# VITAMIN D DEFICIENCY RICKETS

**Background to Study:**

Vitamin D is critical for calcium homeostasis and for mineralization of the skeleton, especially during periods of growth. The most severe consequence of vitamin D deficiency during childhood is rickets (a mineralization defect at the epiphyseal growth plates). Rickets is associated with pain, fractures, skeletal deformity, growth restriction, dental defects, delayed developmental milestones and, in severe cases, hypocalcaemic seizures. Deficiency of vitamin D in children, without overt rickets, is also associated with low bone mineral density which has potential long-term implications particularly in adulthood. Of added concern, it has been recognised that Vitamin D has a major role to play in the developing and developed immune system. Low levels during foetal life, early infancy and into adulthood potentially have a major effect on the development of infection, autoimmune and cardiovascular disease, and possibly cancer.

Vitamin D is unique among vitamins as its main source is not dietary, but direct synthesis in the skin following exposure to UVB radiation from sunshine. UVB radiation exposure varies based on latitude, skin colour, sunscreen use and clothing. Changes in human lifestyle including sun avoidance practices, indoor occupations and recreation, added to New Zealand's geographical location at 35ºS to 47ºS mean that children and adults cannot depend on adequate skin exposure to sunlight for vitamin D synthesis, especially during winter months. Dietary intake is a secondary source, but for a few exceptions, without additional supplementation there is little in the foods humans normally ingest. Compounding this issue, human milk, is often not rich in vitamin D.

There is evidence from across NZ and around the globe that vitamin D deficiency and rickets are increasingly common. Particularly at risk are infants who are breast-fed by mothers with poor vitamin D intake or inadequate sun exposure (due to veiling/southern latitudes/dark skin)

Despite this knowledge, there are no national recommendations for supplementation (targeted or universal) of exclusively breast-fed infants, and as opposed to many countries, NZ currently has no mandatory and little voluntary food supplementation. The current NZ Ministry of Health position is that supplementation is unnecessary.

The precise incidence of vitamin D deficiency rickets in New Zealand is unknown. This lack of available incidence and prevalence data and the concern that this preventable disease is on the rise underline the importance of a prospective surveillance study.

**Objectives:**

1. To ascertain the incidence of simple vitamin D deficiency rickets (also known as nutritional rickets) diagnosed by specialist paediatricians over a two-year period
2. To obtain demographic and medical information which will assist in the:
    a. Identification of risk factors for development of the disease in NZ
    b. Evaluation of current preventive strategies.
3. To supply data that will help develop public health policies to prevent vitamin D deficiency rickets among children living in New Zealand.

**CASE DEFINITION**

Children up to and including 15 years of age with rickets secondary to simple Vitamin D deficiency (also known as nutritional rickets) confirmed biochemically and/or radiographically.

**Inclusion criteria (Biochemical)**

1. Low serum 25-hydroxyvitamin D (25OHD) (<50nmol/L)
2. Elevated serum alkaline phosphatase (ALP) (age specific)

**Supplemental data *ideally* to be obtained prior to treatment (and expected results*):**

1. Serum calcium (normal or low)† and albumin
2. Serum phosphate (normal or low)
3. Serum PTH (elevated)
4. Serum 1,25-dihydroxyvitamin D (1,25(OH)2D); low, normal or high)
5. X-ray confirmation of rickets at the distal ulnar or femoral epiphysis‡

* These results are not essential for reporting.

† Ionized calcium is also acceptable.

‡ In rare instances, the x-ray features of rickets may not be present at diagnosis e.g. if linear growth is arrested (and growth plate activity is blunted) or in the very early phase of the disease when x-ray changes at the growth plate are not yet visible. For this reason, although x-ray confirmation of rickets is not a strict inclusion criterion it should be obtained during the initial patient evaluation.

**Follow-up of positive returns:**

A questionnaire requesting further details will be sent to notifying paediatricians.

**If you have any questions please contact:**

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