This lowers the ethnic and socioeconomic diversity of the sample population, reducing generalisability of the results. Collecting data from a larger number of school populations across a variety of regions would allow for comparisons of vitamin D status and UVB exposure across different latitudes in New Zealand, giving a greater understanding of the effect of this and whether public health messaging needs to be different depending on location. Sampling at more time points across the year and increasing the study length to gain data across multiple years would increase understanding of changes in status throughout the seasons. Collecting data from the same participants across years would give insight into how vitamin D status tracks through developmental milestones like puberty and its link to future disease, such as asthma incidence or cognitive outcomes such as educational achievements. The potential of engagement in physical activity as a confounder was not considered which may be a limitation, as it may influence both outdoor exposure to UVB and mobilisation of vitamin D from tissue stores. Dosimeters were only worn during school hours, and this does not consider weekend and after school exposure meaning total exposure could be underestimated. Further, although the majority of vitamin D is gained from sun exposure, dietary intake is a contributing factor, and this was also not recorded and considered.

## *4.5 Recommendations and directions for future research*

These research findings suggest vitamin D insufficiency and deficiency is a problem among childhood populations and there may be a necessity for screening of school-aged children to identify those in

need of intervention to prevent deficiency. However, a major hurdle for understanding vitamin D is establishing a universally accepted 'normal' status. Until this is achieved it will be hard to set down policies regarding hypovitaminosis D screening, prevention and treatment (Antonucci et al., 2018). Further research should look at using other tools to determine vitamin D status, for example using artificial intelligence, clinical symptom questionnaires, parathyroid hormone concentrations, and other vitamin D metabolites (Annweiler et al., 2015; Harvey et al., 2024; Sempos et al., 2018). This would potentially allow for validation of cheaper diagnostic tools and reduce the potential confounder of variability of assay testing among laboratories, facilitating both easier research and more targeted health care. It may also further reduce the issue of consistency of measurement across different assays as you would not need to rely on one measurement. Improving the wearable technologies, such as integrating UV dosimeters into smart watches would enable much larger-scale monitoring of these measures and allow easy collection across differing sites. This data would give a much clearer picture of exactly where cutoffs for sufficiency, insufficiency and deficiency should be set.

Further research regarding the association of increased body fat percentage and low vitamin D status would be appropriate. This mechanism is not fully understood with some questioning whether obesity affects vitamin D metabolism, for example it may reduce CYPR2R1 activity, an enzyme involved in the conversion of vitamin D3 to the active hormone, or whether high body fat percentage may affect the body's ability to mobilise the vitamin D stored in adipose tissue as our data suggests (Saponaro et al., 2020). In the current climate with rates of overweight and obesity rising in childhood populations in New Zealand, it is important to elucidate how these impact health outcomes in varying ways.

Research investigating vitamin D status in conjunction with other relevant micronutrient concentrations, for example calcium status, would help to explain how these combinations of nutrients play a role in health outcomes (Harvey et al., 2024). Further, linking micronutrients such as vitamin D status to socioeconomic factors such as access to outdoor environments could identify vitamin D status as a marker for wider inequalities in nutrition. This would allow for more robust guidance for public health policy and guidelines on health improving behaviours in childhood populations.

With vitamin D seen to be low in the childhood population, interventions to see what could improve status would be a natural progression of the research. Intervention trials looking at whether behaviour change programmes, supplementation or fortification of food could change vitamin D status especially in winter months when serum concentrations are lowest, would elucidate how

vitamin D status could be improved. School-based interventions such as free vitamin D fortified milk, mandatory outdoor playtime during the winter months or education on the importance of balancing sun exposure with sun safe practices, could all improve understanding of how to improve low vitamin D levels in the childhood population. This could further be targeted specifically at high-risk groups such as those with higher body fat percentage to see if this improved status in these subsets of the population. Other factors in the school environment that might affect vitamin D status beyond personal behaviours could also be considered, for example school layout and design, e.g. access to outdoor space, or class scheduling and timing of outside breaks in the day, or school sun safe policies or practices.

When examining vitamin D status in childhood populations in New Zealand, where there are multitude of different ethnicities, it is important to adopt an equity-focused approach. Although this study did not identify ethnicity as a factor explaining differences in vitamin D status, as Māori and Pacific children are disproportionately affected by vitamin D insufficiency and related health inequities, reflecting broader social and environmental determinants of health, future research may target these communities to see if interventions could improve outcomes. For example giving free vitamin D fortified milk to see if status improves in a particular school population. Any intervention should however be community-led, partnering with Māori and Pacific researchers and organisations to ensure that study design and interventions are culturally relevant, acceptable, and effective.

