Copyright is owned by the Author of this thesis. Permission is given for a copy to be downloaded by an individual for the purpose of research and private study only. The thesis may not be reproduced elsewhere without the permission of the Author.

# Complementary Feeding Practices, Nutrient Intake, and Iron Status of Māori, Pasifika, and Other Infants in Aotearoa New Zealand

A thesis presented in partial fulfilment of the requirements for the degree of

Doctor of Philosophy
in
Nutritional Science

Massey University, Albany
Aotearoa New Zealand

Maria Casale
2023

# Dedication | Whakaihi

This thesis is dedicated to the beautiful pēpi and their whānau who took part in the First Foods New Zealand study.

*Kia kaha*

*Kia māia*

*Kia manawanui*

*Ioane William Kupa, shared with permission*

# Abstract | Tuhinga Whakarāpopoto

**Background:** The period of transition from a solely milk-based diet to sharing family foods at around 12 months of age is a critical time for infants. Complementary feeding practices, from the age of introduction to complementary foods, method of feeding (baby-led weaning vs. traditional spoon-feeding), use of the novel baby food pouches, use of traditional cultural foods and practices, and the characteristics and nutrient density of first foods offered support the healthy growth and development of the infant, as well as shape long term dietary patters and food preferences. Additionally, iron status is crucial for healthy infant growth and development, and while this is impacted by myriad maternal, genetic, and environmental factors, complementary feeding practices and the characteristics of foods offered are key modifiable practices that influence infant iron status.

**Aims and objectives:** The overall aim of this study was to investigate and describe early infant feeding practices, key nutrient intake and density, and the iron status of Māori, Pasifika and other infants living in Aotearoa New Zealand, using an observational cross-sectional study design. The primary objective was to conduct an intra-ethnic analysis of infant complementary feeding practices, nutrient intake and density from complementary foods, and iron status between Māori, Pasifika, and 'other' infants. 'Other' refers to any infants who were not self-identified by the adult respondent as Māori or Pasifika.

**Methods:** Infants aged 7.0–10.0 months along with their primary caregiver participated in an observational cross-sectional study, with 625 infant–caregiver dyads recruited from Auckland and Dunedin, New Zealand. Participants were recruited from a range of ethnic groups and deprivation statuses. Infants were stratified by ethnicity using total response for Māori and Pasifika, with all non-Māori, non-Pasifika infants categorised into a single 'others' group. Demographic and feeding practices data were collected via questionnaire. Nutrient intake from complementary food was measured using the multiple-source method from two multiple-pass 24-hour diet recalls. Nutrient density of complementary food was calculated as the concentration of selected nutrients per 418 kJ (100 kcal) of energy. For iron status, haemoglobin, plasma ferritin, soluble transferrin receptor, C-Reactive protein, and alpha-glycoprotein were obtained from a venous blood sample. Inflammation was adjusted for using the Biomarkers Reflecting Inflammation and Nutritional Determinants of Anaemia (BRINDA) method. Body iron concentration (mg/kg body weight) was calculated using the ratio of sTfR and ferritin.

**Results:** A total of 1424 infant-caregiver dyads were assessed for eligibility, and 625 eligible dyads were enrolled in the study, all of whom provided written consent. Data for complementary feeding practices and nutrient intake were analysed for all 625 infants, with blood samples obtained from 365 of these infants for the assessment of iron status. Within the cohort 131 infants were identified by their caregiver as Māori, and 82 as Pasifika. The remaining infants are allocated into a single 'others' group as the primary focus of this manuscript is Māori and Pasifika infants. The mean (SD) infant age was 8.4 (0.8) months for Māori, 8.5 (0.9) months for Pasifika, and 8.4 (0.8) months for 'other' infants. Over half of all ethnicities introduced CF at around six months of age (56.5% of Māori, 62.2% of Pasifika, and 80.9% of 'others'). BLW prevalence increased from 11.5% of Māori, 3.7% of Pasifika, and 12.4% of 'other' infants at the time of introducing CF to 29.2% of Māori, 17.1% of Pasifika, and 27.3% of 'others' currently. Baby food pouches were used at least once by 89.3% of Māori, 85.4% of Pasifika, and 75.6% of 'other' infants. Of those who always or frequently were fed pouches, 27.1% of Māori, 25% of Pasifika, and 12% of 'other' infants always or mostly sucked directly from the nozzle. Vegetables and 'pureed' were the most common first food and texture offered, respectively, for all ethnic groups. At six months red meat was consumed by 54.6% of Māori infants, 63.4% of Pasifika infants, and 61.8% of 'other' infants, and approximately half had iron-fortified baby rice (Māori 57.3%, Pasifika 56.1%, 'other' 48.7%). Age-inappropriate drinks were currently given to 17.6% of Māori, 20.7% of Pasifika, and 3.8% of 'other' infants. In total, 9.1% of Māori and 20.7% of Pasifika respondents reported offering traditional cultural foods to their infants. Energy intake increased with age for all ethnic groups and was higher for boys than girls. Protein as a percentage of energy intake from CF was significantly lower for Māori compared to 'others'. Fat as a percentage of energy intake from CF was significantly lower for both Māori and Pasifika than 'others', whereas carbohydrate as a percentage of energy intake from CF was significantly higher. Sugar intake in grams from CF was significantly higher for Pasifika when compared to 'others', and sugar as a percentage of energy intake from CF was significantly higher for Māori than 'others'. Iron, zinc, and calcium density of the complementary diet was inadequate for all groups: Māori and 'others' had an iron density of 0.8 mg/418 kJ, and Pasifika 0.9 mg/418 kJ. Zinc density was 0.5 mg/418 kJ for all groups. Calcium density was 37 mg/418 kJ for Māori, 40 mg/418 kJ for Pasifika, and 38 mg/418 kJ for 'others'. In total, 96.4% of Pasifika infants were iron sufficient, compared to 82.5% of Māori and 76% of 'other' infants. 'Other' infants had the highest prevalence of iron deficiency overall, with 3% categorised with iron-deficiency anaemia, 12% with early functional iron deficiency, and 9% with iron depletion. For Māori infants, 4.7% had iron-deficiency anaemia and early functional iron deficiency, respectively, and 8% were iron depleted. One (3.6%) Pasifika infant was iron depleted, and the remainder were iron sufficient. Mediation analysis suggested that the difference in body

iron concentration between Pasifika and ‘others’ was partially explained by the frequency of their higher consumption of baby food pouches.

**Conclusions:** The high prevalence of Māori and Pasifika infants feeding directly from baby food pouch nozzles is concerning in light of the increasing popularity and prevalence of this novel feeding device, with concerns for both the safety and impact on development of this way of feeding. The low density of iron, zinc, and calcium in the complementary diet warrants further investigation into feeding and fortification strategies, due to the key role these nutrients play in the growth and development of infants. The rate of iron deficiency was very low for Pasifika infants despite little iron intake from complementary food, indicating non-dietary factors as the likely cause for this group.

# Acknowledgements | He Mihi

*Ehara tāku toa i te toa takitahi, engari he toa takitini*
*Success is not the work of an individual, but the work of many*

The opportunity to do this PhD project has been an enormous privilege. It has been a huge honour to be supervised and supported by a team of strong and fiercely intelligent mana wāhine. To my primary supervisor, Pamela von Hurst: thank you for giving me this incredible opportunity and for your faith in me. Your kindness and patience when I struggled, along with your steadfast belief and support have been invaluable and so deeply appreciated. You have always been in my corner, fielding my many off the cuff questions, and giving me clear and insightful guidance with my writing and thinking. To my co-supervisors Cathryn Conlon and Kathryn Beck: you encouraged me to have confidence in myself and allowed me the space and time I needed to figure out things on my own with understanding and grace. You have both been a quietly strong and ever-present source of strength and support for me throughout this journey, and I feel blessed to have had this. To my co-supervisor Lisa Te Morenga: what a gift it was to have you on my team – keeping me grounded and centred in my intentions to show respect, understanding, and acknowledgement of the past harms to our Māori and Pasifika communities through Māori- and Pasifika-centred research. Ngā mihi nui ki a koe.

Thank you to Anne-Louise Heath and Rachael Taylor for leading the First Foods New Zealand (FFNZ) research study, your advice, support, and reviewing of manuscripts.

My colleagues and friends: Kimberley Brown, Rio Jupiterwala, Emily Jones, and Andrea Wei – thank you for your unending support through challenging times, and for your total dedication to our study.

To Rebecca Paul: getting to know you has been one of the highlights of the past few years. Your strength, resilience, support, and tough no-nonsense kindness has meant so much to me.

To the wider FFNZ team in Dunedin: Jenny McArthur, Lisa Daniels, Jill Haszard, Liz Fleming, Ioanna Katiforis, Neve McLean, Alice Cox, and Maddie Rowan: thank you all so much for your incredibly hard work with data collection, participant management, quality control, and the innumerable other jobs you all did to keep our study going so smoothly.

Finally, to my family, Will, Charlotte, and Ioane, and my parents, Remo and Jenny: thank you for your encouragement, support, and understanding as I have gone through this process and through the difficult times.

# Table of Contents | Rārangi Take

# List of Tables | Ngā Tēpu

# List of Figures | Ngā Hoahoa

# Glossary | Kuputaka

## AOTEAROA

The Māori language name for New Zealand. In this thesis Aotearoa is used interchangeably with New Zealand, or referred to as "Aotearoa New Zealand", abbreviated as NZ. Aotearoa was first used for the North Island only, with the post popular meaning being "long white cloud".

## ATUA

Atua (singular and plural) refers to Polynesian spirits or Gods and were central to Māori spirituality. Atua also means "God" in Samoan, and the literal Polynesian translation is "power" or "strength". Atua may also be used to refer to the spirits of powerful Chiefs.

## KAI

Kai is the Māori word for food and can also be used to mean "eating" or "eat".

## MANA

Mana refers to individual and collective power, prestige, and status, but at the same time humility. Traditionally mana was thought of as a supernatural force conferred or inherited at birth and can be enhanced by success or decreased by failure or undesirable behaviour. Inanimate objects may also be imbued with mana due to their association with people with mana or their use in important events.

## MANAAKITANGA

The care, respect, and generosity shown to others (guests, family, the wider community). This may refer to hospitality given, or respect and care given for others' stories and information.

## MANUHIRI

Manuhiri are visitors or guests. For example, a group of people who visit a marae are considered manuhiri.

## MĀTAURANGA MĀORI

Māori knowledge / wisdom drawn from traditions, values, concepts, philosophies, world views, and understandings derived from a uniquely Māori cultural perspective. It is differentiated from a Western approach in that there is an emphasis on the personal or human perception of a thing, rather than what the role of that thing is.

### MINISTRY OF HEALTH / MANUTŪ HAUORA

The government department responsible for the health and disability system in Aotearoa New Zealand, including administering legislation and associated regulations.

### ŌTEPOTI

The Māori language name for Dunedin, a main urban centre in Aotearoa New Zealand.

### PĀKEHĀ / PALANGI

The Māori and Pacific Island term for people of primarily European descent, originally applied to English-speaking Europeans living in Aotearoa New Zealand. Historians agree that the origins of the word Pākehā is likely to be "pale, imaginary beings resembling men". There has been backlash previously, likely due to the coloniser being asked to refer to themselves in the language of the colonised, but Pākehā is now a mainstream term.

### PASIFIKA

This refers to Polynesian people, languages, and culture, including Samoan, Tongan, the Cook Islands, Fijian, Niuean, Tokelauan, Tuvaluan, and other Pasifika heritages. It describes both migrants from the Pacific regions and their descendants who may be New Zealand-born.

### PĒPI

The Māori language word for baby / infant.

### PLUNKET

A charitable organisation that provides support for the health and wellbeing of children from birth until they turn 5 years of age. The service provides free health and development checks, a 24-hour parenting helpline, and a range of community services such as parenting groups and group education seminars for whānau.

### RANGATIRA

Tribal chiefs in Māori culture, with high mana and ranking. Usually financially well off and revered.

### TAHA HINENGARO

This is one of the four pillars of Māori health and wellbeing and refers to mental health. It encompasses the mind, heart, conscience, thoughts, and feelings, as well as communication.

### TAHA TINANA

The second pillar of Māori health and wellbeing, this refers to physical well-being.

### TAHA WAIRUA

The third pillar of Māori health and wellbeing, this refers to one's spiritual wellbeing. It refers to your life force – who and what you are, where you have come from, and where you are going. This can be viewed in different ways depending on the individual's spiritual beliefs.

### TAHA WHĀNAU

The fourth and final pillar of Māori health and wellbeing, this refers to those who give the individual a feeling of belonging or a sense of community, friendship, and love. It can refer not just to family, but colleagues, friends, and people that a person cares about.

### TĀMAKI-MAKAURAU

The Māori language name of Auckland, the largest city in Aotearoa New Zealand.

### TAMARIKI

The Māori language word for "children".

### TANGATA WHENUA

The literal definition is "people of the land" and refers to local people who have a deep ancestral and spiritual connection to the land they live on.

### TAONGA

Referring to a "treasure", whether it be tangible or intangible.

### TE TIRITI O WAITANGI / THE TREATY OF WAITANGI

The founding document of New Zealand, the Treaty is an agreement between the British Crown and approximately 540 Māori chiefs. It has been written in English and translated into Māori by Henry Williams and his son Edward overnight. There is controversy around the accuracy of the translation. The Treaty is a broad statement of principles upon which the British and Māori agreed to found a nation state and build a government in New Zealand.

### TE WHARE TAPA WHĀ

This is a Māori model of health and wellbeing, consisting of four sides or pillars: taha Hinengaro (mental health), taha tinana (physical health), taha wairua (spiritual health), and taha whānau (family health)

### WHĀNAU / 'AIGA / WHĀMERE

The Māori, Samoan, and Cook Island Māori words, respectively, for "family".

# Abbreviations | Whakapoto

| | |
|---|---|
| 24h-recall | 24-hour dietary recall |
| AGP | α-1-Acid Glycoprotein |
| AI | Adequate Intake |
| BLISS | Baby-led Introduction to Solids |
| BLW | Baby-Led Weaning |
| BMI | Body Mass Index |
| BRINDA | Biomarkers Reflecting Inflammation and Nutritional Determinants of Anaemia |
| CF | Complementary Food |
| CFFQ | Complementary Food Frequency Questionnaire |
| CHD | Coronary Heart Disease |
| CIF | Commercial Infant Food |
| CRP | C-Reactive Protein |
| CV | Coefficient of Variation |
| DFE | Dietary Folate Equivalent |
| DNA | Deoxyribonucleic Acid |
| DQ | Developmental Quotient |
| DRI | Dietary Reference Intakes |
| EAR | Estimated Average Requirement |
| EDR | Estimated Dietary Records |
| EER | Estimated Energy Requirement |
| EFSA | European Food Safety Authority |
| ESPGHAN | European Society for Pediatric Gastroenterology, Hepatology, and Nutrition |
| FFNZ | First Foods New Zealand |
| FFQ | Food Frequency Questionnaire |
| FNB:IOM | Food and Nutrition Board: Institute of Medicine |
| GI | Gastrointestinal |
| GUINZ | Growing Up in New Zealand |
| H.Pylori | Helicobacter Pylori |
| Hb | Haemoglobin |
| ID | Iron Deficiency |
| IDA | Iron Deficiency Anaemia |
| IQ | Intelligence Quotient |
| KCAL | Kilocalorie(s) |
| LBW | Low Birth Weight |
| MCH | Mean Cell Haemoglobin |
| MCV | Mean Corpuscular Volume |
| MELAA | Middle Eastern, Latin American, and African |
| MFP factor | Meat-Fish-Poultry factor |
| MOH | Ministry of Health / Manutū Hauora |
| NHANES | National Health and Nutrition Examination Survey |
| NHMRC | National Health and Medical Research Council |
| NRVs | Nutrient Reference Values for Australia and New Zealand |
| NZ | Aotearoa New Zealand |

| | |
|---|---|
| NZE | New Zealand European |
| PEM | Protein-Energy Malnutrition |
| PF | Pouch Feeding |
| PUFA | Polyunsaturated Fatty Acids |
| RCT | Randomised Controlled Trial |
| RDA | Recommended Daily Allowance |
| RDI | Recommended Dietary Intake |
| RE | Retinol Equivalents |
| RNA | Ribonucleic Acid |
| SCL | Southern Community Laboratories |
| SD | Standard Deviations |
| SF | Serum Ferritin |
| SI | Serum Iron |
| ST | Serum Transferrin |
| sTfR | Soluble Transferrin Receptor |
| TEE | Total Energy Expenditure |
| TIBC | Total Iron-Binding Capacity |
| TS | Transferrin Saturation |
| TSF | Traditional Spoon-Feeding |
| UK | United Kingdom |
| UL | Upper Limit |
| VLBW | Very Low Birth Weight |
| WDR | Weighed Dietary Records |
| WHO | World Health Organization |
| ZPP | Zinc Protoporphyrin |

# Chapter 1 | Upoko Tuatahi

## Preface

*This chapter provides an introduction and justification for this thesis. The objectives and outcomes are then presented, followed by an outline of the thesis structure and a description of researchers' contributions.*

Whaowhia te kete mātauranga

*Fill the basket of knowledge*

## Introduction and Justification for Study

How and what we feed our infants is a crucial component of their growth and development, both physically and socially. It is how we connect with, nurture, and love our children. It is how we share and teach a love of food, all while providing crucial nutrients for growth and development. The complementary feeding period where the infant makes the transition from a solely milk-based diet to sharing the family foods by 12 months of age is a critical time in the infant's life, and a large body of evidence guides the recommendations on infant feeding. The World Health Organisation (WHO) recommend exclusive breastfeeding for the first 6 months, and nutritionally adequate and safe complementary food (CF) to be introduced at 6 months together with breastfeeding up to 2 years of age or beyond (1).

Complementary feeding practices play an important role in the healthy growth and development of the infant and influence dietary patterns and preferences into adulthood. Currently, the World Health Organisation (WHO) and the Ministry of Health (MOH) recommend introducing safe and appropriate complementary food at around six months of age (1, 2), starting with a thin smooth puree via the spoon after a breast or formula feed. Variety of foods and textures should gradually be introduced with the infant becoming more independent with self-feeding until sharing the family foods at 12 months of age (2). The practice of baby-led weaning (BLW), whereby the infant has total control over their own feeding with non-texture modified family foods has been popular for many years now but is not recommended by the MoH (3). This is due to safety concerns around choking risk, nutrient intake, and growth. A novel form of infant feeding are baby food pouches, which are made of soft squeezable plastic with a nozzle attached that infants are able to suck food from directly. These are virtually unstudied beyond prevalence data. Health related concerns for pouch use are nutrient content (high sugar, low iron), dental health, increased energy intake, and lack of appropriate texture progression (4-9). The German Society for Pediatrics and Adolescent Medicine have released a position statement stating that young children should not suck directly from the pouch nozzle (8), but prevalence data for pouch use are currently unknown in NZ.

The food offered in the complementary feeding period also has a crucial role to play in the healthy growth and development of the infant. The infant's nutritional requirements per kilogram of body weight are greater than at any other life stage, yet relatively little energy is consumed from CFs. Therefore, CFs must have enough nutrient density to satisfy the infant's requirements while meeting their estimated energy requirement. Furthermore, this is the stage at which long-term taste

preferences are shaped. Common nutrients of concern in the infant's complementary diet are iron, zinc, and calcium. When assessing the nutrient intake of infants, each nutrient is typically measured from estimated milk feeds, which is unreliable for breast milk, and a diet record. This does not consider the infant's energy intake, so an alternative is to examine the nutrient density (the ratio of nutrient to energy) of complementary foods, which allows energy intake to be captured. Building on this, a reference nutrient density can then be established which is referred to as the "critical nutrient density". If an infant's diet has adequate critical nutrient density, the recommended nutrient intake will be met when the energy requirements are met. Therefore, use of nutrient density will allow any shortfalls in the infant's complementary diet to be identified. This is also a useful approach as in this age group it is appropriate to manipulate the complementary diet to meet requirements rather than breast or formula feeds.