Looking forward, monitoring long term UVB availability in relation to climate change and potential changes in weather such as longer periods of heavy rain and increased cloud cover could be important to understand how this could impact vitamin D status and health and if public health policy guidelines are needed to mitigate climate change effects.

## *4.6 Conclusion*

This study demonstrates a marked seasonal decline in vitamin D status among Auckland intermediate school children, driven by reduced winter UVB exposure and influenced by behavioural and physiological factors such as sun-safety practices and body fat. Adiposity appears to limit mobilisation of stored vitamin D rather than alter its metabolism. These findings highlight the need for balanced public-health guidance that promotes safe sun exposure while preventing deficiency. A comprehensive, equity-focused approach to maintaining adequate vitamin D status in childhood will be critical for supporting musculoskeletal, immune, and neurodevelopmental health across the lifespan in Aotearoa New Zealand.

## *Appendices*

MASSEY UNIVERSITY

DISC Study

Vitamin D in Intermediate School Children

INFORMATION SHEET FOR PARENTS

Thank you for your interest in the DISC (vitamin D) study. This sheet gives information on the conduct and organisation of this study, including confidentiality and data protection. It is important that you read this and are happy with the information given before agreeing to take part in the study.

**Why is this research important?**

Vitamin D is an important nutrient for health and wellbeing in growing children. We want to find out about vitamin D status in Intermediate School children living in Auckland. Because we get most of our vitamin D from the sun, we are going to look at how status varies between the end of summer and end of winter. We will be also be investigating things that affect vitamin D status such as dietary intake, sun exposure behaviours, physical activity and body composition.

Who are we looking for?

We are inviting all children at Birkdale Intermediate School to take part in this study. Each child and at least one of the child's parents/guardians need to be able to read and comprehend English to a sufficient level that they can understand the information provided about the study and make an informed decision about whether or not they wish to participate.

Children who have any type of bleeding disorder cannot participate in this study. Also, children who have any metal in their body, for instance screws or plates from fractures, should not be measured on the BIA, but can still participate in the study. Please note this on the questionnaire about your child.

What is going to happen?

All data collection from the children will take place in school. Our first visit will be at the end of March, and the second visit at the end of August. Further information will be sought from you, parents or caregivers, through questionnaires, which your child will bring home, along with a consent form.

One questionnaire will have questions about your child such as: gender, date of birth, ethnicity, history of chronic illness. There will two other questionnaires; one about your child's physical

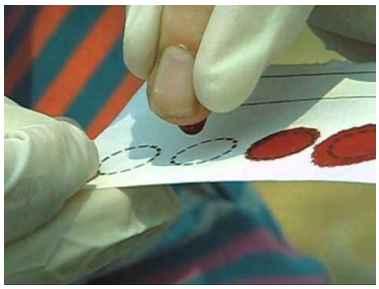
activity, and the other is called the DICE (Dietary Index for a Child's Eating) questionnaire and is a validated questionnaire about food intake. These questionnaires will take approximately twenty minutes to complete. You will only need to complete these questionnaires once.

All data collection from the children will take place in school. The children will have measurements taken to determine their body composition (level of muscle and fat), and a finger prick blood spot to measure their vitamin D levels. Vitamin D will be measured again at the end of winter.

**Height and waist measurement**
We will ask your child to remove their shoes to measure their height. Standing height using a stadiometer and waist circumference using a measuring tape will be measured for each child.

**BIA**
We will ask your child to remove their shoes and socks and stand on the machine's scale holding on to the two handles. This will measure your child's weight and determine body lean and fat mass percentages.

**Finger prick blood spot**
We will ask children to wash their hands with warm water. They will then be asked to shake their hands and choose a finger for finger prick. The finger will be pricked with a lancet and 2 drops of blood will be collected for vitamin D analysis and will be sent to a laboratory in Christchurch.

Who will see the information about your child?
All information about your child will be stored in a locked filing cabinet accessed by the research team only. No names or any other information that could be used to identify your child will be used in any publication.

We are required to keep any data that may be medically relevant for your child in the future for ten years. All electronic data will be stored password-protected on the University's secure server. For the first 5 years we will store any paper copies of data in a locked filing cabinet within a locked office. For the remainder of the time, data will be stored in a secure archive in boxes labelled by barcode only. This data will be accessible by nominated staff only. After the mandatory storage time has passed, all data filed on paper will be shredded and electronic data will be deleted from our computer records and databases.

Would your child like to take part?
If "YES"
If your child would like to take part in this study and you are happy for them to do so, please sign the attached consent form and ask your child to return it to their teacher.