Research quantifying the nutrient density of NZ infants' CFs has not been identified, yet the relationship between early-life nutrition and health outcomes through the lifecycle, such as type 2 diabetes and cardiovascular disease, are well-established (10, 11). Māori and Pasifika people are differentially impacted by both communicable and non-communicable disease (12, 13), the likelihood of which can be traced back to early-life nutrition.

Healthy term infants are born with sufficient iron stores to last approximately six months (14), necessitating early introduction of iron rich foods at the initiation of complementary feeding (15). However, iron is arguably one of the more difficult nutrients to ensure adequacy of for infants, partly due to haem iron dietary sources (from meat, fish and poultry) being more challenging foods to texture-modify for an infant, as well as the low absorption rate of non-haem iron (16). An infant's iron stores are determined by a complex interplay of myriad factors though, from maternal iron stores, gestational age at birth, iron losses and requirements, genetic factors, illness and inflammation, and dietary iron and accompanying modifiers. Understanding the causative factors of iron deficiency in infants is important as it is associated with immune deficiencies, delayed motor skill development, and cognitive development. These effects can be permanent even in the absence of anaemia (17, 18), and with iron repletion (18, 19). Iron status of infants has been well-studied internationally, but recent studies in NZ, especially those stratified by ethnicity, are scarce. Historical data suggest that 2.8 – 22% of infants in NZ aged 6 – 12 months have at least some degree of iron deficiency (20-22), with Māori and Pasifika infants having a rate of as high as 65%. However, most of these studies failed to control for infection, used varying indices and cut-offs to determine iron

status, and some used data from hospitalised infants which may not be generalisable to the population.

In NZ, the greatest inequalities in childhood outcomes related to nutrition occur due to differences in ethnicity (23). While there is a consensus on appropriate food and nutrients with appropriate texture progression for the weaning infant, and emerging but conflicting evidence for BLW as a feeding method, it is less clear what, and how, different ethnic groups within New Zealand are feeding their pēpi. In NZ, no studies specifically investigating ethnic differences in infant feeding practices exist, and there are few examining dietary or nutrient intake.

## Study Objectives

The First Foods New Zealand (FFNZ) was a multi-centre study in Auckland and Dunedin, running from July 2020 to February 2022. The overarching aim of this thesis was to investigate and describe early infant feeding practices, key nutrient intake, and iron status of Māori, Pasifika, and other infants using an observational cross-sectional study design.

The primary objective of this thesis was to conduct an intra-ethnic analysis of infant complementary feeding practices, nutrient intake and density from complementary foods, and iron status between Māori, Pasifika, and 'other' infants. 'Other' refers to any infants who were not self-identified by the adult respondent as Māori or Pasifika.

## Aims & Objectives

1. Investigate complementary feeding practices between Māori, Pasfiika, and non-Māori, non-Pasifika infants aged 7.0 – 10.0 months.

   *Objectives:*

   - Categorise complementary feeding practices
   - Evaluate and describe complementary feeding practices by ethnic group

2. Explore differences in nutrient intake and density from the complementary diet of Māori, Pasfiika, and non-Māori, non-Pasifika infants aged 7.0 – 10.0 months.

*Objectives:*

- Analyse ethnic differences in the intake of energy and key nutrients (protein, fat, saturated fat, carbohydrate, sugar, iron, zinc, calcium, vitamin C, vitamin A, and vitamin $B_{12}$ of Māori, Pasfiika, and non-Māori, non-Pasifika infants aged 7.0 – 10.0 months.
- Compare and contrast key nutrient intake between Māori, Pasfiika, and non-Māori, non-Pasifika infants aged 7.0 – 10.0 months.

3. Evaluate the iron status of Māori, Pasfiika, and non-Māori, non-Pasifika infants aged 7.0 – 10.0 months.

*Objectives:*

- Determine the iron status of Māori, Pasfiika, and non-Māori, non-Pasifika infants aged 7.0 – 10.0 months using body iron concentration (mg/kg body weight)
- Explore ethnic differences in the iron status of Māori, Pasfiika, and non-Māori, non-Pasifika infants aged 7.0 – 10.0 months.
- Explore intra-ethnic relationships between complementary feeding practices and iron status in Māori, Pasfiika, and non-Māori, non-Pasifika infants aged 7.0 – 10.0 months.

## Outcomes

### Primary Outcomes

Complementary feeding practices, key nutrient intake from complementary food, nutrient density of complementary food, and prevalence and predictors of iron deficiency (ID) and iron deficiency anaemia (IDA). Complementary feeding practices encompass timing of introduction to complementary foods, method of feeding (BLW versus traditional spoon-feeding (TSF)), pouch use, types of first foods offered, traditional cultural foods offered, and inappropriate drinks offered.

### Secondary Outcome

Characteristics of participants who did not provide a blood sample stratified by reason for not providing a blood sample and ethnicity.

## Hypotheses

1. A higher prevalence of non-Māori, non-Pasifika infants will use BLW.
2. There are a high proportion of infants being fed directly from pouches.
3. Iron intake will be below the estimated average requirement (EAR) for all ethnicities.
4. Māori and Pasifika infants will have a higher rate of ID and IDA than other ethnicities.

## Thesis Structure

Chapter 2, a review of the literature, follows after this chapter. This is divided into three sections, each relating to the three overall aims for this thesis. The first section examines the existing research in infant feeding practices. It begins with an overview of the history of infant feeding in NZ and current infant feeding practices, followed by a comprehensive review of the literature examining baby-led weaning, traditional spoon-feeding, traditional cultural practices, pouch use, first foods, and timing of introducing complementary foods. The second section provides a detailed rationale for the choice of key nutrients examined in the study, followed by a review of the roles and requirements of each nutrient for infants. The third section reviews infant iron status. This encompasses definitions of iron status, impacts of ID and IDA, dietary and non-dietary factors impacting iron status, and a thorough review of all previous studies in NZ investigating infant iron status.

Chapters 3, 4, and 5 are the results sections and each presents different primary outcome findings. These are presented in manuscript form. Chapter 4 presents the data on early infant feeding practices, encompassing BLW prevalence, pouch use, timing of introduction of complementary foods, and characteristics of first foods. Chapter 5 reports nutrient intake and prevalence of inadequate intake by ethnicity. Chapter 6 reports the iron status data, and the secondary outcome of predictors of iron status by ethnicity.

Chapter 6 provides an overall summary of the study findings, discussion, and conclusions. The strengths and limitations of the study are assessed, and recommendations for future research are provided.

Appendices encompass those for all chapters, information sheets, and consent forms, and additional detailed information on participant ethnicity and recruitment flow charts.

Researchers' Contribution

| Researcher | Contribution |
|---|---|
| Maria Casale<br>PhD Candidate | Jointly responsible for recruitment, participant management, data collection, data checking and cleaning, data analysis planning, and writing of all aspects of thesis including manuscripts.<br><br>Responsible for writing the thesis and manuscripts: design, literature search, and submission, and study website design and creation. |
| Professor Pamela von Hurst<br>Primary supervisor | Auckland Principal Investigator<br>Study conceptualisation and design. Review of thesis and manuscripts. |
| Professor Cathryn Conlon<br>Secondary Supervisor | Auckland Principal Investigator<br>Study conceptualisation and design. Review of thesis and manuscripts. |
| Associate Professor Kathryn Beck<br>Secondary Supervisor | Auckland Principal Investigator<br>Study conceptualisation and design. Review of thesis and manuscripts. |
| Associate Professor Lisa Te Morenga<br>Secondary Supervisor, Cultural Advisor | Study conceptualisation and design. Cultural support and advice. Review of thesis and manuscripts. |
| Professor Anne-Louise Heath<br>Co-Principal Investigator | Named Principal Investigator.<br>Study conceptualisation and design. |
| Professor Rachael Taylor<br>Co-Principal Investigator | Named Principal Investigator.<br>Study conceptualisation and design. |
| Associate Professor Jillian Haszard<br>Investigator, Biostatistician | Review of statistical analyses plans. Conducted all statistical analyses. |

Auckland Research Team

| Researcher | Contribution |
|---|---|
| Rebecca Paul<br>Auckland Project Manager | Recruitment, participant management, data collection, dietary data entry |
| Emily Jones<br>PhD Candidate | Data collection. |
| Kimberley Brown<br>PhD Candidate | Data collection, dietary data checking. |
| Andrea Wei<br>Research Assistant | Data collection |
| Rosario Jupiterwala | Data collection |

Dunedin Research Team

| Researcher | Contribution |
|---|---|
| Professor Lisa Houghton<br>Investigator | Study conceptualisation and design. |
| Dr Lisa Daniels<br>Post-Doctoral Fellow | Study conceptualisation and design. |
| Jenny McArthur<br>Project Manager, Dunedin Project Manager | Project management, recruitment, participant management, data collection. |
| Alice Cox<br>PhD Candidate | Data collection, anthropometry quality control. |
| Ioanna Katiforis<br>PhD Candidate | Data collection, dietary data entry and quality checking. |
| Madeline Gash<br>Student | Data collection. |
| Neve McLean<br>PhD Candidate | Data collection, dietary data collection quality control. |
| Elizabeth Fleming<br>Study Advisor | Dietary data |
| Professor Benjamin Wheeler<br>Study Advisor and Paediatric Endocrinologist | Interpretation of blood test data outside of the expected reference range |
| Lizzie Jones<br>Research Assistant | Dietary data entry and advisor |

# Chapter 2 | Upoko Tuarua

## Literature Review

*In Aotearoa New Zealand, the greatest inequalities in childhood outcomes related to nutrition occur due to differences in ethnicity. The purpose of this literature review is to examine the existing literature on feeding methods, nutrient intake, and iron status of infants through the lens of ethnicity.*

Taua raurau, taku raurau, ka ora te iwi

*With your food and my food, we will feed the people*

## Introduction

In Aotearoa New Zealand (NZ), Māori and Pasifika peoples carry the greatest burden of disease compared to the general population (1-3), which mirrors the health inequities faced by indigenous peoples internationally (4). Māori and Pasifika peoples from all stages of life are disproportionately represented in the majority of chronic and infectious diseases, as well as morbidity rates (2). A fundamental determinant of these gross inequities in health outcomes for Māori and Pasifika peoples is systemic racism, which manifests in healthcare polices and administration (5, 6). Compounding this, legacy racism stemming from over 160 years of European colonisation also impacts profoundly on intergenerational health, as well as social and socio-economic outcomes (7-10). Te Tiriti o Waitangi (The Treaty of Waitangi), the founding document of NZ made between the British Crown and 540 Māori Rangatira (chiefs) forms the basis of the obligations of the Crown to Māori in the context of the healthcare and disability system. However, literature suggests that there has historically been a disparate distribution of healthcare resources resulting in poorer health outcomes for Māori and Pasifika. This is due to disagreement over whether the concept of "equal citizenship rights" as stated in Te Tiriti refers to equal opportunities, or equal outcomes (11, 12). Māori and Pasifika children are held up as sacred taonga (treasures) to be protected and cared for: something kai (food) plays a central role in (13), however they are more than twice as likely as Pākehā children to grow up with significant hardships, including financial, education, and food security (14). As a result, the status quo of ethnicity being the greatest predictor of nutrition-related health outcomes remains.

## Section 1: Infant Feeding Practices

Infant feeding practices encompasses method of feeding, timing of introduction of complementary foods, and first foods offered. These practices can significantly impact the nutrient intake, absorption, and status of the infant at a crucial time of growth and development. Infant feeding practices also have the potential to affect adult food preferences and eating behaviours. This section will describe and evaluate the literature for traditional spoon-feeding, baby-led weaning, and pouch-feeding, as well as the timing of introduction of complementary foods and the types of first foods offered.

### A Brief History: Infant Feeding in Aotearoa New Zealand

How, as well as what, infants are fed has the potential to impact their eating preferences, behaviours, and health through to adulthood. Infant feeding practices in NZ and the neighbouring Pacific Islands has evolved over time. Pre-colonisation Māori exclusively breast-fed, and when the birth parent was unable to breastfeed wet nursing was a normalised practice (15). Post-colonisation breastfeeding rates declined due to industrialisation and increased authority of doctors, with a shift towards commercial feeding products which were freely marketed, and more women entering the workplace (16). Furthermore, the Native Health Act was passed in 1909 forbidding Māori women to breastfeed in public (17). Both Māori and Pacific Islanders introduced complementary foods from as young as 8 weeks, offering a pre-masticated form of traditional foods that the older tamariki and adults ate (18-20). This practice similarly changed post-colonisation with the advent of commercial baby food products, as well as the marginalisation of traditional Māori values, knowledge, and practices in favour of a western biomedical approach that favours Pākehā practices (21). As a result, this mātauranga Māori of infant feeding practices has now largely been lost in present-day NZ.

### Infant Feeding Methods: Overview

In 2003 The World Health Organisation (WHO) published guidelines recommending exclusive breastfeeding for the first six months of life, and thereafter nutritionally adequate and safe complementary foods while breastfeeding continues for up to two years of age or beyond. Where infants are unable to be breastfed, a suitable infant formula is recommended (22). The Ministry of Health (MoH) in NZ recommends that first foods consist of a thin smooth puree, and quantities and textures progress with the infant eating more family foods at around one year of age (23). This

method of feeding is colloquially known as traditional spoon feeding (TSF) and is the practice of first offering a thin smooth puree via the spoon, starting with half to two teaspoons after breast or formula feeding. Flavours, variety of foods and textures are introduced then gradually increased. Textures are progressed from a thin to thick puree, to mashed to chopped with the infant feeding themselves more as they grow older. These characteristics of TSF are well supported with robust evidence (24-27) as well as being recommended by the MoH.

An alternative and increasingly popular method of feeding is baby-led weaning (BLW), a term coined by Gill Rapley in 2005 (28). In its purest form BLW is when an infant is only offered food that they feed to themselves (29). The infant has total control over their own eating from the time of complementary food introduction (30). This sets BLW apart as its own distinct method of feeding as compared to TSF. To date, there are over 100 published studies investigating BLW. While these have identified various potential risks and benefits, many offer conflicting findings and there is not yet enough research in this area to confidently support or discourage BLW (31, 32). This is echoed by the MoH. Potential risks of BLW are choking, inadequate nutrient intake, sub-optimal iron status, impaired satiety responsiveness and reduced diet quality (33).

Baby food pouches are a novel method of feeding that has exploded onto the commercial baby food market in recent years. Baby food pouches are squeezable plastic sachets with a plastic nozzle that the food can be dispensed from onto a spoon or bowl, or directly sucked out of by the infant. Examples of food pouches available for sale in NZ can be seen in Figure 1. Pouches command a remarkable market share of 56% in the United States (34), and sales growth of 125% annually in Europe (35). In NZ it is not yet known what proportion of commercial baby food comprises of pouches.


**Figure 1.** *Baby food pouches*

There are several potential concerns regarding the use of baby food pouches, encompassing the nutritional quality of the food, appropriateness of the texture, ease of consumption leading to higher energy intake, displacement of nutrient-rich milk, effect on dental health, the impact on the infant's learning about food and eating, and social development. There has been little research into the health, nutrition, social and safety impacts of this new method of food delivery, or the prevalence of direct feeding from the pouch nozzle versus squeezing the food onto a spoon first. A paper examining novel infant feeding devices recommends that urgent research into pouches is needed (36).

**Traditional Spoon-feeding**

The transition from exclusive breast or formula milk consumed every 2 to 4 hours to solid food at around 6 months of age is an important stage. It is a time of rapid growth and development, and infants' nutritional requirements increase rapidly and can no longer be fulfilled just by milk. This is also a time where the infant's eating behaviours and food preferences are shaped – something parents have a significant influence on (37).

The WHO revised their recommendations that complementary feeding is initiated when the infant is 4 to 6 months of age (38) to approximately six months of age in 2002 (22) – advice that has been adopted by NZ and the United Kingdom (UK) (39). There is some flexibility in this guidance, as certain developmental milestones will indicate that the infant is ready. These include reduction and/or loss of the tongue extrusion reflex, the ability for the infant to sit and hold their head up, to have the gross motor skills to put objects in their mouths and chew as well as to be able to pick up objects accurately (40).

Conversely the European Food Safety Authority (EFSA) and the European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) recommend that complementary foods can be safely introduced from four to six months of age (32, 41). The risks for early introduction of solids (before four months) are agreed upon, and include immaturity of the digestive and renal systems, an increased risk of illness and infection and an immature swallow reflex resulting in an increased choking risk (42).

Chewing textured food is important for oral-motor skill development and is a difficult skill for an older child to master if the window of opportunity is missed. On the other hand, the safe oral

manipulation of first foods by an infant is an important and complex consideration. When an infant receive milk feeds, be it directly via the breast or from a bottle, the milk is dispensed directly at the posterior of the mouth which automatically triggers the swallow reflex (25, 43), as shown in figure 2.

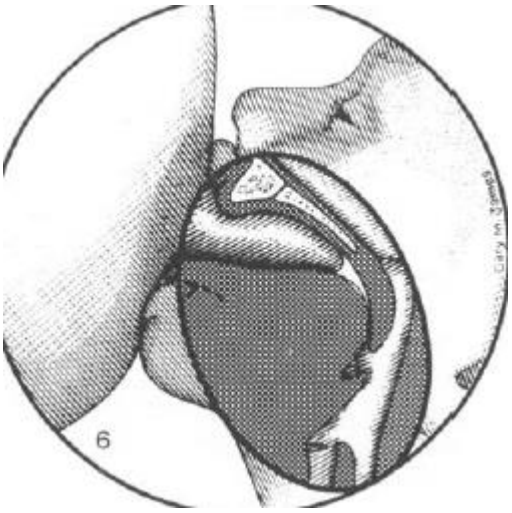



**Figure 2.** Placement of the nipple in the suckling infant

*Note:* Adapted from Bu'Lock F, Woolridge MW, Baum JD. Development of co-ordination of sucking, swallowing and breathing: ultrasound study of term and preterm infants. Developmental Medicine & Child Neurology. 1990 Aug;32(8):669-78.

However, when receiving solids from a spoon the infant must move the bolus from the front of their mouth to the back to initiate the swallow reflex. With a puree this is reasonably straightforward: the base of the tongue, which is usually positioned up to protect the airway, drops down and the bolus is propelled easily to the posterior of the mouth, through the throat, past the airway, and into the oesophagus. However, it is a more complex process for textured solids that require chewing. For this, lateral tongue movement to move the bolus around the mouth whilst chewing is required, allowing the food to be broken down and bound by saliva before being moved to the posterior of the mouth to initiate the swallow reflex (25, 43). This is made more challenging by the basic up–down, or vertical mechanism of the infants' early chewing, which is adequate only for very soft small pieces of food. More fibrous or crunchy foods must be positioned close to the ridges of the gums using lateral tongue movements, and a result of this process the airway is not well protected and there is a higher risk of choking (26, 44, 45). Additionally, the strength and stamina required to chew different foods varies greatly.

The infant's ability to safely process and break down crunchy and fibrous foods is further aided by the eruption of teeth, which typically starts between six and eight months of age, with the molars erupting between 12 and 24 months of age which coincides with the development of a rotary chewing pattern rather than a simple vertical munching action (25, 26, 43, 46).

These complex factors around safe oral manipulation help inform the TSF approach: by introducing purees as first foods the infant has the opportunity to practice moving a bolus to the back of the mouth, then progresses gradually to a mashed texture to soft foods to hard and fibrous foods, enabling the infant to develop their oral motor skills to better manage a wide variety of textures, and thus a wide variety of foods.

A common feature of TSF and BLW is the introduction of many different foods and tastes early, and early spaced introduction of common allergy causing foods. It is recommended a milk feed be offered prior to the complementary foods until they are 8-9 months of age. Caregivers are encouraged to praise their infant when they try a new food, be patient when the infant seems to dislike a food as it can take many exposures for the infant to accept a new food, and to include them in mealtimes and model enjoyable eating and social occasions (23).

**Traditional Cultural Feeding Practices in Aotearoa New Zealand and the Pacific**

*Nau te rourou, naku te rourou, ka or ate manuhiri.*
*With your food basket, and my food basket, the people will thrive*

The MoH guidelines for infant feeding in NZ include specific sections for the consideration of Māori whānau and Pasifika families. Kai (food) carries great meaning both culturally and spiritually and is about much more than purely nutrition. Mātauranga Māori holds that kai is a prized taonga (treasure) that is seen as a sacrifice from the atua (gods), and provides not just nutrition, but sustenance for the domains of Te Whare Tapa Whā: Taha Tinana (physical wellbeing), Taha Whānau (social wellbeing), Taha Hinengaro (mental and emotional wellbeing), and Taha Wairua (spiritual wellbeing). Kai has the power to connect the past, present, and future. Kai is provided to manuhiri (guests) as a sign of respect, with the quality and abundance of kai indicating the mana held by the tangata whenua in the manaakitanga (care for guests) shown.

Traditionally, the introduction of solids is rooted in intuitively following the infant's developmental cues such as sitting and grasping, rather than specific advice or timelines (47). This theme of responsive feeding carries through to the types of first foods offered and the method of infant feeding. Vegetables are commonly offered as first foods (47), and the method of feeding is not based on published guidelines for TSF or BLW, but rather carries on the responsive theme that is central to traditional care of Māori tamariki (children) (48, 49).

In Pacific Island culture, similarly to Māori culture, food represents prosperity, health, generosity, culture, tradition, and community support – over and above the role of pure sustenance. Distinguishing features of Pacific Island peoples are a strong affiliation with churches, which hold a special place in Pasifika communities, a strong affiliation with their home country, and relatively recent immigration to NZ (50). Traditional Pacific Island foods are mainly starchy foods consisting mostly of root crops (taro, yam, cassava, kumara), as well as breadfruit, banana and plantain, and coconut (51). Protein-based food consumption is lower, with the main form of protein being fish (51). Despite the import of Western foods, these traditional Pasifika staple foods are still hold a significant role in connecting families, tribes, and communities. Gifting the highest quality produce to elders, church leaders and chiefs is a sign of respect.

Not all whānau have, or have previously had, access to the tools needed to create a puree or access to commercial baby food. In many cultures adults have previously, and in some cases continue to pre-masticate food before giving it to the infant on either their finger or a utensil (18, 19, 52-56). By doing this, the food is broken down to approximately 2 mm sized pieces that are then bound together by saliva, approximating a puree that the infant can safely move to the posterior of the mouth to initiate the swallow reflex. Pre-mastication by the mother or another adult woman has been a common traditional practice in the Pacific Islands (18, 19, 52, 57) – a practice handed down from previous generations (20). The prevalence of pre-mastication amongst Māori and Pasifika whānau in NZ in the present day is not known.

The early introduction of solids has historically been a common practice for both Māori and Pasifika caregivers. (18, 19, 52). A 1994 qualitative study examining weaning practices in Tonga reports that wage-earning mothers first introduced solids at three months of age, with non-wage-earning mothers weaning at around four months of age (19). Abel et al. explored infant care practices in a 2001 qualitative study, and found that Māori, Pasifika, and young Pākehā mothers introduced solid food at three months of age, with some Pasifika caregivers reporting beginning as early as 6 – 8

weeks (18). The most common reason given for early weaning was if they felt their pēpi was not settling after feeding, and not sleeping well. Other parents explained that they felt that Pacific Island infants feel hungrier than Palangi (European) infants, so the age recommendations for complementary feeding does not apply to them (18). Growing Up in New Zealand (GUINZ) is a longitudinal study of 5770 infants and provides insight into non-timely introduction of complementary foods in NZ in 2015. This showed that 39.3% of infants were introduced to solids before 5 months of age, while another study showed that amongst a cohort of 318 NZ infants, 21.3% first had solid food between 0–3 months of age, and 31.4% between 4–5 months of age (58). Early introduction is also a more common theme with Māori infants, with 35.7% of infants in one study being introduced to solid food by three months of age, as compared to 29.6% European and 24.2% of "other" infants (59).

**Baby-Led Weaning**

Baby-led weaning has European / Western origins and is a purposeful and distinct method of introducing complementary foods to infants with clearly proposed potential health benefits. Baby-led weaning is a term coined in 2005 by Gill Rapley (28). The rationale given by Rapley for development of BLW as a distinct method of feeding is that when the general infant feeding guidelines changed in 2002 from four to six months to approximately six months, this failed to consider that the significant developmental differences between a four-month-old and a six-month-old infant. Rapley (60) suggests that:

- a six-month-old infant is developmentally ready to purposefully grasp food and bring it to their mouth
- a six-month-old infant is capable of chewing food
- there is no rationale nor research to support the use of purees or spoon-feeding for "normal" six-month old infants
- Breastmilk or formula should be the main source of nutrition up to 12 months of age, and is offered on demand, unconnected with mealtimes
- To become proficient at chewing and self-feeding, infants require frequent opportunities to practice these skills
- Infants require the opportunities to develop chewing and self-feeding skills within a critical time period of 6-9 months of age

- The limited food intake in the first couple of months of BLW should not be of concern due to obesity being one of our biggest current public health concerns

This formative publication describing the rationale and process for BLW lacks references for many of Rapley's claims, and there exists substantial evidence to the contrary (23, 24, 45, 61-64).

Rapley suggests that a six-month-old infant is developmentally ready to purposefully grasp food and bring it to their mouth. This is generally supported by infant development studies, with one finding that object size is a determinant (65), and other studies noting that vision has a prominent role in purposeful grasping of objects, with the infant tracking their hand movement in relation to the object they are aiming to grasp – a skill that develops between five and seven months of age (66, 67). While most six-months old who are developing as expected are likely able to grasp large objects, they have not yet developed the pincer grasp. Thus with BLW large pieces of food are suggested for this age (60), however Cichero (24) suggests that infants are not "born with an innate understanding of an appropriate bite size", and are likely to take much larger bites than they are able to manage, thus greatly increasing their choking risk.

It is feasible that choking risk is further heightened with BLW due to the range of textures offered from 6 months of age, including hard, fibrous, and crunchy textures. Rapley (60) states that an individual of any age who is capable of chewing does not require purees or spoon-feeding. This contrasts with research that shows the basic vertical chewing pattern and lack of lateral tongue movement in six-month old infants creates a choking risk when crunchy, fibrous, or hard textured food is ingested (24, 25, 43). Choking risk with BLW has been examined in several studies. A small survey of 92 BLW infants and 63 TSF infants reported no differences in choking incidences (68), but it was not reported if parents were educated on the difference between gagging and choking. Another study reported at least one choking episode in 32.6% of their participants (*n*=199), with 71.4% of those choking incidences caused by a whole piece of food (69). Brown et. al. conducted an observational study on 1151 infants showed that 11.9% of the strictly BLW group, 15.5% of the partial BLW group, and 11.6% of the TSF group had at least one choking episode (70). However, the authors acknowledge a potential bias with recruitment methods, resulting in a self-selected sample with more educated and older mothers, as well as participants who are well-educated about BLW.

Rapley (60) proposes that breastmilk or formula should be the main source of nutrition for an infant until 12 months of age and be offered on demand with the timing be unconnected to

complementary feeds. In contrast, the MoH and Plunket recommend that breast or formula milk feeds are recommended to be offered before a solid feed until 8–9 months, as this is the primary and most important source of nutrition until this age (23, 71). From 8–9 months, it is recommended that complementary foods are offered before a milk feed (23, 72). Breast or formula milk remains an important source of nutrition from 6–12 months, but it is not sufficient to fulfil all the infant's rapidly growing requirements (22). Brown and Lee (2013) showed a difference in the number of milk feeds between BLW and TSF infants aged 6–12 months, with BLW infants having 5–6 milk feeds per day and 70% still having night milk feeds, as compared to 4–5 milk feeds a day in the TSF group with 46% having night milk feeds (40). Another small study with 15 participants noted that mothers using the BLW approach had the attitude of "food before one is just for fun", and although they would offer finger food they were not concerned as to whether it was ingested or not (61). In addition, this same cohort reported that milk is sufficient until infants are 12 months old and would prefer their infants to receive nutrition from milk rather than solid food. This is somewhat in contrast with Rapley's claim that infants require "frequent opportunities to practice these skills" in reference to chewing and self-feeding (60), as if food is a low priority compared with milk feeds these opportunities may be scarce depending on the individual infant.

In addition to the choking risk described earlier, there are several other potential negative health outcomes associated with BLW that have been investigated to varying degrees. These are underweight related to energy intake, iron intake and status, zinc intake and status, and key nutrient intake.

Weight is both a concern and a proposed benefit with BLW. It has been noted that the BLW method impacts the infant's ability to consume enough energy which can potentially affect their weight. To date, nine studies have investigated the impact of BLW on infant weight (62, 68, 73-79), with a varying focus on either underweight as a negative outcome of BLW, or reduction of overweight as a positive outcome of BLW. Three of these studies found no difference in weight between TSF and BLW infants (73, 76, 79), however one of these studies was the Baby-Led Introduction to Solids (BLISS) study (79), a randomised controlled trial (RCT) in which a modified version of BLW was developed, where caregivers were provided with specific advice on offering high energy foods at each meal. The two other studies were observational and used parent-reported infant weight (73, 76). Five studies (four observational and one RCT) found that BLW infants had a lower weight than TSF infants (62, 68, 74, 75, 78). The focus on these studies varied between investigating prevalence of underweight in BLW infants and prevalence of overweight in TSF infants. Overweight and

underweight were classified in varying ways. One observational study (62) categorised infants as "overweight" if they were over the 85$^{th}$ percentile for infant age and gender on the WHO Child Growth Standard charts. Another observational study (68) calculated body-mass index (BMI) z-scores using the WHO growth Standards and then calculated BMI percentile ranks using Centres for Disease Control and Prevention (CDC) Child and Teen BMI Calculator and the National Health Service Choices BMI Calculator. This study found that TSF infants had a significantly higher BMI than BLW infants ($p$<0.05). Rapley herself acknowledges in her 2011 paper (60) that "many self-feeding babies appear to eat very little for the first couple of months", and that this causes concern for parents and healthcare professionals "in spite of the fact one of our biggest current public health concerns is obesity", suggesting that potential infant under-nutrition is justified due to the prevalence of adults with higher weights.

Weight is a thoroughly researched factor for Māori and Pacific Island peoples, with the research extending to infants as young as three months (59). Higher body weight as a child is proposed to be a risk factor for cardiovascular health (80), type 2 diabetes (81), mental health (82), but not educational achievement (83). A systematic review of studies examining the association between coronary heart disease (CHD) and higher BMI of people aged 2–30 years showed that BMI was positively related to CHD (80), however dietary intake and physical activity were not adjusted for. Such considerations are important given that healthy lifestyle habits are associated with significantly lower mortality rates regardless of BMI (84).

In contrast to these findings, low birth weight and malnutrition are key determinants of childhood communicable disease and adulthood non-communicable disease and are concerns for Māori and Pasifika children (85), however there is a lack of recent robust research. In 2007, more Māori infants were born small for gestational age as compared to non-Māori, with the risk positively correlated with social deprivation index (86). Subsequent weight gain in the first two years of life is more rapid than that of non-Māori, but possible causal links between low birth weight and subsequent rapid weight gain in infancy have not been well researched. Regardless of weight, the concept of passively accepting under-nutrition through BLW is unsafe and predictive of increased chances of childhood communicable disease for Māori and Pasifika infants (85).

Both iron and zinc intake and status of BLW infants is a commonly cited concern amongst healthcare professionals (31, 64). With the nature of BLW being that infants are given family foods in their whole form, iron and zinc-rich foods tend to be meat which could be more difficult for an infant to

form into a soft bolus and swallow. Foods that are easy to grasp and often offered early during BLW tend to be fruits and cooked vegetables which are typically low in iron (39, 69). A study by Morison et al. (2016) suggests that BLW infants have a lower iron (1.6 vs. 3.6 mg, $p < 0.001$) and zinc intake (3.0 vs. 3.7 mg, $p = 0.001$) than TSF infants (64). In contrast the BLISS study showed no statistical difference in zinc and iron intake between BLW and TSF infants (87, 88), however it should be noted that the BLISS BLW cohort were provided with specific advice to provide iron-rich foods, which by extension would increase the intake of zinc-rich foods. Alpers (2019) had similar findings (89), but unlike the BLISS study did not offer advice around the types of food that should be offered.

Iron and zinc status amongst BLW infants has also been a concern for healthcare professionals (31, 64), however iron intake is not predictive of iron status (90). Little is known about iron status in BLW infants. Three studies have examined iron and/or zinc status to date with conflicting findings. Hanindita et. al. (2019) found that in a cohort of 30 infants aged 9-15 months, 50% of whom were classified as BLW and the other 50% as TSF, there was a significantly higher rate of IDA amongst the BLW group than the TSF group ($p < 0.001$) (63). However, this is a very small study, and the infants were recruited from hospitalised children, although those with chronic conditions were excluded. A second study by Dogan et. al. (2018) examined 280 infants who were recruited at 5-6 months of age and had haematological parameters assessed at recruitment and again at 12 months of age. The infants were allocated into either a BLW group ($n$=142) or a TSF group ($n$=138), and at 12 months there were no significant differences in haemoglobin ($p$=0.52), transferrin saturation ($p$=0.9), or ferritin ($p$=0.96) (75). However, while this was a large prospective RCT, only breastfed infants were studied, and it is possible that formula fed infants would have a different growth trajectory. Additionally, all parents in this study were recommended to offer iron-rich foods from the start of complementary feeding. A similar but more overt approach was taken in the BLISS study, which investigated the iron status in infants who followed a version of BLW modified to prevent iron deficiency (ID) (87). The BLISS study compared 73 TSF infants and 82 modified-BLW infants, and found no significant differences in plasma ferritin, body iron, prevalence of depleted iron stores, early functional iron deficiency or iron deficiency anaemia (IDA) (all $p \geq 0.65$). However, parents in the modified-BLW group were provided with detailed support and advice on including iron-rich foods at each meal, as well as being given complimentary iron-fortified infant cereal so this is not a clear indication of the true impact of BLW in its pure form.

Given the significant disparities in nutrition-related health outcomes of Māori and Pasifika infants and children in NZ it is important to consider if the impact of BLW on dietary iron intake and iron

status in the context of indigenous health. To date there is just one study adequately examining iron status by ethnicity in NZ by Grant et. al. (2007). Iron status was investigated in 324 infants, and ID prevalence was significantly different amongst ethnicities, with 20% in Māori, 17% in Pasifika, 27% in "other", and 7% in New Zealand European (NZE) ($p$=0.005). Socio-economic status and social deprivation were ruled out as confounding variables, and infants with c-reactive protein over 4 mg/L were excluded. Low birth weight was adjusted for. Virtually no specific research on the dietary iron intake of Māori and Pasifika infants in NZ exists. Most recently the BLISS study had a control group of 77 TSF infants who did not receive dietary advice. On average, the infants dietary iron adequacy was poor (87). An earlier 2002 study conducted on European infants in Dunedin born between October 1995 and May 1996 similarly found inadequate iron intake (91). Another small South Island study in 2003 showed 15% of infants and 66% of toddlers having poor iron adequacy (92, 93). If BLW continues its trajectory in popularity and become widely adopted amongst Māori and Pasifika whānau, there could well be further impact on iron status and iron intake.

Proposed benefits of BLW that are supported by the literature are improved satiety responsiveness, reduced food fussiness, and lower parent stress around feeding. A fundamental aspect of BLW is infant self-feeding, which naturally lends itself to a responsive style of feeding controlled by the infant. This promotes satiety responsiveness in the infant, as they are in control of what and how much they choose to eat. Increased satiety and food responsiveness is associated with post-prandial glucose response (94, 95), and from a psychological perspective an improved relationship with food and eating (94-96). A longitudinal study showed that BLW infants were rated as being less fussy by their mother at 18 to 24 months than TSF infants, however when responsive feeding practices in the TSF group were adjusted for the difference in food fussiness was no longer significant, suggesting the responsive feeding aspect is responsible for decreased fussiness (62). This is consistent with other studies that show that controlling and restrictive parent feeding styles lead to increased food intake when control is lifted (97, 98), and that food fussiness increases when pressure to eat is applied (99, 100). Compounding this, controlling parental feeding practices are associated with a decreased ability of the child to regulate their appetite (101, 102).

These benefits of BLW and responsive infant feeding practices described above also mirror those of mātauranga Māori of infant feeding. Mātauranga Māori holds that infant feeding practices are spiritual, intuitive, instinctive, and natural. Māori and Pasifika people had many healthful, responsive, and whānau-centric infant feeding practices pre-colonisation. These practices were largely lost through colonisation and the subsequent decades. The health of Māori and Pasifika

children has concurrently declined, with Māori and Pasifika infants bearing an inequitable burden of disease today. The proposed benefits of BLW described above have been well researched (62, 73, 94-98), with the responsive aspect of infant feeding also being widely researched (103-106). In the present day, whānau are starting to make a return to these traditional ways of infant feeding (47). These practices are whānau-centric, with pēpi (baby(s)) sharing whānau meals of traditional kai in a modified form (pureed or finger foods). This is also seen by Māori as a way to decolonise through kai (47). Best practice for infant feeding has been widely researched; the advice signifies a return to mātauranga Māori infant feeding.

**Pouch-feeding**

Unlike TSF and BLW, pouch-feeding is not a conscious and clearly defined method of feeding with purposeful goals: rather, it is a method of feeding borne out of convenience and opportunity. The food offered in pouches is generally similar to that in baby food jars and tins, however there is concern around the nutrient content of pouches and the impact on eating behaviour and oral motor skill development. Questions around safety and environmental sustainability have also been raised. Overall, there is very little research into the impact of pouches due to their relatively recent surge onto the commercial baby food market.

Pouches have dominated the commercial infant food market in recent years but were first conceptualised and introduced in 2007 in the United States (107). Pouches have grown exponentially in popularity internationally, with one third of American infants aged 6-12 months consuming at least one pouch per week by 2016 (108). A small observational study in 2017 in the United States found that 28% of pre-school children's lunch boxes contained at least one pouch, however there were only 50 children in this study (109). Tedstone et. al. (2019) found that 35% of all commercial infant foods in the UK were packaged in pouches (110), however another exhaustive study the same year found 54% of all infant foods are packaged in pouches (111). There is scarce research investigating the prevalence of commercial infant food packaged in pouches, but as the proportion grows it is likely that the prevalence of use will also increase.

From a nutrition perspective a common theme is sugar content of pouches relative to commercial infant food packaged in tins, boxes, and jars. Sugar content of pouches has been quantified in nine studies (112-120) and compared to other commercial infant food in three (112, 117, 118). All three comparative studies found that sugar was higher in pouches than in infant foods packaged in jars,

tins, and boxes. Overall sugar content of pouches is as high as 84-98% of total energy (113, 114), which suggests that if pouches become the predominant form of packaging for commercial infant food, then sugar intake would increase.

High sugar intakes have an established negative effect on the rate of dental caries in young children, but another aspect of pouch use when the mode of delivery is direct feeding from the pouch nozzle is that the teeth and/or gums of the infant are bathed in the puree, which adheres to the surface of the tooth more easily than chewed textured foods (114). Compounding this, the acidic properties of fruit pouches further impacts the tooth enamel, thus further increasing the risk of dental caries (113). It has been suggested that weight is associated with a higher sugar intake, however a 2021 study that quantified consumption of fruit pouches in relation to BMI z-score at 18 months of age in 1499 children found that moderate fruit pouch consumption was not correlated with weight when other factors were adjusted for (121). This study looks at 18-month-old children, and the longer-term effects are not currently known. Additionally, exposure to sweeter foods may impact sweet taste preferences that can last into adulthood (113).

The pureed texture has been cited as a concern for appropriate gross oral motor skill development in infants. Many baby food pouches are marketed at infants over the age of 8 months and up to as old as 12 months (112, 117), and over 92% of pouches sold in NZ have little to no textural complexity (119). At the other end of the spectrum over 21% of pouches are also advertised as “4 months +”, encouraging the early introduction of complementary foods (114, 119).

These concerns are partially predicated on the assumption of direct feeding of the pouch via the nozzle. Textural and nutrient issues would remain regardless of the mode of delivery, but certainly other concerns could be offset by spoon-feeding the pouch. A UK-based study assessed feeding advice on pouches and found that a third of pouches did not provide feeding advice on the label (122). It is not currently known how caregivers are feeding their infants when using pouches in NZ.