If "NO"
If you do not want your child to participate or your child does not want to take part in this study then you do not need to do anything. We will not collect any data from or about your child.

**What are the benefits and risks of taking part in this study?**

- You will receive a brief report summarising the main findings of the project via mail or email.
- The principal benefit of taking part in this study is that you will contribute to a study and our understanding of vitamin D status and its relationship with dietary intake, physical activity, sun exposure behaviours and body composition in children.
- If, when the blood spots are analysed, we find that your child had unusually low levels of vitamin D, we will inform you via the contact information given on your returned form.
- It is not envisaged that there will be any discomfort or risk to the children as a result of participation, other than the minor discomfort of the finger prick blood test.
- If you have any specific requirements including cultural requirements or concerns about the project, or about being a participant, please contact a member of the research team to discuss.

**Who is funding the research?**
This is funded by a grant from the Massey University Research Fund.

What are my rights and the rights of my child?

We respect your rights and your child's rights to:

- refuse to answer any particular question or take part in any testing (finger prick blood spot or BIA)
- withdraw from the study at any time
- ask further questions about the study that occur to you during your participation
- provide information on the understanding that it is completely confidential to the researchers. All information is collected confidentially, and it will not be possible to identify you or your child in any reports that are prepared from the study
- be given access to a summary of the findings from the study when it is concluded.

Compensation for Injury

In the unlikely event that physical injury results from your child's participation in this study, you should visit a treatment provider to make a claim to ACC as soon as possible. ACC cover and entitlements are not automatic and your claim will be assessed by ACC in accordance with the Compensation Act 2001. If your claim is accepted, ACC must inform you of your entitlements,

and must help you access those entitlements. Entitlements may include, but not be limited to, treatment costs, travel costs for rehabilitation, loss of earnings, and/or lump sum for permanent impairment. Compensation for mental trauma may also be included, but only if this is incurred as a result of physical injury.

If your ACC claim is not accepted you should immediately contact the researcher. The researcher will initiate processes to ensure you receive compensation equivalent to that to which you would have been entitled had ACC accepted your claim from Massey University.

If you have any questions please contact Dr Pamela von Hurst who will be happy to discuss the project in more detail.

Contact details:

Dr Pamela von Hurst
School of Sports, Exercise and Nutrition, College of Heal
Massey University
Email P.R.vonHurst@massey.ac.nz
Phone (09) 213 6657

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 19/13 If you have any concerns about the conduct of this research, please contact Dr Lesley Batten, Chairperson, Massey University Human Ethics Committee: Southern A, telephone 64 6 356 9099 ext 85094. Email humanethicsoutha@massey.ac.nz.

Identification Number .....

Date .....

# DISC Study

## Vitamin D in Intermediate School Children

**Thank you for participating in this study, if you have any questions please feel free to discuss them with the researcher.**

**Principal Investigator:**

Dr Pamela von Hurst
School of Sports, Exercise and Nutrition, College of Health
Massey University
Email P.R.vonHurst@massey.ac.nz
Phone (09) 213 6657

**All information you provide will remain strictly confidential**

**Please return this questionnaire in the envelope provided**

Identification Number. .....

## Participant Demographics

**First Name of your child**

.....

**Family Name of your child**

.....

**Date of birth of your child**

.....

**Gender** (please tick)

**Female** ☐ **Male** ☐ **Gender Diverse** ☐

**Phone (home)**

.....

**Phone (mobile)**

.....

**Email**

.....

**How would you describe your child's skin colour? (Please √ one)**

☐ Fair ☐ Medium ☐ Olive ☐ Dark/Brown

☐ Other Please specify:.....

**Is your child taking any medication or supplements? Please list**

.....

.....

.....

**Which ethnic group or groups does your child identify with? (Please √ all that apply)**

| | | |
|---|---|---|
| New Zealand European | □ | |
| Maori | □ | |
| Pacific | □ | Please specify _____ |
| South Asian | □ | |
| Chinese | □ | |
| Korean | □ | |
| Southeast Asian | □ | Please specify _____ |
| Other ethnicity | □ | Please specify _____ |

**Is your child taking any medication or supplements? Please list**

.....
.....
.....

**Does your child have any chronic illness (for example, asthma), a bleeding disorder or food allergy? Please list**

.....
.....
.....

**Does your child have any metal implants, such as plates or screws in his/her body?**

.....
.....

**Does your child wear sunscreen at school? Please circle correct answer below**

| | | |
|---|---|---|
| In summer | Yes | No |
| In winter | Yes | No |

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