There are many concerns and questions around the use of commercial infant food pouches, some of which have started to be investigated as previously described, and others that remain to be elucidated – particularly around social aspects of feeding and the impact on the child’s relationship with food and eating. Additionally, the impact of the explosion of pouches onto the market may have significant implications for Māori infants. One study in 2007 showed that commercial infant food is offered more frequently than home-made food and is an established risk factor for IDA in

Māori infants (58), however this is conflicted by a more recent small qualitative study showing a general distrust of commercial infant foods by Māori whānau (47). There is no available research on use of commercial infant foods by Pasifika families, but Pasifika infants would have a similar risk profile to that of Māori if the use of commercial infant food is similar.

### Ethnic Prevalence Within Feeding Methods

Research into ethnic prevalence within feeding methods is lacking in NZ, with brief insights from two observational studies (69, 76). Fu et. al. (2018) conducted an online survey and analysed responses from 876 parents in NZ who were recruited at random: 72% used TSF, 18% followed full BLW, and 11% followed partial BLW (76). Within the full BLW group, 77% were NZE, 15% Māori, and 3% Pasifika. Similar numbers appeared in the partial BLW group: 78% NZE, 15% Māori, and 5% Pasifika. The average age of the child was 16.9 months.

Cameron et. al. (2013) conducted a similarly designed study of 199 participants aged 6-7 months, and found that within the BLW group, 64.7% were NZE, 0% Māori, 0% Samoan, with "other" ethnicities (excluding Indian, Chinese, and English reported on individually) grouped together with a prevalence of 5.9%. Numbers were slightly higher in partial BLW group: 76.2% were NZE, 9.5% Māori, 0% Samoan, and 7.1% "other" (69).

This indicates that while BLW in NZ initially appeared to be a primarily Pākehā phenomenon, there may be increasing uptake by Māori, and to a lesser extent Pacific Island whānau in NZ.

### First Foods and Timing of Introduction

The recommendations for the timing of introduction to complementary foods has evolved over time. In the 1960s infants were first exposed to complementary foods at 8 weeks of age on average, with most being introduced by 4 months of age (123, 124). In the 1970s it was recommended to wait until after 4 months of age, based on an assumption that early exposure to gluten was responsible for an increase in the incidence of coeliac disease (125). With a rising awareness and prevalence of allergy diagnoses in the 1990s it was recommended to wait until after six months to commence solid foods (126). It now agreed that the introduction of solids before four months of age provides a significant risk for gastrointestinal, respiratory disorders, and increased weight gain (127-131), while late introduction (after six months) increases the risk of food allergies. The current recommendation for

the introduction of complementary foods is at around six months of age when the infant is showing developmental signs of readiness (22, 132).

Early introduction of complementary foods is a common practice amongst Māori and Pasifika whānau in NZ. The GUINZ study provides insight into non-timely introduction of complementary foods in NZ. A 2022 paper showed that 40.2% of infants introduced early at ≤ 4 months of age, and 3.2% introduce late (≥ 7 months) (133). This cohort were stratified by ethnicity, showing that 57.3% of Māori and 45.6% of Pasifika infants were introduced to solids early (≤4 months), compared to 35.9% of European infants. An earlier 2007 study showed that amongst a cohort of 318 NZ infants, 21.3% first had solid food between 0-3 months of age, and 31.4% between 4 and 5 months of age (58), however this was not stratified by ethnicity.

An earlier 2018 paper from the GUINZ study showed that 62.6% of NZE parents, 39% of Māori mothers, and 49.4% of Pacific Island parents were introducing solids at around 6 months of age, and that Māori ethnicity (vs European ethnicity) is predictive of early introduction to solid foods (134). Furthermore, the prevalence of infants offered solid foods prior to the age of 4 months has not changed significantly since 2006/2007 (135). Two qualitative studies exploring Māori whānau beliefs around infant feeding identified that guidelines for introduction of complementary foods are rigid and not applicable to Māori infants (18, 47). This is echoed by research done with Pasifika families, showing that solid food is introduced around three months of age, and although some mothers report starting solids as early as 6 to 8 weeks of age (19, 52), little literature supports this claim. The main reason for early weaning is due to mothers needing to return to their employment (19). Another reason for early weaning was that some parents felt that Pacific Island infants feel hungrier than Palangi (European) infants, so the age recommendations for complementary feeding do not apply to them (18).

Given that Māori and Pasifika infants carry an inequitable burden of childhood illnesses, particularly lower respiratory tract infections (136), the common practice of early food introduction in these population groups may be one contributing factor. Whether it is the early introduction of solid food, or early cessation or reduction in breast milk that is implicated in the increased rate of infections has been studied. Forsyth et. al. (137) investigated the independent effect of early introduction to complementary foods in a developed country and found no significant association between early introduction and gastrointestinal illnesses. However, there was an increased rate of respiratory illnesses associated with early introduction of complementary foods, which persisted when a

smoking household and low socioeconomic conditions were adjusted for. It was not clear if the respiratory symptoms were atopic in nature, or due to recurring infections. A seven-year follow-up study of this same cohort suggested that breastfeeding and later introduction of complementary foods may have a favourable effect on the probability of respiratory illnesses during childhood. It is therefore likely that both the early feeding and the subsequent cessation or reduction in breastfeeding play independent roles in increased childhood respiratory illnesses.

The types of first foods offered are also an important factor in health outcomes. Infant iron stores are depleted by about six months of age, so it is important that iron-rich foods are offered early, along with a variety of fruits and vegetables to ensure the infant is receiving a range of fruit and vegetables (132). Common first foods for all ethnicities in NZ are pureed fruits, vegetables, and soft cereals (18, 133). A 2022 paper from the GUINZ study showed the prevalence of food groups introduced early were breads and cereals (36.3%), iron-rich foods (34.1%), and fruits and vegetables (23.8%) (133).

Traditional cultural Māori and Pasifika values around first foods have been described in several qualitative studies. Giving whole foods, such as fruits and vegetables grown by the whānau or extended whānau, as first foods is a traditional cultural practice for both Māori and Pasifika whānau (18, 47). Older generations of Māori and Pasifika parents and grandparents typically do not approve of commercial infant foods and consider fresh fruit and vegetables to be optimal first foods (18). Common traditional Pasifika first foods include sua alaisa (coconut and rice soup) for Samoan ‘aiga, pia (arrowroot) and mokomoko (coconut milk) for Cook Island whāmere (18). Adapting whānau meals for Māori and Pasifika infants is another common traditional practice where either cooking methods and foods are adapted so they are suitable for pēpi, or a small amount of the food for the main meal being placed in a separate pot without salt or other inappropriate additives, and then cooked and pureed for pēpi, thus assimilating pēpi into whānau foods from the start of complementary feeding (47).

The early infant feeding practices described in this section can have far-reaching effects on the health and wellbeing of infants – effects that can be life-long. A shift for Māori whānau to traditional cultural feeding practices has started to emerge which may help redress the current health inequities faced by this vulnerable group (47). Less is known about the present-day prevalence of traditional cultural infant feeding practices in Pasifika communities. Understanding the nutrient intake of infants alongside current infant feeding practices helps provide a more complete picture of

the nutritional status of our infants in NZ. The next section is dedicated to describing the current evidence for key nutrients for infants.

## Section 2: Key Nutrient Intake for Infants

The traditional approach to infant feeding by Māori and Pasifika whānau is a holistic approach involving many interconnected behaviours, rituals, and traditions. Indeed, this is also true for many cultures and varies the world over. As such, the examination of individual nutrients cannot always be extrapolated to specific health outcomes, and the bigger picture should be considered. However, understanding intake of specific nutrients provides a solid foundation for understanding the overall nutritional status of infants, and helps identify opportunities for improving the infant's nutrition status.

Appropriate adequate nutrition is a vital aspect of an infant's growth and development in their first year of life. From 6 months of age, infants require enough essential nutrients through consumption of complementary foods to support their rapid growth. Proportionally, the nutrient requirements of an infant per kilogram of body weight is greater than that of any other stage in the life cycle (138), consequently complementary foods need to be nutrient dense. The average energy intake of breast-fed weaning infants is lowest between 6 and 8 months of age at ~837 kJ/day (139), and increases from 9-11 months (139). As a result of the lower average energy intake from 6-8 months, first foods need to have a high nutrient density to ensure the infant is able to meet all of their nutritional requirements.

Whilst all nutrients have important roles and are necessary for optimal health, some nutrients can be harder for infants to obtain – such as iron and zinc which are found in tougher textured foods, and are particularly important for the infant's rapid growth and development (140).

Internationally, the literature examining infant nutrient requirements is largely focussed on nutrients that present public health challenges worldwide, and this has informed the choice of nutrients examined in the present study. Dietary energy, whilst not a nutrient, is included in this review, as it is crucial for the physiological needs of the infant (141). Vitamins A, D, C, B12, folate, thiamine, riboflavin, and niacin deficiencies have been identified internationally in the context of socio-economic hardship (140). Similarly, deficiencies of phosphorus, magnesium, iron, zinc, iodine, fluoride, and selenium have also been identified in a public health context internationally (142). Deficiencies of vitamins E, K, pantothenic acid, biotin, calcium, copper, molybdenum, chromium and manganese do not present a public health concern as they are only found in severe disorders or as side effect of medical treatment (140).

In NZ, the nutrient deficiencies that pose a public health concern are energy, protein, fat, carbohydrate, iron, fluoride, iodine, selenium, zinc, and vitamins A, D, $B_{12}$ (85, 140, 141, 143, 144). Whilst calcium and vitamin C are not nutrients of concern in terms of deficiencies, they act as absorption modifiers for iron and are therefore important to consider (141). Dietary fibre on the other hand does not have a recommendation for infants under 12 months, although it can impact energy intake and nutrient absorption (145-147). Vitamin D comes primarily from non-dietary sources, while fluoride is mostly obtained from water and toothpaste, so evaluating these nutrients in the context of dietary intake is likely to give misleading information about the infant's true status. Iodine, folate, and selenium are also challenging to assess with FoodWorks data not providing reliable data for these nutrients. For this reason, the following section focusses on energy, protein, fat, carbohydrate, iron, zinc, calcium, and vitamins A, $B_{12}$ and C.

**Nutrient Recommendations: Definitions**

The recommendation for a given nutrient is based on the amount of the nutrient that must be consumed regularly to maintain the good health of an otherwise healthy individual, with the assumption that all other energy and nutrient requirements are also met (148). The Recommended Dietary Intakes (RDI) were developed for each nutrient in 1991 (149, 150), and are defined as "the levels of intake of essential nutrients considered, in the judgement of the National Health and Medical Research Council (NHMRC), on the basis of available scientific knowledge, to be adequate to meet the known nutritional needs of practically all healthy people" (149). When developing the RDIs an extensive range of factors affecting absorption and metabolism were considered, and while they are appropriate to apply to groups, they are not appropriate for assessing an individual's dietary needs. As a result, a set of values for each nutrient has been developed in the UK and by the Food and Nutrition Board: Institute of Medicine (FNB:IOM) to help prevent widespread misuse of the RDIs in relation to individuals (151-157). In 2006, the Working Party for the NHMRC established comprehensive recommendations for nutrient intake for Australia and NZ (141). The recommendations aim to not just achieve adequate sustenance but to also reduce chronic disease risk. The Working Party for the NHMRC adopted the FNB:IOM guidelines, but adapted the terminology – in particular, retaining the "Recommended Dietary Intake" term (141). The adapted definitions are summarised in table 1.

**Table 1.** Adapted definitions from FNB:IOM for dietary recommendations

| Terminology | Definition |
|---|---|
| EAR (Estimated Average Requirement) | Daily nutrient level estimated to meet the requirements of half of the healthy individuals in each life stage and gender group. |
| RDI (Recommended Dietary Intake) | Average daily dietary intake of a nutrient that is sufficient to meet the requirements of 97-98% of healthy individuals in each life stage and gender group. |
| AI (Adequate Intake) (used when an RDI cannot be determined) | Average daily intake of a nutrient based on observed or experimentally determined approximations or estimates of nutrient intake by a group(s) of apparently healthy people that are assumed to be adequate. |
| EER (Estimated Energy Requirement) | Average dietary energy intake predicted to maintain energy balance in healthy adults of a specific age, gender, weight, height, and level of physical activity that is consistent with good health. |
| UL (Upper Limit) | The highest daily intake of a nutrient that is likely to pose no adverse health effects to almost all individuals in the general population. |

*Note.* Reproduced from National Health and Medical Research Council. (2006). *Nutrient Reference Values for Australia and New Zealand: including recommended dietary intakes* (Version 1.1 updated March 2017 ed.): National Health and Medical Research Council.

When developing estimated average requirement (EAR) values, all available evidence for each criterion is critically evaluated with a rationale provided for the final choice (158). The EAR implies a median rather than a mean and is appropriate for research purposes as it is better applied to groups than individuals. As the EAR will be inadequate for 50% of a given group, any individual in a group who is consuming the EAR for a nutrient has a 50% chance of their intake being inadequate. The EAR data was extrapolated for use in infants aged 7-12 months using a reference body weight of 9 kilograms and a growth factor of 0.3.

The RDI value of a nutrient is calculated using the EAR. An RDI, which refers to the average daily intake of a nutrient that will meet the needs of the majority of healthy individuals within a set life stage or gender group is set at 2 standard deviations (SD) above the EAR, assuming that an SD is available, and the EAR data is symmetrically distributed (159). In the case of no SD being available

for the EAR a coefficient of variation (CV) is used. Typically, this is set at 10% for the EAR unless data supporting greater variation exist (160, 161), thus the RDI would be 1.2 x EAR (141).

In the case of insufficient or conflicting evidence for establishing an EAR (and thus an RDI), an AI is established. This is determined using either experimental evidence, or by assessing recent median population intake with the assumption that Australian and New Zealand populations are not deficient in the nutrient at hand. AIs are susceptible to greater errors as they rely upon greater subjective assessment and may differ greatly to the RDI if an RDI was able to be set.

An upper limit (UL) has been determined for nutrients where applicable, and reflects the highest daily amount of a nutrient an individual can have without risking adverse health effect, and is applicable to almost all of the general population (141).

In estimating the estimated energy requirements (EER) for infants the equations used are those used by the Food and Nutrition Board when the dietary reference intakes (DRI) values for the United States (US) and Canada were established (141). The DRIs are a set of reference values used to assess and plan the nutrient intakes for healthy people. Total energy expenditure (TEE) was determined in 14 doubly-labelled water studies in infants (162), which took into consideration age, gender, body weight and length. Physical activity level categories were not used, but requirements for growth were included in the total energy estimate using estimates from a study examining the energy content of tissue deposition (163) along with the assumption of a 50$^{th}$ centile for weight gain (141). The relevance of these calculations to infants in NZ is important to consider, given the ethnic diversity. Māori and Pasifika adults have a different pattern of tissue deposition with higher bone, fat, and muscle mass than their European or Asian counterparts (164-166), and while it is not known if this can be extrapolated back to infancy, it is well established that Māori and Pasifika infants tend to be heavier than NZE (59, 167, 168). This would have an impact on the EER of Māori and Pasifika infants.

## Dietary Energy and Nutrients: Roles and Recommendations

### Energy

Dietary energy results from the oxidation of macronutrients and is used for all metabolic and physiological functions, including the growth and synthesis of new tissue, heat production and muscular activity (141, 148). Basal metabolism and physical activity use the most energy, and

encompass all bodily functions necessary for life, including cell metabolism, enzyme and hormone synthesis and metabolism, substance transport, organ function, and homeostasis. The amount of energy required for these processes differs by age, gender, body size and body composition. The rapid growth of infants in the first three months of life uses 35% of energy, which falls to 5% by 12 months (169). Infants require three times as much energy per kilogram of body weight than adults (141), with inadequate energy intake leading to faltering growth, impaired cognitive development, and increased risk of disease (170, 171). The impact of BLW and pouch feeding on infant energy intake is described in section 1 of this literature review.

The recommendation for energy for infants in Australia and NZ is calculated by age in months and is summarised in table 2.

**Table 2.** Estimated Energy Requirements (EER) of Infants Aged 6-12 months

| **Age (months)** | **Reference weight (kg)** | | **EER (kJ/day)** | |
|---|---|---|---|---|
| | *Boys* | *Girls* | *Boys* | *Girls* |
| 6 | 7.9 | 7.2 | 2700 | 2500 |
| 7 | 8.4 | 7.7 | 2800 | 2500 |
| 8 | 8.9 | 8.1 | 3000 | 2700 |
| 9 | 9.3 | 8.5 | 3100 | 2800 |
| 10 | 9.7 | 8.9 | 3300 | 3000 |
| 11 | 10.0 | 9.2 | 3400 | 3100 |
| 12 | 10.3 | 9.5 | 3500 | 3200 |

*Note.* Reproduced from Lupton, J. R., Brooks, J., Butte, N., Caballero, B., Flatt, J., & Fried, S. (2002). Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. *National Academy Press: Washington, DC, USA*, *5*, 589-768. Reference weights from Kuczmarski RJ. CDC growth charts: United States. US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; 2000.

## Protein

Proteins are long chains of amino acids which have both functional and structural roles. There are 20 amino acids, nine of which are essential – meaning they cannot be synthesised by the body. Amino acids not only act as the building blocks of proteins, but also the precursors for hormones, enzymes, and other essential molecules (141). Dietary protein can be obtained from plant and animal sources, and although all essential amino acids can be gained from both, different sources of protein are considered higher quality than others. The bioavailability and relative load of essential amino acids, along with digestibility are the primary determinants of protein quality (140). Protein sourced from animals, such as meat, fish, poultry, eggs, and dairy products contain all nine essential amino acids

and are considered high quality, while plant-based protein sources are usually deficient in one or more essential amino acids, so a vegetarian or vegan diet needs to ensure a wide range of plant-based proteins to ensure all essential amino acids are obtained (140).

Unlike fat and carbohydrate, protein does not act as an energy store. As a result, if there is insufficient protein intake, as can occur in severe disease or fasting, energy needs take priority and protein is lost. If this loss becomes prolonged and significant then muscle loss, including cardiac tissue, occurs resulting in protein-energy malnutrition (PEM). This can also occur if the type of amino acids is imbalanced, compromising protein metabolism. PEM is uncommon in infants and children in NZ and tends to be associated with severe disease and older adults (141), but low intake of protein in early childhood can result in stunted growth (172).

The recommendations for Australia and NZ use AI as no RDI was able to be established for protein in infants. Infants aged 0-6 months require 1.43 g/kg body weight per day (BW/day), while 7–12-month-old infants require 1.60 g/kg BW/day (141). Table 3 shows total protein requirements using standard reference weights. These guidelines differ from those established by FNB:IOM for the USA and Canada (173), who use an EAR of 1.0 g/kg BW/day, and have established an RDA (recommended daily allowance, the equivalent of RDI in the US and Canada) of 1.2 g/kg BW/day (173). The rationale for the NHMRC recommendations was based on the protein concentration in breast milk (11 g/L) multiplied by the average consumption of breast milk (0.6 L/day), whilst allowing for an additional 7.1 g/day from complementary foods.

**Table 3.** Protein Requirements of Infants Aged 6-12 months

| **Age (months)** | **Reference weight (kg)** | | **Protein AI (g)** | |
|---|---|---|---|---|
| | Boys | Girls | Boys | Girls |
| 6 | 7.9 | 7.2 | 11.30 | 10.30 |
| 7 | 8.4 | 7.7 | 13.44 | 12.32 |
| 8 | 8.9 | 8.1 | 14.24 | 12.96 |
| 9 | 9.3 | 8.5 | 14.88 | 13.6 |
| 10 | 9.7 | 8.9 | 15.52 | 14.24 |
| 11 | 10.0 | 9.2 | 16.00 | 14.72 |
| 12 | 10.3 | 9.5 | 16.48 | 15.2 |

*Note.* Reference weights from Kuczmarski RJ. CDC growth charts: United States. US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; 2000.

## Fat

Fat is a highly concentrated source of energy at 37 kJ/g, as compared to protein and carbohydrate at 16.7 kJ/g and is required for the absorption of fat-soluble vitamins (A, D, E, K) (141). Fat occurs in three major forms: saturated, *cis*-monounsaturated, and *cis*-polyunsaturated. Hydrogenation of polyunsaturated fats also results in *trans* fatty acids (140). Saturated fat is found primarily in animal products such as meats, dairy, palm oil and coconut oil, and monounsaturated fatty acids (MUFA) in nuts, avocado, seeds, as well as peanut, olive, and canola oils (140). Saturated fats and MUFAs can be synthesised by the body, so are not considered essential (174). Polyunsaturated fatty acids (PUFA) are classified further as either 'n-3' or 'n-6', depending on the position of the double bonds. The n-3, or omega-3, PUFAs are unable to be synthesised by the human body and are therefore considered essential and must be obtained via the diet (141). Food sources of omega-3 PUFAs are oily fish, legumes, canola oil, walnuts and in very small amounts in leafy vegetables (174).

Dietary fats primarily play a role in the regulation of cell membrane fluidity and are essential for the infant's developing brain and retina. Fat also helps support rapid weight gain and growth in the first year of life (140, 174). Trans fatty acids have a harmful affect as they not only increase the atherogenic low-density lipoprotein (LDL), but decrease high-density lipoprotein (HDL) which is important for the reduction of LDL (140). An increased LDL and decreased HDL results in an altered ratio of total cholesterol to LDL, which is a significant risk factor for cardiovascular disease (CVD) (174). Dietary n3-PUFAs have an important role in the reduction of coronary heart disease, are required as structural membrane lipids — particularly in developing retinal and nerve tissue — and give both anti-inflammatory and cardiovascular benefits (175-183).

The recommendation for fat intake uses AI, and is classified by total fat, n-6 PUFAs and n-3 PUFAs, and is summarised in table 4. The AI was established by multiplying the average intake of breast milk with the average concentration of total fat, n-6 and n-3 PUFAs in breast milk, then adding the median intake of each type of fat from complementary food using data from the US Continuing Survey of Food Intake by Individuals (CSFII) (141, 155).

**Table 4.** Dietary Fats Recommendations for Infants Aged 7-12 Months

| Fat | Recommendation (AI) |
|---|---|
| Total fat | 30 g/d |
| n-6 polyunsaturated fat | 4.6 g/d |
| n-3 polyunsaturated fat | 0.5 g/d |

*Note*: Adapted from National Health and Medical Research Council 2006. *Nutrient Reference Values for Australia and New Zealand: including recommended dietary intakes*, National Health and Medical Research Council.

## Carbohydrate

Carbohydrate can be classified as either digestible or indigestible. Digestible carbohydrates are monosaccharides (glucose, fructose, galactose), disaccharides (lactose, sucrose, maltose) and polysaccharides (plant starch) (141, 148). Indigestible carbohydrates are soluble and insoluble fibre (140). Carbohydrate metabolism begins in the mouth with salivary amylase and is primarily absorbed in the small intestine in the case of digestible carbohydrates, and via bacterial fermentation in the large intestine for oligosaccharides and prebiotics, resulting in short-chain fatty acids which are processed in the liver in the case of acetate, or used directly as energy metabolism by colonocytes in the large intestine in the case of butyrate (140).

Dietary carbohydrates primarily provide energy in the form of glucose for cells and the brain which requires glucose for metabolism (140, 141, 148). This is of particular importance for infants as their brain is large relative to the size of their body (184). Carbohydrates also carry other important nutrients such as fibre and B vitamins. Although glucose can also be provided through gluconeogenesis, a pathway that is well developed even in premature infants (185), it has not been established whether this is sufficient to meet the glucose requirements of infants (141).

There has previously been no RDI or AI set for carbohydrate for Australia and NZ infants, and this is mirrored in the USA and Canadian guidelines, as well as the European Union guidelines, which is a reflection of the lack of data on essentiality (141, 148, 162). Therefore, the average carbohydrate content of breast milk (74 g/L), the average daily intake of milk (0.60 L/day) and a further 51 g/day from complementary food, based on findings from the National Health and Nutrition Examination Survey (NHANES) III (155) have been used to determine an AI of 95 g/day for 7-12 month old infants.

## Iron

Iron is a trace mineral that is critical for the healthy growth and development of an infant and has several important physiological functions, including haemoglobin synthesis and oxygen transport (140). Iron also has an essential role in the development of the nervous system, with ID in infancy being associated with poor behavioural outcomes as well as learning and memory deficits that may persist into adolescence (186). Other functional outcomes from ID include reduced attention span, reduced exercise capacity, impaired body temperature homeostasis, and impaired immune function (140).

Iron in breast milk is more easily absorbed (49% absorbed (187)) than that from infant formula and is sufficient until approximately 6 months of age, at which point iron-rich complementary foods are required to meet infants' iron requirements (188). Haem iron, found in animal products, is much more bioavailable (15-35% absorbed) than non-haem iron (2-20% absorbed) which is found primarily in plant-based foods (141, 189). The amount of non-haem iron that is absorbed can vary by 1-40%, depending on the meal composition (154). Ascorbic acid and the so-called MFP factor (meat, fish, poultry factor) enhance the absorption of non-haem iron, whilst phytate, polyphenols, and calcium inhibit iron absorption through the formation of complex unabsorbable compounds (141, 154, 188).

An EAR of 7 mg/day and an RDI of 11 mg/day has been set for iron for 7–12-month-old infants in NZ (141). In setting the EAR the requirements for absorbed iron at the 50$^{th}$ centile was used in modelling, with an upper limit of 10% iron absorption applied. The RDI was established by estimating the requirement for absorbed iron at the 97.5$^{th}$ centile, also with an upper limit of 18% absorption applied (141). These recommendations are based on a mixed western diet that includes animal foods. If an infant is vegetarian or vegan, they have about a 10% iron absorption rate (141), and will need a higher intake which has not been specifically set. The UL of safe iron intake for infants aged 0-12 months is 20 mg/day. This was set by extrapolating from the lowest level of iron intake at which adverse effects are observed to the intake level where possible detrimental growth outcomes are observed (190).

## Zinc

As with iron, complementary foods that are zinc-rich are required from the start of complementary food introduction to ensure the infant's zinc requirements are met. Zinc plays a role in immune function, as an antioxidant, in the structural integrity of proteins, and gene expression (191-193). Zinc deficiency in infants presents as faltering growth, impaired immune function, altered

gastrointestinal function, impaired cognitive development, alopecia, skin lesions, and loss of taste and appetite (194, 195).

Dietary zinc is found in a wide variety of foods, with meat, fish and poultry being the richest sources. Grains and dairy products also contain a significant amount of zinc. The bioavailability of zinc is affected by the dietary components of the meal. Protein binds zinc, and even a small change in protein digestion can cause a significant difference in zinc absorption (196). Furthermore, zinc in human milk is much more bioavailable and forms soluble compounds in the small intestine compared with cow's milk and soy milk based infant formulas, which have a much lower rate of zinc bioavailability (197, 198). It is likely this is due to the phytic acid content, as phytates inhibit zinc absorption through the formation of large molecular weight compounds (199). Indeed, a vegetarian or vegan diet that has a ratio of 15:1 or greater of phytate to zinc has a 50% higher zinc requirement (141).

An EAR for zinc of 2.5 mg/day, and an RDI of 3 mg/day has been set for infants aged 7-12 months in NZ. In setting the EAR for infants the adult needs were extrapolated and growth needs were factored in. Additionally, the complexity of zinc absorption was considered, with the recommendation providing enough absorbable zinc to offset intestinal losses in a typical Australian and New Zealand diet (141). A CV of 10% for the EAR was used to set the RDI, as no data was available on the SD of the requirement, and it is acknowledged that vegetarian and vegan infants will need higher intakes, although what this should be is not formally established. An upper limit of zinc intake has been set at 5 mg/day and applies to total zinc intake including supplements and fortified food (141). No adverse effects from zinc that occurs naturally in foods has been found (141).

## Calcium

Calcium is abundant in human bodies, and is stored in bone from gestation onwards, with approximately 80% of calcium accumulated from 24 weeks gestation till term (200). Calcium is essential for normal bone development and maintenance, cardiac and muscular function (200), and blood calcium is under homeostatic control. Due to the majority of calcium accrual occurring later in gestation, pre-term infants are at risk of calcium deficiency (201, 202). In the first 6 months of life calcium needs of a term infant are met by breast milk or formula, but requirements exceed that provided by milk in the second 6 months of life, with food sources of calcium becoming increasingly important (202). Inadequate calcium intake, along with inadequate vitamin D, in infants can result in

rickets (201). The proportion of calcium deposition in bone in the first year of life is at least equal to, or higher than, any other one-year period in life (202). This likely makes an important contribution to the attainment of peak bone mass as an adult which is protective of osteoporosis and fractures in later life (141).

Calcium is present in high amounts in dairy, and in smaller amounts in bony fish, legumes, some nuts, and fortified foods (141). Calcium bioavailability is impacted by the phytate and oxalate content of a meal, so infants who eat a vegetarian diet may have higher requirements for calcium to overcome this (141, 203). Infants who eat a vegan diet get less calcium than vegetarian or omnivore infants (204, 205), but it is possible for vegan infants to get enough calcium through non-dairy sources (203), which may include fortified alternatives. The intestinal absorption of calcium supplementation (through tablets or in fortified food products) is similar to that of calcium from dairy products (206-209), but supplementation doses of over 500mg saturates the active transport mechanism with minimal further calcium absorbed (210, 211).

Calcium recommendations for infants have been based off the concentration of calcium in breast milk. For infants aged 7-12 months, an AI of 270 mg/day was established by estimating 126 mg/day of calcium from breast milk at this age (212) and adding it to an estimate of 140 mg/day of calcium from complementary foods (213, 214), then rounded (141).

## Vitamin A

Vitamin A is crucial for normal vision, reproduction, and immune function, and appears as retinol, retinoic acid, and retinyl ester. Vitamin A also includes dietary precursors of retinol (carotenoids, in particular β-carotene which the body converts to vitamin A) and is expressed as retinol equivalents (RE). Retinol is involved in epithelial cell structure (215) and immune function maintenance (216-218), while retinoic acid is involved in gene expression and is important for normal embryonic development (219). Retinoic acid is also required for vision through the maintenance of the rod and cone cells and is protective against xerophthalmia (220). Adequate vitamin A intake is associated with lower morbidity and mortality in developing countries, and protective in children with serious illnesses such as measles in developed countries (221).

Carotenoids are found in abundance in dark leafy green vegetables as well as some orange or yellow-coloured fruit and vegetables, such as kumara, pumpkin, carrot, and fruits such as mango,

orange, and apricot. Pre-formed vitamin A is found in animal-based food products such as liver and eggs which are the richest sources, as well as dairy, and some fatty fish. (222).

The AI for RE for infants aged 7–12 months is set at 430 µg/day. This was calculated by multiplying the average intake of breast milk by the concentration of retinol in breast milk, with an additional 244 µg from complementary food. An UL for vitamin A as retinol of 600 µg/day has been set for 0-12 months and is based on reports of hypervitaminosis A (141). No UL for β-carotene has been set, as it is not necessary to set a UL due to the metabolic conversion of β-carotene being under homeostatic control via vitamin A status (141). Excessive supplemental β-carotene of greater than 20 mg/day has been associated with an increased risk of lung cancer in smokers and individuals exposed to asbestos, but not enough data is available to set a specific limit (223, 224).

## Vitamin $B_{12}$

Vitamin $B_{12}$ is essential for the synthesis of fatty acids and DNA (alongside folate), and healthy neurological and blood function (141). A severe deficiency in infants results in various neurological symptoms, including failure to thrive, anorexia, behavioural difficulties, and developmental regression, with the mechanism thought to involve delayed myelination, demyelination of neurons, lactate accumulation, and an imbalance of neurotoxic cytokines (225, 226).

Vitamin $B_{12}$ is found almost exclusively in animal-based foods, which is the primary source for humans. These include meat, dairy, eggs, shellfish, and fish (227-229). Milk and dairy products are the most common source for children, followed by red meat (230, 231). Certain edible algae are rich in vitamin B12, and dried green and purple algae are commonly used to make nori (229). Despite the concentration of vitamin $B_{12}$ in these specific algae, Dagnelie et. al suggest the bioavailability is very low (232). In animal-based foods the bioavailability of vitamin B12 varies and typically ranges from 42-89% depending on the food source, with the rate of intestinal absorption decreasing as dietary intake increases (229). It is suggested that the saturation point for intestinal absorption is reached at 1.5-2.0 µg per meal. Additionally, intestinal absorption is dependent on intrinsic factor (IF) which is secreted in the stomach, and can be impaired by infections such as helicobacter pylori (233). Deficiency in infants can be linked to the mother's diet during pregnancy, and low levels in breast milk which is also related to maternal status and diet (234-236).

An AI of 0.5 µg/day has been set for infants aged 7-12 months. This was extrapolated from the AI for 0–6-month infants, which was established based on the average intake of breast milk for this age

group. The vitamin B12 concentration in breast milk was multiplied by the average intake of breast milk then rounded. As the concentration in breast milk can be variable depending on the mother's vitamin B12 status and intake, a value was taken from a study examining nine well-nourished mothers in Brazil, that found a concentration of 0.42 μg/L at 2 months. The concentration dropped to 0.34 μg/L at 3 months, but the 2-month value was chosen to ensure adequate intake (141). It is recommended that vegan mothers take a vitamin B12 supplement throughout pregnancy and lactation that meets the adult RDI of 2.4 μg/day, otherwise the breast-fed infant will require supplementation from birth (141).

## Vitamin C

Vitamin C (ascorbic acid) is an essential nutrient, as it cannot be synthesised by the human body (237). Ascorbic acid is a redox modulator that also maintains the redox homeostasis of other antioxidants in the brain, an electron donor for several enzymes, and is protective against N-nitroso compound formation in gastric juices. It has also shown as protective against oxidative damage to lipids present in human plasma in *ex vivo* studies (238), but there is no evidence for *in vivo*. Ascorbic acid assists in the intestinal absorption of iron and copper (239, 240), maintenance of reduced glutathione (241), sparing of α-tocopherol (242), and maintenance of folate (243). Vitamin C has been well investigated for its role in viral infections, including COVID-19 and acute respiratory distress syndrome, with convincing evidence for a protective function (244-248). Insufficient vitamin C in infants is associated with increased oxidative neuronal damage thereby impacting brain development (249). Intakes of <8 mg/day of vitamin C causes scurvy (250), which presents in infants as pseudo-paralysis of the limbs and lesions at sites of active bone growth (251).
Vitamin C is present in a wide range of fruits and vegetables – kiwifruit, citrus, broccoli, and blackcurrants are particularly rich sources. However, vitamin C is a very labile nutrient and can be impacted by cooking, heat, bruising, storage conditions, season, and transport (141). Research investigating the impact of commercial packaging such as jars, tins, and baby food pouches on vitamin C content showed the bio-availability of vitamin C is very low at between 0.3 and 26.3% as compared to 70 to 90% for a usual intake from whole fruits, vegetables, and supplements (252). Absorption from supplements falls to 50% with doses over 1 g/day (253).

An AI of 30 mg/day for infants aged 7–12 months has been set. The rationale for the recommendation is clinical scurvy has not been observed in exclusively breast-fed infants, even when maternal vitamin C intake is low (141, 254). Breast milk concentration ranges from 30 mg/L to

80 mg/L (255, 256). To obtain the AI, the lower end of breast milk concentration of vitamin C (30 mg/L) was multiplied by the average intake of breast milk per day (0.78 L/day) and then rounded. This was then used to calculate the AI for 7-12 months on a body weight basis (141).

**Dietary Assessment Methods**

Obtaining an accurate and reliable estimation of the complementary diet of infants is the foremost priority when assessing nutrient intake. Methods for gathering and assessing dietary intake vary in quality, so it is important to evaluate the method used carefully. Factors to consider with infants are that dietary patterns and behaviours change rapidly during infancy, the feeding responsibility may be shared amongst multiple adults, and not all food offered to an infant is necessarily consumed. Additionally, different data collection methods may be more accurate depending on the nutrients being examined (257). Assessment methods include estimated dietary records (EDR), food frequency questionnaires (FFQ), weighed dietary records (WDR), and single or repeated 24-hour dietary recalls (24h-recall).

EDRs may be subject to recall bias, especially when considering that any uneaten food could be spread over several surfaces by the infant and be difficult to quantify from memory. Although the WDR gives a greater participant burden, a 2010 systematic review reported that a WDR in infants aged 6 months to 4 years was found to provide the best estimate of total energy intake (258). Another systematic review found that a 3-day WDR was more accurate than an FFQ for assessing the micronutrient intake of infants.

FFQs for infants may be useful to assess nutrient intake and are commonly used, but this is dependent on the quality and detail in the information collected (257, 259). There has been a wide variation in the number of food items in FFQ validation studies, ranging from 7-191 items, with a systematic review suggesting that FFQs are unable to identify ID, and FFQ validation studies in infants only gave good correlations for vitamin C (257). Judd et. al. (2020) conducted a complementary FFQ (CFFQ) validation for infant nutrient intake in NZ, which showed acceptable agreement with a reference 4-day WDR, and good reproducibility (260). Lovell et. al. (2016) conducted a systematic review assessing the validity of FFQs and found that ten showed good correlation for dietary assessment in children aged 12-36 months of age. However, weighed food records and 24-hour recalls have a greater precision than FFQs (261).

Weighed diet records create a high participant burden which can result in a poorer quality response, or an increased rate of attrition and incomplete data. With young children and infants, this may be further complicated by the number of adults involved in providing food for the child, particularly if they attend a childcare centre. Furthermore, weighing leftover food may present a particular challenge, especially if pureed or wet foods are in the child's hair, clothes, or on the floor. It has been estimated that approximately 10% of the food served to children under the age of four years is left behind (262).

24h-recalls give less participant burden, and correlate well with WDR for micronutrients, although unstructured 24h-recalls overestimate energy, fat, protein, and sugar (259). The 24hr-recall encapsulates all dietary intake over a 24-hour period, and includes detailed information about the food, recipes, cooking methods, beverages, and timing of eating. Props such as cup or spoon measures, visual aids, and measuring aids can be used to further enhance the quality of the information obtained. A multiple-pass method for 24hr-recalls is now widely used, (263, 264)and helps prompt for forgotten foods or other details. A second non-consecutive recall allows better assessment of usual intake by adjusting for day-to-day variation (265).

The validity of 24-hour recalls was assessed in a recent study (266). Kittisakmontri et. al (2021) compared a single 24-hour recall to a 3-day weighed food record in infants aged 9-12 months and reported acceptable to excellent correlation levels (r = 0.37-0.87) (266). However, the validity of some micronutrients, particularly vitamin A, were not as reliable – but this would likely be improved upon with a second 24-hour recall. The feasibility of a multiple-pass 24-hour dietary recall method in preschoolers was also assessed by Trolle et. al. (2011). The authors determined that this method was best used in conjunction with a food record book for recording food when the child is at a childcare centre, or out of the home, or with other adults, and that the interview be accompanied by props such as cups, spoons, and photos of common items (267).

A less commonly used method of assessing the nutrient intake for infants is assessing the nutrient density of the complementary diet. Further developing this idea, a reference nutrient density, or "critical nutrient density" can be established (268). If an infant's diet meets the critical nutrient density, this means that the required nutrients will be ingested so long as the required energy is adequate. Nutrients of concern are then highlighted by any gaps between the nutrient density of the diet and the critical nutrient density. Arguably, it is appropriate to modify the complementary diet,

rather than the milk feeds, thus a focus on the nutrient density of the complementary diet helps direct where interventions are needed (268, 269).

Nutrient density has been most commonly used to assess the infant's diet in developing countries where nutrient-sparse gruels and other grains are commonly introduced first foods for infants (270-272). Nutrient density was also used to examine Canadian infants' complementary diet, suggesting a role for this method in both developed and developing countries (273, 274).

An alternative to nutrient density is the individual dietary diversity score, a core indicator for assessing diet quality and adequacy. This is a simplified method to measure and assess the quality of an individual's diet but may not be as precise as a 24-hour recall (275). Additionally, infants have the highest requirements for nutrients per kilogram of body weight than at any other life stage, which may weaken the accuracy of an individual dietary diversity score when examining the infant's complementary diet.

From a Kaupapa Māori perspective, it is important to consider the relevance and adequacy of these tools. The Heart Foundation identified particular challenges with FFQs in terms of cultural appropriateness (lack of culturally diverse foods), health literacy, and ease of completing FFQs (276). It is possible that a 24-hour recall may be able to be better tailored be culturally sensitive and supportive, however little research in this area has been identified. A method of dietary evaluation informed by mātauranga Māori, Kaupapa Māori research methods and validation spanning hinengaro (mental), tinana (physical), wairua (spirit), and whānau (family) using whānaungatanga (respectful collaborative relationships) should ideally be used when gathering and assessing dietary data for Māori.

**Key Nutrient Intake of Infants in NZ**

As described in the previous section, methods for gathering and assessing dietary intake vary in quality and type and typically rely upon recall which provides challenges when comparing findings from different studies. Additionally, evidence for intake of key nutrients in NZ infants is sparse. The 2002 National Children's Nutrition Survey (currently the most recent data available) focuses on children aged 5-14 years. It may be possible to assume that this data can be extrapolated to infant intake, but the data is two decades old. At the time of the writing, a government-run national nutrition survey encompassing infants is in the planning stages. At present, four studies give some

insight into the nutrient intake of NZ infants but lack ethnic diversity (64, 91, 260, 277). These studies are described in detail below.

An examination of the control group data from the BLISS study in 2015 consisted of 77 infants living in Dunedin who received no dietary input. A 3-day WDR was analysed at 7 and 12 months and is shown in table 5 (87, 88, 278). Nutrient intake was not stratified by ethnicity in the published papers.

**Table 5.** Nutrient intake from weighed diet records from the BLISS control group

| **Nutrient** | **Recommendation†** | **7 months** ***n*=77** | **12 months** ***n*=70** |
|---|---|---|---|
| Energy (kJ) | 2500-3500 | 2831 (2728, 2938) | 3373 (3179, 3580) |
| Protein (g) | 14* | 16.3 (15.2, 17.5) | 28.5 (26.3, 30.9) |
| Protein (% energy) | Not set | 9.8 (9.4, 10.2) | 14.4 (13.7, 15.1) |
| Total fat (g) | 31 | 33.2 (32.1, 34.3) | 33.0 (31.0, 35.0) |
| Saturated fat (g) | Not set | 14.8 (14.2, 15.4) | 15.3 (14.3, 16.3) |
| Total CHO (g) | 95 | 78.0 (74.1, 82.1) | 99 (92, 105) |
| Total CHO (% energy) | Not set | 46.8 (45.9, 47.9) | 49.7 (48.4, 51.0) |
| Dietary fibre (g) | Not set | 2.6 (2.2, 3.2) | 7.3 (6.5, 8.1) |
| Vitamin C (mg) | 30 | 59.1 (53.9, 64.7) | 49.4 (44.5, 54.8) |
| Vitamin B12 (μg) | 0.4-0.5 | 0.5 (0.4, 0.6) | 1.1 (1.0, 1.3) |
| Calcium (mg) | 270 | 399 (365, 435) | 556 (502, 616) |
| Iron (mg) | 11 (EAR 7) | 2.7 (1.3, 6.9) | 5.3 (3.1, 8.4) |
| Zinc (mg) | 3 (EAR 2.5) | 3.5 (2.7, 4.8) | 4.4 (3.6, 5.7) |
| Sodium (mg) | 170 | 223 (204, 243) | 666 (613, 722) |

*Note.* Adapted from Baby-Led Introduction to Solids (BLISS) study: a randomised controlled trial of a baby-led approach to complementary feeding by L Daniels *et al.* 2015, *BMC Pediatrics, 15*(1), 1-15. Values are presented as median (25$^{th}$ percentile, 75$^{th}$ percentile).
*Based on 1.6g/kg body weight
†Australian NZ Nutrient Reference Values (141).

Although the above data cannot be extrapolated to the general population due to the small sample size, it indicates that infants in this study were receiving adequate energy, protein, fat, vitamin C, calcium, and zinc. Conversely carbohydrate intake at 7 months of age and iron intake at 7 and 12 months were inadequate, and sodium intake is high. The iron intake reported is especially concerning, as it is well below 50% of the RDI. As previously mentioned, this is a small sample size, but it does indicate an urgent need for updated and larger scale research in this area.

An earlier 2002 study examined dietary energy, iron, zinc, calcium, and vitamin C intake of 68 healthy term European infants in Dunedin at 9 months (91). The authors reported energy, iron, and zinc intake as inadequate as compared to the UK Reference Nutrient Intakes. Examining the intake against the current Australia New Zealand Nutrient Reference Values show inadequate iron intake only (table 6).

**Table 6.** Nutrient intake from estimated diet records of infants aged 9 months

| **Nutrient** | **Recommendation*** | **Actual intake (9 months)** *n*=68 |
|---|---|---|
| Energy (kJ) | 2800–3100 | 3284 (2763, 3868) |
| Calcium (mg) | 270 | 542 (385, 684) |
| Iron (mg) | 11.0 | 7.0 (3.7, 10.7) |
| Zinc (mg) | 3.0 | 4.0 (3.3, 5.1) |
| Vitamin C (mg) | 30 | 52 (39, 73) |

*Note.* Adapted from Heath ALM, Reeves Tuttle C, Simons MS, Cleghorn CL, Parnell WR. Longitudinal study of diet and iron deficiency anaemia in infants during the first two years of life. Asia Pacific journal of clinical nutrition. 2002;11(4):251-7.
Values are presented as median ($25^{th}$ percentile, $75^{th}$ percentile).
*Australia New Zealand Nutrient Reference Values (141).

This study had a small non-ethnically diverse sample. Dietary data was collected via an EDR and included breast and formula milk feeds. Mothers were provided with detailed written instructions for recording the infants' dietary intake, and they were probed for any missing detail when the EDR was collected. Formula milk feeds were recorded and included in the analysis; however, breast milk intake was estimated, using the average intake for infants at 9 months of age. Given the small sample size and lack of ethnic diversity the findings cannot be extrapolated to the general population.

A small cross-sectional study compared the dietary intake of 51 infants aged 6-8 months living in Dunedin and Auckland following BLW with those following TSF (64). Infants were classified as either European (65%) or "other" (35%). The nutrient intake was stratified by feeding method and is summarised in table 7.

**Table 7.** Nutrient intake from weighed diet records of infants aged 6-8 months

| | | | BLW | |
|---|---|---|---|---|
| **Nutrient** | **Recommendation*** | **TSF (*n*=26)** | **Partial (*n*=7)** | **Full (*n*=18)** |
| Energy (kJ) | 2500 (girls) 2800 (boys)** | 2897 (2718 – 3088) | 3073 (2382 – 3115) | 2800 (2518 – 3115) |
| Protein (g) | 14 | 17 (15 – 19) | 18 (14 – 19) | 15 (12 – 17) |
| Total fat (g) | 30 | 33 (31 – 35) | 36 (33 – 39) | 36 (33 – 39) |
| Saturated fat (g) | - | 14 (13 – 16) | 16 (15 – 18) | 17 (15 – 18) |
| Carbohydrate (g) | 95 | 82 (75 – 90) | 86 (70 – 105) | 72 (64 – 82) |
| Fibre (g) | - | 3.6 (2.2 – 5.8) | 3.7 (2.1 – 6.3) | 2.0 (1.2 – 3.4) |
| Iron (mg) | 11 | 3.6 (2.7 – 4.9) | 3.3 (1.3 – 8.0) | 1.6 (1.2 – 2.1) |
| Zinc (mg) | 3.0 | 3.7 (3.3 – 4.1) | 4.0 (2.9 – 5.4) | 3.0 (2.6 – 3.3) |
| Vitamin C (mg) | 30 | 66 (57 – 76) | 67 (53 – 86) | 46 (38 – 55) |
| Vitamin B12 (μg) | 0.5 | 0.5 (0.3 – 0.8) | 0.6 (0.3 – 1.1) | 0.2 (0.1 – 0.3) |
| Calcium (mg) | 382 | 382 (352 – 436) | 437 (311 – 616) | 318 (290 – 349) |
| Sodium (mg) | 170 | 235 (200 – 275) | 235 (171 – 323) | 232 (178 – 302) |

*Note.* Adapted from Morison BJ, Taylor RW, Haszard JJ, Schramm CJ, Williams Erickson L, Fangupo LJ, et al. How different are baby-led weaning and conventional complementary feeding? A cross-sectional study of infants aged 6-8months. BMJ Open. 2016;6(5). Values are presented as mean (SDs).
*Australia New Zealand Nutrient Reference Values (141).
**EER for infants 7 months of age

The sample size in this study was very small, non-random, and had mostly NZE participants (77%), with all other ethnicities being classified under “other”. A non-consecutive three-day WDR was used to obtain the dietary data which was collected over two weekdays and one weekend day. Accurate scales were provided to all participants to ensure consistency. Breast milk intake was assumed. Although the study was well-designed, the sample size is too small and non-diverse to extrapolate to the general population.

A 2019 validation of a complementary food frequency questionnaire conducted a four-day weighed food record for 95 infants aged 9-12 months living across NZ, who were primarily NZE and Asian (table 8) (260). Breast milk and formula were included in the dietary analysis. The four-day weighed food record shows very inadequate intake of iron, with slightly low vitamin E, fat, and carbohydrate.

**Table 8.** Nutrient intake from weighed diet records of infants aged 9-12 months

| Nutrient | Recommendation* | Four-day weighed record |
|---|---|---|
| Energy (kJ) | 2800–3500 | 3295 ± 810 |
| Protein (g) | 13.6–16.48 | 26.6 ± 8.8 |
| Fat (g) | 30 | 33.2 ± 8.7 |
| Carbohydrate (g) | 95 | 92.7 ± 31.2 |
| Fibre (g) | - | 9.3 ± 4.5 |
| Calcium (mg) | 270 | 478 ± 207 |
| Iron (mg) | 11 | 1.5 ± 0.6 |
| Zinc (mg) | 3 | 4.8 ± 1.9 |
| Potassium (mg) | 700 | 1257 ± 405 |
| Iodine (µg) | 110 | 49.1 ± 29.6 |
| Selenium (mg) | 15 | 23.7 ± 7.8 |
| Vitamin C (mg) | 30 | 64.6 ± 33.6 |
| Vitamin E (mg) | 5 | 4.8 ± 2.2 |
| Folate (µg) | 80 | 137.8 ± 74.4 |
| Thiamine (mg) | 0.3 | 0.8 ± 0.5 |
| Riboflavin (mg) | 0.4 | 0.9 ± 0.5 |
| Niacin (mg) | 4 | 6.1 ± 3.0 |
| Vitamin B12 (µg) | 0.5 | 1.3 ± 0.9 |

*Note.* Adapted from Judd AL, Beck KL, McKinlay C, Jackson A, Conlon CA. Validation of a Complementary Food Frequency Questionnaire to assess infant nutrient intake. Maternal & child nutrition. 2020;16(1):e12879. Values are presented as mean ± SD.
*Australia New Zealand Nutrient Reference Values (141).

Using a non-consecutive four-day WDR to assess the nutrient intake of infants is a robust and well-supported method for obtaining dietary data. There were some limitations to this study, with the authors acknowledging that there was no standardisation of scales as participants had the option of using their own which may introduce inaccuracies. There were 95 infants who completed this study, which was just 51% of those who expressed interest. This suggests the burden of a four-day weighed record is too high. There was a risk of self-selection bias as recruitment took place via social media and community groups. There was also a lack of ethnic diversity, and it is unlikely these results can be extrapolated to the general population.

Māori and Pasifika infants were not recruited in adequate numbers to stratify by ethnicity in the above studies and was not part of their aims, thus very little is known about nutrient intake in these groups.

The GUINZ study does not look at specific nutrients but does provide some insight with their investigation of the adherence to infant nutrition guidelines. These indicate that Māori and Pasifika infants have low rates of adherence to several guidelines (279):

- Exclusive breast feeding till around 6 months of age
- Eating fruit and vegetables twice or more daily at 9 months of age
- Inappropriate foods never tried at age of 9 months (sweets, chocolate, hot chips, crisps)
- Inappropriate drinks never tried at age of 9 months (coffee, cordial, juice, tea, soft drinks)

This suggests that important nutrients primarily found in fruit and vegetables such as folate, vitamin C and vitamin A, as well as those reliant upon these nutrients for absorption, such as iron, may be lacking in in the diet of Māori and Pacific infants. However, this needs to be more clearly investigated.

These brief insights into the current nutrient intake of NZ infants suggest iron intake is consistently inadequate, with intakes ranging from 1.5 – 7 mg/day across all four studies described above (*n*=367) (64, 91, 260, 277). Vitamin B12 intake was inadequate in the full BLW group of one study, at 0.2 µg/day (*n*=18), but adequate in the partially BLW and TSF groups (64). Iodine, vitamin E, and carbohydrate intake were all low in one study using a four-day weighed food record (*n*=95). The overall findings for all four studies described above are summarised in table 9.

**Table 9.** Comparison of infant nutrient intake in NZ

| Nutrient | Heath et. al. (2002) | Daniels et. al. (2015) | | Morison et. al. (2016) | | | Judd et. al. (2019) |
|---|---|---|---|---|---|---|---|
| | 9 months | 7 months | 12 months | TSF 6-8 months | Partial BLW, 6-8 months | BLW, 6-8 months | 9-12 months |
| Energy (kJ) | 3284 (2763, 3868) | 2831 (2728, 2938) | 3373 (3179, 3580) | 2897 (2718 – 3088) | 3073 (2382 – 3115) | 2800 (2518 – 3115) | 3295 ± 810 |
| Protein (g) | - | 16.3 (15.2, 17.5) | 28.5 (26.3, 30.9) | 17 (15 – 19) | 18 (14 – 19) | 15 (12 – 17) | 26.6 ± 8.8 |
| Fat (g) | - | 33.2 (32.1, 34.3) | 33.0 (31.0, 35.0) | 33 (31 – 35) | 36 (33 – 39) | 36 (33 – 39) | 33.2 ± 8.7 |
| Saturated fat (g) | - | 14.8 (14.2, 15.4) | 15.3 (14.3, 16.3) | 14 (13 – 16) | 16 (15 – 18) | 17 (15 – 18) | - |
| Carbohydrate (g) | - | 78.0 (74.1, 82.1) | 99 (92, 105) | 82 (75 – 90) | 86 (70 – 105) | 72 (64 – 82) | 92.7 ± 31.2 |
| Fibre (g) | - | 2.6 (2.2, 3.2) | 7.3 (6.5, 8.1) | 3.6 (2.2 – 5.8) | 3.7 (2.1 – 6.3) | 2.0 (1.2 – 3.4) | 9.3 ± 4.5 |
| Calcium (mg) | 542 (385, 684) | 399 (365, 435) | 556 (502, 616) | 382 (352 – 436) | 437 (311 – 616) | 318 (290 – 349) | 478 ± 207 |
| Iron (mg) | 7.0 (3.7, 10.7) | 2.7 (1.3, 6.9) | 5.3 (3.1, 8.4) | 3.6 (2.7 – 4.9) | 3.3 (1.3 – 8.0) | 1.6 (1.2 – 2.1) | 1.5 ± 0.6 |
| Zinc (mg) | 4.0 (3.3, 5.1) | 3.5 (2.7, 4.8) | 4.4 (3.6, 5.7) | 3.7 (3.3 – 4.1) | 4.0 (2.9 – 5.4) | 3.0 (2.6 – 3.3) | 4.8 ± 1.9 |
| Sodium (mg) | - | 223 (204, 243) | 666 (613, 722) | 235 (200 – 275) | 235 (171 – 323) | 232 (178 – 302) | - |
| Potassium (mg) | - | - | - | - | - | - | 1257 ± 405 |
| Iodine (µg) | - | - | - | - | - | - | 49.1 ± 29.6 |
| Selenium (mg) | - | - | - | - | - | - | 23.7 ± 7.8 |
| Vitamin C (mg) | 52 (39, 73) | 59.1 (53.9, 64.7) | 49.4 (44.5, 54.8) | 66 (57 – 76) | 67 (53 – 86) | 46 (38 – 55) | 64.6 ± 33.6 |
| Vitamin E (mg) | - | - | - | - | - | - | 4.8 ± 2.2 |
| Folate (µg) | - | - | - | - | - | - | 137.8 ± 74.4 |
| Thiamine (mg) | - | - | - | - | - | - | 0.8 ± 0.5 |
| Riboflavin (mg) | - | - | - | - | - | - | 0.9 ± 0.5 |
| Niacin (mg) | - | - | - | - | - | - | 6.1 ± 3.0 |
| Vitamin B12 (µg) | - | 0.5 (0.4, 0.6) | 1.1 (1.0, 1.3) | 0.5 (0.3 – 0.8) | 0.6 (0.3 – 1.1) | 0.2 (0.1 – 0.3) | 1.3 ± 0.9 |

There is a clear need for an adequately powered study to provide an accurate insight into the current nutrient intake for infants in NZ stratified by ethnicity. Given that ethnicity is the greatest predictor of nutrition-related health outcomes in NZ this will help build much better understanding around intra-ethnic nutrients of concern.

## Section 3: Infant Iron Status

One third of the world's population is affected by anaemia, with half of all cases due to iron deficiency (280). Iron, arguably the most studied of nutrients for infants, is critical for healthy development of the central nervous system and motor skills (281, 282). Iron status can be determined through various biochemical indices and is complex to measure (188). Studies also use different indices and cut-offs, which makes comparison of iron status across populations challenging. The effects of IDA on infant motor and neurodevelopment have been extensively studied (283, 284). Iron status can be affected by several factors –dietary intake (93, 285-287), infection and inflammation (288), low birth weight (289, 290), low socio-economic status (291-293), genetics, physiological factors, and ethnicity (58, 92). The evidence for maternal iron status on infant iron status is inconclusive (294). The prevalence of ID and IDA amongst infants and toddlers in NZ is between 4 and 20% (58, 92, 295), with Māori and Pasifika infants bearing the burden of the highest rates of ID (58). However, there is a paucity of evidence and most available studies are somewhat limited by their design.

Dietary iron occurs in two forms: haem iron, found in meat, poultry and seafood, and non-haem iron, found in both meat and plant-based foods. Haem iron is highly bioavailable and readily absorbed in the small intestine, whilst non-haem iron is less easily taken up by the body (141, 189). Infant formula is fortified with iron, and while iron concentration in breastmilk is lower, it contains lactoferrin which enables very high rates of iron absorption (296). There are several dietary modifiers of iron absorption. Calcium and phytates are inhibitory, with cereals being high in phytate (297). Cereals are a commonly consumed infant food, and are often fortified with iron, thus some of the iron fortification may be offset by the phytate content. Ascorbic acid enhances non-haem iron absorption by forming a soluble chelate with it in the acidic conditions of the stomach (298). The chelate remains soluble in the alkaline conditions of the duodenum where the iron is absorbed. Ascorbic acid is also able to reverse the inhibitory effects of phytates and calcium (299).

### Iron Status Definitions

Defining iron status can be complex due to the varying biochemical measures available, with no single test available (300), and different markers are often used in conjunction depending on the individual to assess iron status. Iron status falls on a continuum, from replete through to iron-

deficiency anaemia. Commonly used markers with varying cut-offs are haemoglobin (Hb), ferritin, and soluble transferrin receptor (sTfR) (280). Other assays can also be useful in certain circumstances, such as chronic illness, where the commonly used indices could be confounded. These are serum iron, serum transferrin concentration, or total iron-binding capacity which is used to identify transferrin saturation.

Haemoglobin concentration is required to diagnose anaemia – where the number of red blood cells which are required for oxygen transportations are insufficient (301). Anaemia can result from vitamin A, folate and $B_{12}$ deficiencies, inflammation, parasitic infections, and Hb synthesis disorders but is most commonly a result of ID (301). Other factors affecting Hb concentrations are age, sex, ethnicity, altitude, smoking, and pregnancy (301). Due to these biological and physiological variations Hb is not sufficient on its own to diagnose IDA. Varying thresholds are used worldwide to define anaemia, and differ depending on age, sex, and pregnancy. The WHO defines anaemia in infants and children aged 6 months to 4 years of age as an Hb of less than 110 g/L – a figure extrapolated from older children. In NZ, Starship Children's Hospital uses the same cut-off of <110 g/L as the WHO (302), conflicting with the lower cut-off used by Southern Community Laboratories (SCL) of <105 g/L. The SCL reference range is in line with the recommendation of <105 g/L by the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN) (285).

Serum or plasma ferritin is a commonly used marker of iron stores and is highly specific in the absence of inflammation. Ferritin is an acute-phase protein and is raised regardless of iron status in inflammatory disorders, liver disease, chronic kidney disease, and malignant disease (303). The WHO recommend that the inflammatory markers C-reactive protein (CRP) and α-1-acid glycoprotein (AGP) are tested to rule out any inflammatory confounders (304). CRP rapidly changes in response to acute infection and inflammation, whilst AGP responds more slowly but is sustained for longer than CRP (305). Statistically, inflammation may be adjusted for, for example on a continuous scale by the BRINDA (Biomarkers Reflecting Inflammation and Nutritional Determinants of Anaemia) method (306, 307). Methods for adjusting for the effect of inflammation and infection in studies is to exclude those with inflammation, stratification by level of inflammation, or adjusting the ferritin concentration in the presence of elevated CRP or AGP. Similar to Hb, there are varying thresholds for ferritin cut-offs vary. The WHO recommendation is that a ferritin concentration of <12 µg/L is suggestive of low iron stores in children under 5 years of age, which is similar to that of ESPGHAN, who give a range of <10–12 µg/L. In NZ the Southern Community Laboratory has a higher cut-off of <15 µg/L for very young children (0–2 years), while Starship Children's Hospital use a cut-off of <10

µg/L (302). It has been suggested that increasing the ferritin cut-off to 30 µg/L improves the diagnostic accuracy in children, with the sensitivity of a cut-off at 12 µg/L being 25% compared to 30 µg/L being 92% (308).

Serum sTfR has the advantage of not being affected by inflammation but can be raised in conditions that upregulate erythropoiesis (haemolytic anaemia, chronic lymphocytic leukaemia) (309). Raised sTfR has also been demonstrated with vitamin $B_{12}$ and folic acid deficiency regardless of iron status (310). The number of transferrin receptors are increased during ID to help regulate iron levels. The transferrin receptors on the cell membranes undergo proteolysis, from which the sTfR are produced, so increasing ID is correlated with increasing sTfR. It is a highly specific and sensitive measure, with a meta-analysis showing a sensitivity of 86% and a specificity of 75% for sTfR (311), however standardised reference ranges have not been established worldwide. The receiver operator characteristics curve approach for establishing sTfR cut-offs in data analysis is considered reliable in studies (312, 313), and body iron — the ratio of sTfR and ferritin — is also a highly reliable method for determining tissue ID. Body iron is assessed using the following formula (314):

$$\text{Body iron (mg/kg)} = -\frac{\left[\log 10 - \left(\frac{\text{sTfR} \times 1000}{\text{ferritin}}\right) - 2.8299\right]}{0.1207}$$

This index corresponds directly to tissue ID, with a positive value indicative of anaemia due to a tissue iron deficit, and a negative value indicating any anaemia is likely due to chronic disease. Body iron has been used to determine ID in the NHANES survey (315) but has only been validated for use in adults at present (285).

Other measures of iron status that may be useful used in conjunction with other indices are serum iron, total iron-binding capacity (TIBC), transferrin saturation, zinc protoporphyrin concentration, mean cell volume (MCV), and mean cell haemoglobin (MCH). Serum iron alone is not overly useful as it is subject to recent dietary intake, and has considerable variation hour–to–hour and day–to–day (285). Transferrin saturation, the ratio of serum iron to transferrin is a better marker than serum iron alone, and decreases in the earlier stages of ID (285), but the sensitivity of this marker is not high at 61% (316). Zinc protoporphyrin concentration in erythrocytes and the ratio of zinc protoporphyrin to haem concentrations may indicate ID (300, 317), but has a low specificity (318).

Zinc is used as an alternative substrate for protoporphyrin binding during haem synthesis when iron is not available, resulting in a high amount of circulating zinc protoporphyrin – however, increased concentrations can also be suggestive of infection, inflammation, lead poisoning, haemolytic anaemia, and in haemodialysis (280). Reduced MCH corresponds to hypochromia, and decreased MCV is suggestive of microcytic anaemia, but only tend to be abnormal with long-term ID (280, 319). Lowered MCV is also an indicator for α-thalassaemia, which is present in approximately 15% of Pacific Islanders in NZ (302).

### Stages of Iron Deficiency

Iron status is categorised into three distinct stages: iron depletion, iron deficiency without anaemia (early functional iron deficiency), and iron deficiency anaemia (320). Progression through the stages of ID can be caused by one, or a combination of several factors: inadequate dietary intake, poor iron absorption, and increased iron losses. Confounding factors such as chronic disease and inflammation make determining the stage of ID challenging, and a multi-parameter model in which multiple indices are used is required to identify the stage of ID. The WHO recommend using Hb, ferritin, and sTfR to identify the stage of ID (table 10), however body iron described earlier allows for inflammation, and has been used in children (315).

**Table 10.** Progression of iron status by biochemical indices

| **Marker** | **Normal iron status** | **Stage 1:** *Iron Depletion* | **Stage 2:** *Early functional iron deficiency* | **Stage 3:** *Iron Deficiency Anaemia* |
|---|---|---|---|---|
| Ferritin | Normal | Decreased | Decreased | Decreased |
| sTfR | Normal | Normal | Increased | Increased |
| Haemoglobin | Normal | Normal | Normal | Decreased |

*Note.* Adapted from Suominen P, Punnonen K, Rajamäki A, Irjala K. Serum transferrin receptor and transferrin receptor-ferritin index identify healthy subjects with subclinical iron deficits. Blood, The Journal of the American Society of Hematology. 1998 Oct 15;92(8):2934-9.
Abbreviations: sTfR – soluble transferring receptor

**Stage 1**, iron depletion, is measured by ferritin and occurs when the body's physiological need for iron is not met. At this stage, other markers for ID are not affected but unless iron supply is addressed then ID will continue to progress to the next stage (301).

**Stage 2**, early functional iron deficiency without anaemia, is reflected by an increase in sTfR concentration but haemoglobin remains within the normal range (301). As with iron depletion, if iron supply to the tissues is not increased, ID will continue to progress and Hb will begin to drop.

**Stage 3**, iron deficiency anaemia, is indicated by a low Hb concentration due to decreased erythropoiesis (301). Other nutritional deficits, as well as some diseases can also result in low Hb, so multiple biochemical markers are needed to identify the cause of anaemia (280). These markers may include serum ferritin, sTfR, serum iron, and inflammatory markers. Symptoms of IDA can vary, but most commonly are paleness, fatigue, dyspnoea, and headache (280).

### Impact of ID and IDA on Infants

ID and IDA have several functional consequences for infants and young children, which are summarised briefly in table 11. Infants' risk for ID is greatest from 6 – 12 months of age, coinciding with a critical time of rapid growth and brain development (321). Infants with ID are at risk in both the short-term and long-term for poor cognitive, motor, behavioural, and neurophysiologic development (322). Infants living in low socio-economic areas, those in ethnic minorities, and those who are immigrants are at increased risk for ID (323).

**Table 11.** Functional consequences of iron deficiency

| |
|---|
| Reduced cognitive development |
| Reduced attention span |
| Poor learning and impaired academic performance |
| Decreased exercise stamina |
| Reduced muscular force and strength |
| Impaired body temperature regulation |
| Immune deficiencies |
| Impaired immune function |

*Note:* Adapted from Koletzko B, Bhatia J, Bhutta ZA, Cooper P, Makrides M, Uauy R, et al. Pediatric nutrition in practice: Karger Medical and Scientific Publishers; 2015.

The impact of ID on cognition and behaviour in infants has been extensively studied. In six case-control studies the mental development index test scores of children under 2 years of age were on average 6 – 15 points lower for those with IDA than those without (324-329). Four of these studies

also demonstrated decreased psychomotor development index (324-326, 329). All of the above studies controlled for socio-economic status and chronic illness. Recent reviews in 2007 and 2019 provide further support for these findings (330, 331). In addition to the effect on cognition, ID impacts infant and child behaviour. Social-emotional behaviours (wariness, hesitancy, solemnness, unhappy, clingy to parent) were markedly different in infants with IDA than those without (332). Neurophysiological differences have also been observed and summarised in a 2006 review (332), including speed of auditory neural transmission (333, 334), rapid eye movement in sleep (335), memory processing (336), and electroencephalogram frontal asymmetry (337). All the preceding studies examine the impact of ID in infancy and very young children, but the effects are far-reaching through to childhood and adolescence.

An Israeli study examined the developmental quotient (DQ) at 2 years (*n*=873) and intelligence quotient (IQ) at 3 years (*n*=373), and 5 years (*n*=230) of age, after Hb was initially determined at 9 months of age (338). Socio-economic status, birth weight, sex, and the mothers' education were controlled for. The authors found an increase of the DQ and IQ with an increase in Hb, and a correlation between Hb at 9 months of age and IQ at 5 years, at which point every 10 g/L increment of Hb corresponded with an increase in 1.75 points in the IQ score (338). Similar findings were published in a Yugoslavian study, where children were followed from birth to 4 years (339). Iron status was assessed at 6, 12, 18, and 24 months (340), and at 3 and 4 years (339), and Hb was predictive of IQ at 3 and 4 years (*n*=332) (339). Another noteworthy study (*n*=3771) in the United States found that anaemia in infancy was associated with placement in a special education class, based on criteria for mild or moderate intellectual developmental disorders at age 10 (341). The study found that this likelihood of what increased by 1.28 for each decreasing unit of Hb. A limitation was that Hb was the only measure of iron status but given how common ID is in infancy it is probable that the anaemia was mainly related to iron. Another cohort where iron-deficient infants were followed up at 11–14 years (*n*=167), of the 48 participants who had been given treatment for IDA in infancy, all of them tested lower in numeracy, literacy, and motor function compared with those who had normal iron status (342). This is supported by other studies which also suggest even with supplementation there are ongoing lower scores on cognitive and motor testing through to school age and adolescence (343-345). It has been suggested that these detrimental effects may be irreversible (346), even in ID without anaemia (281, 282). Considering these outcomes in the context of NZ, where Māori and Pasifika children and adults have significantly lower literacy and numeracy rates than NZE (347-349), it would be worthwhile investigating what role iron status and early intervention might play.

## Non-Dietary Factors Influencing Infant Iron Status

Predictors of iron status in infancy are complex and far-reaching (figure 3), but five main non-dietary factors impact iron status (350, 351): perinatal iron stores (which may relate to maternal iron status), post-natal iron requirements, iron losses, inflammation, and genetics (352-359). Confounders of iron status assessment are infection and inflammation (140, 304), haemoglobinopathies (309), chronic illness, diurnal variation, sex, and ethnicity (360-362).

### Peri-natal iron stores

It has been suggested that perinatal iron status is related to maternal iron status (352, 353). The impact of maternal iron status on long-term child health has been assessed in a 2021 systematic review and meta-analysis of 44 studies, comprising of 4737 mother-child dyads (294). Maternal iron status was measured in late pregnancy using ferritin, and cut-offs for defining iron status varied across studies. Serum ferritin was used to determine ID, and maternal and infant serum ferritin cut-offs ranged from 9–16 µg/L, with one study using a cut-off of <20 µg/L and another <50 µg/L. The most common cut-off used was <12 µg/L (*n*=10). Poor maternal iron status or ID was associated with adverse outcomes in children, including infant iron status, Hb, and neurodevelopmental outcomes. Maternal ferritin was associated with infant sTfR, but a consistent association with ferritin, transferrin saturation, or Hb was not found. Maternal iron overload was associated with childhood Type 1 diabetes, and impaired growth and cognition.

Peri-natal iron stores are also affected by prematurity and birth weight. Foetal iron accumulation occurs mostly in the third trimester, with about 60% of iron accreted during this time (363). Therefore, the more premature the baby, the lower the iron stores will be. Premature infants are at much higher risk of either a low birth weight (LBW) (1500 g – 2500 g) or a very low birth weight (VLBW) (<1500 g). Term infants may also occasionally have a LBW, which can be caused by several factors, including intrauterine growth restriction, preeclampsia, maternal smoking status, maternal malnutrition, and a multiples birth (364). LBW and VLBW infants have a more rapid growth in proportion to their iron stores at birth which significantly increases their risk for ID and IDA (365). In NZ LBW and VLBW infants, and all infants born <37 weeks gestation, are supplemented with 3 mg/kg/day of iron from two weeks of age, or discharge (366). Iron supplementation has been widely studied, particularly in developing countries where ID is commonly a result of parasitic and bacterial infections (367). There is a risk of iron supplementation promoting the growth of some bacterial

infections by creating an iron-rich environment, but where appropriate treatment is available both intermittent and daily iron supplementation reduces the risk of ID and IDA (368, 369).

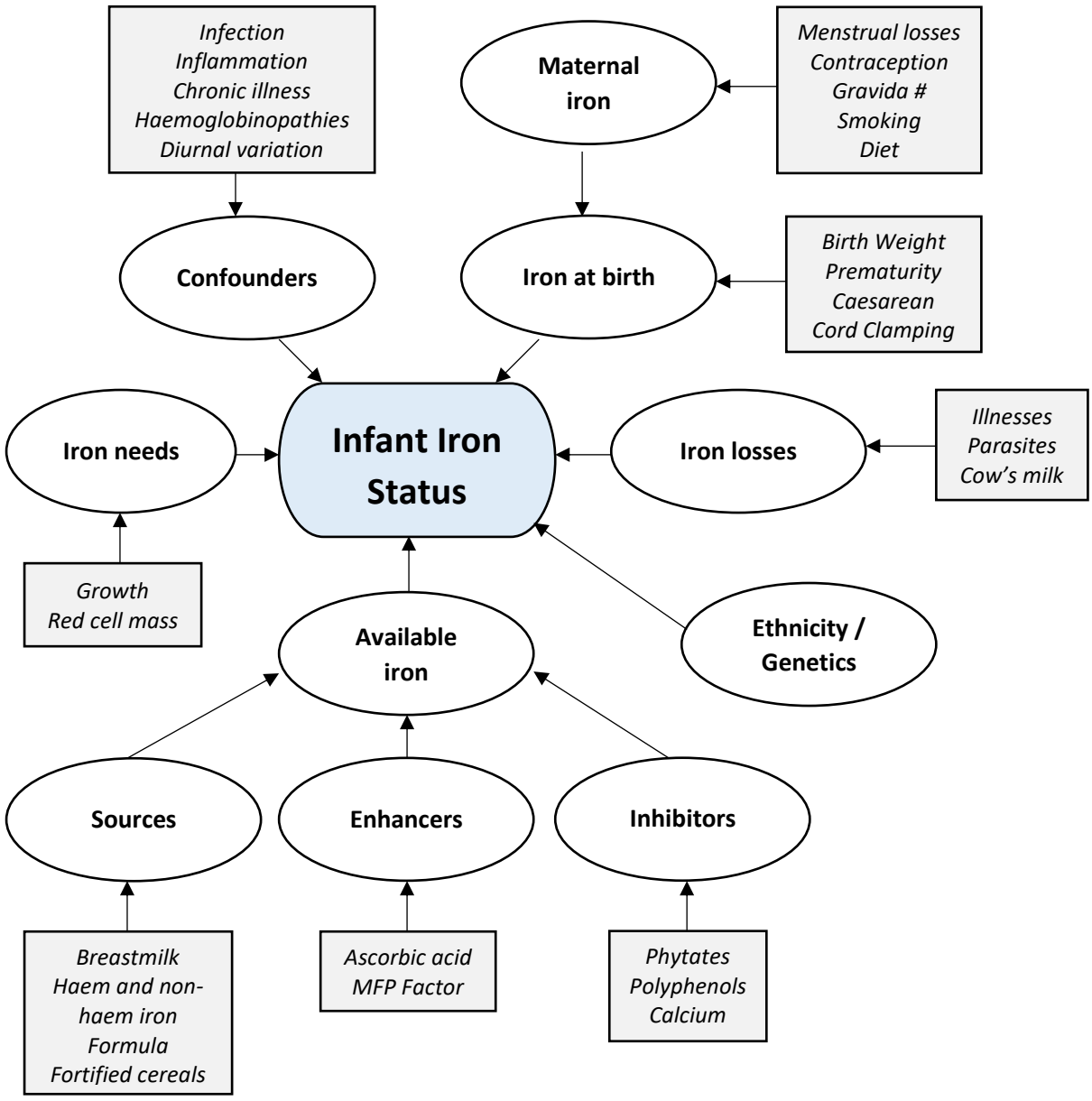



**Figure 3.** Factors impacting infant iron status
Abbreviations: MFP – Meat, Fish, Poultry

### Post-natal iron requirements

Post-natal iron requirements for infants in NZ from birth to six months is 0.2 mg/day (AI), which was calculated by multiplying the average intake of breast milk for this age group (0.78 L/day) by the iron concentration of breast milk (0.26 mg/L) (141). Although breast milk has a low concentration of iron, the bioavailability is very high due to lactoferrin (140). Additionally, there is sufficient total-body iron to meet the infant's needs for the first 4 – 6 months of age (285). The recommendation for formula-fed infants in this age group should be higher due to the decreased bioavailability of iron in infant formula, however a formal recommendation for NZ has not been established. ESPGHAN recommend that infant formula contain a minimum of 0.3 mg iron/100 kilo-calories (kcal) and a maximum of 1.3 mg/ iron100 kcal for cow's milk based formula (370). Iron requirements increase to an RDI of 11 mg/day from 7 – 12 months of age, when the infant's stores from birth are depleted (141). The rationale for this recommendation has been detailed in section two of the literature review and is based on a mixed Western diet containing animal foods.

### Iron losses

Iron losses in infants occur through minor insensible losses, and gastro-intestinal (GI) bleeding, which is multi-factorial. In developed countries parasite-mediated GI bleeding are uncommon, however, GI bleeding from cow's milk consumption before 12 months of age leading to ID is a concern. Cow's milk as a cause of GI bleeding and ID in infants was first investigated in NZ by Anyon et. al. in 1971 (371), who found that GI bleeding associated with prolonged cow's milk feeding was associated with anaemia, using a cut-off of <105 for Hb. The prevalence of cow's milk consumption in infants aged under 12 months has been quantified, with Heath et. al. (2002) finding that 69% of European infants were drinking cow's milk as a beverage before 12 months of age (91). Grant et. al. (2003) showed that infants with ID and IDA were introduced to cow's milk at a younger age than those who were iron replete (293). Another 2007 study by Grant et. al. showed that there was a higher risk of ID when consuming cow's milk prior to 12 months of age with no other non-human milk (58).

### Illness and infection

Chronic and severe acute infections profoundly impact iron metabolism, resulting in anaemia of inflammation (372, 373). This change in iron metabolism is mediated by the acute-phase response of inflammation (374, 375). Serum ferritin is an acute-phase protein and thus rises, as does erythrocyte protoporphyrin, whilst serum iron, TIBC, and serum transferrin saturation decrease (374, 376). This

makes establishing iron status challenging, but sTfR is a helpful diagnostic tool as it is not an acute-phase reactant – especially as serum ferritin can remain high for weeks after recovery (373). The pathogenesis of ID due to inflammation is mediated by hepcidin, a hormone synthesised in the hepatocytes. An increase in inflammatory cytokines stimulates increased production of hepcidin, which acts to lower iron to make it less available as a substrate for pathogens. Hepcidin lowers iron by downregulating cellular expression of iron transporters, which blocks intestinal iron absorption and results in a lower plasma iron and transferrin concentration. This decreases the iron available for erythropoiesis, and can result in anaemia (377).

Gastrointestinal blood loss secondary to *Helicobacter pylori* (*H.pylori)* infection is another cause of ID, and is prevalent in the tamariki of both Māori whānau and Pasifika community living in NZ (378). The *H.pylori* bacteria lives in the stomach and an infection causes peptic ulcers that are prone to bleeding. Infections commonly occur in childhood, with risk-factors including socio-economic status, number of people living in the household, bed sharing, and lack of hot running water.

**Genetic variation**

Hepcidin is also crucial in a non-inflammatory state. It co-ordinates contemporaneous control of iron transporters and regulatory genes to ensure careful balance between absorption of dietary iron and release of iron by macrophages to maintain iron homeostasis (379). Genetic variations in hepcidin regulation result in phenotypic idiosyncrasies. This presents as either disproportionate synthesis of hepcidin, or the resistance if iron transporters to its effects, placing the individual at higher risk for iron overload or deficiency (380). Genetic variation differs between males and females, and it has been suggested that sex hormones contribute to some of the variation in hepcidin expression (381-383). Certain ethnic groups are more likely to have genetic variations affecting hepcidin. Northern Europeans are more likely to have mutations associated with haemochromatosis, with a much higher rate amongst males than females (384, 385). A very low rate of this variation was seen amongst Pasifika adults with a prevalence of 0.01% (385). Overall, most studies investigating genetic variations in hepcidin regulation have been conducted on European cohorts, with many of the findings not reflected in studies on iron status on cohorts with similar demographic characteristics (386).

Another genetic factor impacting iron status and anaemia are the thalassaemias – inherited disorders involving damaged or missing genes for haemoglobin, affecting haemoglobin synthesis.

The synthesis of the α-globin and β-globin chains are affected, giving α- and β-thalassaemia, respectively (387). β-thalassaemia is medically more serious, but α-thalassaemia is more prevalent – with over 100 variants of α-thalassaemia with a wide range of phenotypes having been identified (388). Observational studies estimate the prevalence of a unique α-thalassaemia trait amongst Māori and Pasifika at 16.3–20% (389-392). It is necessary but challenging to distinguish between IDA and α-thalassaemia due to the microcytic hypochromic appearance of the haemoglobin, as the diagnosis will have clinical implications for the individual. An assumption of ID as the cause for anaemia if α-thalassaemia is present can result in iron overload.

## Dietary Factors Influencing Infant Iron Status

Dietary iron, with concurrent modifiers and inhibitors, is a key factor impacting iron status. Dietary iron can be obtained from a variety of animal and plant sources and comes in two forms: haem and non-haem. The absorption of iron is affected by other dietary factors. Vitamin C and the meat-poultry-fish (MFP) factor enhances the absorption of non-haem iron, while calcium, phytates, polyphenols inhibit absorption. Given that breast milk or formula are the sole sources of nutrition for the first six months of life and continue to be a significant contributor well beyond six months, the iron content and availability is important. Fortified commercial infant food is another common source of iron during complementary feeding but can vary depending on the type of food and the packaging, with infant food packaged in pouches on average much lower in iron than that in tins, jars, or boxes (112).

### Haem and non-haem iron

Iron exists in its ferric (Fe3+) state at a physiological pH and is referred to as non-haem iron. To be absorbed, iron needs to be in its ferrous state (Fe2+) or bound to a haem protein, which gives haem iron. Haem iron is found in meat, poultry, seafood, and eggs, and is most abundant in red meat, liver, oysters, and mussels. The haem iron is proteolysed in the stomach to release the iron from the haem molecule, and the iron is then easily absorbed by the enterocytes in the duodenum and proximal jejunum (393). Approximately 25–30% of haem iron absorbed, and just 5–12% of non-haem iron absorbed (394), however non-haem iron accounts for about two thirds of dietary iron intake (395, 396). Non-haem iron is found in both plant foods and animal-based foods and must be reduced into its ferrous form to be absorbed by the enterocytes. The non-haem iron is proteolysed by gastric juices in the stomach, then is reduced to the absorbable Fe2+ by a ferric reductase enzyme or ascorbic acid in the brush border of the enterocytes (393, 394). This pH-dependent mechanism

can be enhanced or inhibited by several dietary factors. Enhancers are vitamin C, MFP factor, and inhibitors are phytates, polyphenols, and calcium.

**Absorption modifiers of iron**

Vitamin C is a well-established enhancer of non-haem iron absorption with two mechanisms of action. Firstly, it acts to form a soluble chelate with non-haem iron that remains soluble in the alkaline conditions of the duodenum and proximal jejunum, thus allowing ready absorption by the enterocytes. This affect is dose-dependent, which was demonstrated by Cook et. al. (1977). Increasing doses of ascorbic acid, starting at 25 mg and increasing to 1000 mg, were added to a liquid meal with 4.1 mg of non-haem iron, resulting in an parallel increase in iron absorption from 0.8% to 7.1% (397). Vitamin C also acts to reduce the inhibitory impact of calcium, polyphenols, and phytate by solubilising the iron which prevents it from binding to these inhibitors and forming an insoluble chelate (398, 399). Despite the demonstratably profound effect of vitamin C on iron absorption, iron status is minimally affected (400-402), with gastrointestinal adaption to vitamin C supplementation has been ruled out as a possible explanation (403).

Another enhancer is MFP factor, a peptide found in meat, fish, and poultry that enhances the absorption of non-haem iron when eaten in the same meal. The mechanism is not fully understood, but Taylor et. al. (1986) suggest that the effect is likely due to cysteine-containing peptides acting to block inhibitors in the lumen of the small intestine, and luminal transportation (404). Absorption of non-haem iron with the addition of beef, chicken, or fish was compared to the addition of egg, with a 2–3 fold increase in absorption with the beef, chicken and fish as compared to the egg (405). These findings are supported by other studies that show a consistently improved absorption of non-haem iron when animal protein is present in the same meal (189, 406). However, other studies claim that the improvement in iron absorption conferred by MFP factor is less important than the contribution of haem iron from those foods (407, 408).

There are several dietary inhibitors of non-haem iron absorption, which are phytates, polyphenols, and calcium. Phytates are found in soybeans, black beans, split beans, lentils, mung beans, nuts, and un-refined grains (e.g., cereals, bran) and rice. These have a dose-dependent effect on non-haem iron absorption (409) and inhibit absorption by binding to the iron and forming insoluble complexes (410). Polyphenolics act upon iron in a similar way: for example, the tannins in tea and coffee form iron-tannate complexes with iron that significantly decrease non-haem iron absorption (411-413). As

well as tea, polyphenols are also found in oregano, red wine, and many fruits (414). Unlike phytates and polyphenols, calcium inhibits both haem and non-haem iron through divalent metal transporter 1 at the enterocyte (398). It has also been suggested that inhibition might occur when iron is moved into circulation via ferroportin and hephaestin peptides (415).

**Iron content of breast milk and infant formula**

The average iron concentration of breast milk is 0.26 mg/L when the infant aged 0–6 months, with levels decreasing as lactation progresses. Ejezie et. al. (2011) measured the iron concentration in the breast milk of new mothers on five occasions, 3–15 days after delivery, and found an average concentration of 32.79 mg/L. The first measure on day 0–3 after delivery found an iron concentration of 43.92 mg/L, while on day 13–15 the iron concentration had dropped to 27.63 mg/L (416). The Nutrient Reference Values for Australia and New Zealand (NRVs) cite an average breastmilk intake of 0.78 L per day for 0–6-month-old infants, which would give an average of 0.2 mg of iron per day. This iron requirement is relatively low from 0–6 months compared to 7–12 months of age as the infant is relatively self-sufficient regarding iron at this time. This is because a healthy term infant is born with an average Hb of 170 g/L, which drops during the first six weeks to 120 g/L (417). Iron is re-distributed from senescent erythrocytes and transferred from Hb to iron stores, which then increase in size. As the infant's blood volume grows with age, iron is moved from stores back into the blood pool, until such time as the infant has approximately doubled in weight or is about 4–6 months of age (285, 417).

The iron in breast milk is easily absorbed due to the presence of lactoferrin. Lactoferrin has several functions in the human body – modulating immune function through binding to microbes, host cells, and other components of the immune system, as well as regulation of the maturation, migration, and immune cell function (418). In infants, lactoferrin in breast milk binds iron, rendering it inaccessible to pathogens that rely on iron to grow. It also has antiviral activity, killing *Escherichia coli*, *Streptococcus mutans*, *Streptococcus peneumoniae*, *Vibrio cholerae*, *Candida albicans*, and *Pseudomonas aeruginosa* (419). The ability of lactoferrin in breast milk to sequester iron is well known. Lactoferrin acts to reversibly bind non-haem (ferric) iron, with the resulting complex stable at a low pH of <3 (420). This enables the complex to traverse the GI tract to the site of absorption intact, where it binds with its cell membrane receptor and facilitates the absorption of iron (421, 422). Hernell and Lönnerdal (2002) fortified infant formula with bovine lactoferrin and found no impact on iron status (423). Much of the research examining bovine lactoferrin fortification in infant

formula to allow a lower concentration of iron, which in turn is hypothesised to reduce the prevalence of illnesses in infancy. James et. al. (2007) supplemented healthy term infants' formula with bovine lactoferrin from 4 weeks to 12 months of age in a double-blind RCT, and found significantly fewer respiratory tract illnesses in the lactoferrin-fed infants (*n*=26) as compared to a control group (*n*=26) on standard infant formula (424). Impact on iron status was not assessed this study. In contrast, Björmsjö et. al. (2022) found no difference between infants provided with bovine lactoferrin fortified formula and those on standard infant formula in a much larger Swedish RCT (*n*=181).

Due to the much lower bioavailability of the iron in infant formula as compared to that in breast milk, infant formula is fortified to a much higher level. American guidelines currently recommend 10–12 mg/L, which would correspond to 7.8–9.36 mg/day. The ESPGHAN recommend fortification of 2–8.5 mg/L, which equates to 1.56–6.63 mg/day. The lower concentration of 2 mg/L, based on the concentration in breast milk and an assumed difference in bioavailability, has not shown to be sufficient in RCTs, while the higher value of 12 mg/L is based on an average iron requirement from 0–12 months of age, not taking into account the much lower iron requirements from 0–6 months (425).

**Iron content of commercial infant foods**

The landscape of commercial baby food has changed drastically over the past few years with the advent of baby food pouches and an increasing variety of foods, combinations, and textures. The commercial infant food market in NZ has grown by 27.2% between 2010 and 2016, with a retail value of NZD$42.6 million in 2019 (119). Commercial infant foods are varied, from iron-fortified powdered rice through to various fruit, vegetable, meat, cereals, and snacks, but the iron content varies. Rice and cereal-based foods tend to be good vehicles for iron fortification. In NZ cereal-based foods that are marketed for infants, and contain ≥70% cereal, are legally required to be iron fortified with at least 20 mg/100g of dry weight under the Australia New Zealand Food Standards Code (426). Katiforis et. al. (2021) investigated the iron content of commercial infant foods in NZ and found that pouches contained just 0.3 mg/100g of iron, compared to up to 10 mg/100g in dry cereals (112). It is important to note that cereal in pouches is pre-prepared, thus it would be diluted, so therefore it would be well under the 70% threshold. This means they not required to meet the Food Standards Code for iron fortification for cereals, but it is feasible that parents and caregivers may assume these products are similarly fortified as dry cereal products. Furthermore, the Food Standards Code of 20

mg/100g iron applies only to cereal products marketed for infants aged 6+ months. This is problematic because the guidelines state that solids should be introduced at about 6 months of age, and baby rice is a common first food – so it is possible parents and caregivers of 6–month-old infants would choose an iron fortified infant rice labelled as "4+ months", not realising it may not be adequately fortified with iron. Additionally, given the over-representation of Māori and Pasifika infants in ID and IDA, it is important that commercial infant food is iron fortified to the required level.

Iron concentration in meat, fish, and poultry infant food is also low, with the proportion of meat or fish 5–10% in pouches, and 5–12% in non-pouches (112). It is likely that a home-made variant would contain a higher proportion of meat or fish, and that pouches that are meat based may well be assumed to be a good source of iron by parents and caregivers.

Another relatively new type of commercial infant food in the New Zealand market are snack foods, such as corn or rice puffs in various shapes and flavours. There are both sweet and savoury varieties of these snack foods, and 27.6% (*n*=16) are iron fortified with up to 35 mg/100g (112). The median serving size is 9g, so those with the upper level of 35 mg/100g of fortification would provide about 3 mg of iron. However, relying upon snack foods as a source of iron would not be recommended due to the sugar content in the sweet variety, and the recommendation not to include commercial snack foods as a regular part of the diet. There may be circumstances with picky eaters where this may be the only source of iron that is accepted though.

**Impact of infant iron intake on iron status**

From approximately six months of age the breastfed infant must obtain 90% of their iron requirements from complementary food to prevent ID (417, 427), and it has been suggested that dietary interventions play a key role in preventing ID (58). Four studies that investigated infant iron intake found ranges from 1.5–7 mg/day (64, 91, 260, 277), suggesting a consistently inadequate intake. None of these studies were stratified by ethnicity so it is not possible to elucidate the iron intake for Māori and Pasifika infants from these. Wall et. al. (2008) looked at iron intake by ethnicity and found the median iron intake for Pasifika infants in NZ aged 6–11 months was significantly higher than that of NZE and Māori. Pasifika infants (*n*=45) had a median intake of 7.7 mg as compared to 6.3 mg of both Māori (*n*=42) and NZE (*n*=28) (*p*=0.04) (428). This is a small sample size, however.

**Iron status of infants in Aotearoa New Zealand**

There have been 22 studies investigating the iron status of infants in NZ since 1963 (58, 87, 91-93, 293, 295, 371, 428-441). These studies are summarised in table 12.

**Table 12.** Summary of studies investigating iron status of infants in NZ

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| Akel et al. (1963) (429) | *n*=344<br>Māori (*n*=174) and NZE (*n*=170 hospitalised infants aged from birth to 12 months in Wellington, Hutt Valley, Whanganui, Gisborne, Rotorua, and Whakatane. | Observational cross-sectional. | <u>Indices</u><br>Hb and blood film.<br><br><u>Cut-offs</u><br>Hb: <105 for anaemia | <u>Iron status</u><br>IDA prevalence at 0–6 months:<br>Māori: 22 (21.6%)<br>NZE: 13 (11.6%)<br><br>IDA prevalence at 6–12 months:<br>Māori: 47 (65.3%)<br>NZE: 23 (39.7%)<br><br>Iron status differences between Māori and NZE in both age groups are statistically significant.<br><br>The variation in anaemia between geographical areas was considerable, but statistical significance was not reported. | • Hb the only status index reported so the link of anaemia to ID uncertain<br>• Infection and inflammation were not measured or controlled for<br>• Hospitalised population means extrapolation to the general population at this time is not reliable<br>• Dietary data not collected |
| Anyon et al. (1974) (431) | *n*=404<br>Full-term NZE infants born in Wellington and Hutt Valley Hospitals | Longitudinal.<br><br>Blood sample taken within four days of birth, 4 months, 9 months, 14 months, and 24 months. | <u>Indices</u><br>Hb, packed cell volume, SI, ST, blood film.<br><br><u>Cut-offs</u><br>Hb <105 for anaemia<br>Other cut-offs not reported.<br><br><u>Dietary data</u><br>A survey on breast-feeding duration, iron supplements, and illness were administered at each visit. | <u>Iron status by age</u><br>1.2% (*n*=3) infants were anaemic at 4 months.<br><br>4% (*n*=13) were anaemic at 9 months.<br><br>6% (*n*=19) were anaemic at 14 months.<br><br>2.8% (*n*=8) were anaemic at 18 months. | • Non-random sample<br>• ID not defined, and prevalence not reported<br>• Infection and inflammation were not measured or controlled for<br>• Dietary data not collected |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | | | 2.5% (*n*=7) were anaemic at 24 months. | |
| Anyon et al. (1976) (430) | *n*=130<br>Full-term self-identified Polynesian infants born in Lower Hutt. | Longitudinal.<br><br>Capillary sample taken by heel prick at 3 days of age, 4 months, 8 months, and 1 year.<br><br>Faecal analysis for occult blood was taken at the same time as the blood sample. | <u>Indices</u><br>Hb only.<br><br><u>Cut-offs</u><br>Hb <105<br><br><u>Dietary data</u><br>A questionnaire around milk feeding was administered at the same time as each blood sample was taken. | <u>Iron status</u><br>Anaemia prevalence: n (%)<br>4 months: 12 (9.8)<br>8 months: 31 (26)<br>12 months: 37 (32)<br><br>Gastrointestinal bleeding associated with prolonged cow's milk feeding was associated with anaemia.<br><br>When compared to an earlier study of NZE infants, there was a statistically significantly higher rate of anaemia amongst Polynesian infants. | • Hb was the only index measured and was inferred to be IDA.<br>• Infection and inflammation were not measured or controlled for.<br>• Dietary data limited to milk feeds |
| Moyes et al. (1990) (436) | *n*=421<br>Very young Māori and NZE children aged 6–24 months in the Bay of Plenty. | Observational cross-sectional.<br>Data obtained from two groups at two different time points:<br>Community: 1981 (*n*=143)<br>Hospitalised: 1979–80 (*n*=110, and 1988 (*n*=168). | <u>Indices</u><br>Hb, SF<br><br><u>Cut-offs</u><br>Hb: <110<br>SF: <10<br><br><u>Dietary data</u><br>Feeding history around formula and breastmilk was obtained but describe by the authors as not successful. | <u>Iron status</u><br>1981 community infants<br>ID prevalence<br>Māori (*n*=80): 51%<br>NZE (*n*=63): 25%<br><br>IDA prevalence<br>Māori (*n*=80): 34%<br>NZE (*n*=63): 6%<br><br>1979–80 hospitalised infants<br>IDA prevalence<br>Māori (*n*=110): 50%<br><br>1988 hospitalised infants<br>IDA prevalence | • ID only reported for the 1981 group, as other two groups hospitalised so SF is a confounder<br>• No NZE infants examined in the 1979–1980 group<br>• Inflammation and infection were not accounted for other than excluding SF as a measure in the two hospitalised groups<br>• Hospitalised groups more likely to be anaemic because of illness<br>• Only two indices for iron status used |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | | | Māori (*n*=123): 40%<br>NZE (*n*=45): 22% | |
| Dickson et al. (1992) (433) | *n*=18<br>Very young children aged 3–19 months of Cambodian refugees in Dunedin. | Observational cross-sectional.<br><br>Status analysed from a venous blood sample. | <u>Indices</u><br>Hb, SF.<br><br><u>Cut-offs</u><br>Hb: <110<br>SF: <10<br><br><u>Definitions</u><br>IDA – low Hb and SF<br>ID – low SF | <u>Iron status</u><br>Mean (range)<br>Hb: 108.1 (85.8 – 125.4)<br>SF not reported.<br><br>ID: *n*=11 (65%)<br>IDA: *n*=6 (37%)<br><br>Infants over the age of 10 months had lower SF. | • Small sample size<br>• Infection and inflammation were not controlled for<br>• Limited status indices were used<br>• Findings not applicable to the general NZ population |
| Poppe (1993) (437) | *n*=82<br>Very young, hospitalised children aged 6–24 months in South Auckland. | Observational cross-sectional. | <u>Indices</u><br>Hb, MCV<br><br><u>Cut-offs</u><br>Hb: <100 at 6–12 months, <110 at 13–24 months.<br>MCV: <70<br><br><u>Definitions</u><br>IDA – low Hb and MCV<br>ID – low MCV | <u>Iron status</u><br>ID prevalence: *n*=27 (32.9%)<br><br>IDA prevalence: *n*=15 (18.3%)<br><br>A further 10 (30%) had an MCV of 70–72, with microcytic hypochromic blood film appearance, indicating probable ID. | • Small sample size<br>• Hospitalised population<br>• Infection and inflammation were not measured or controlled for<br>• Only two indices were used for iron status |
| Crampton et al. (1994) (432) | *n*=43<br>Very young children aged 6–36 months in Porirua, New Zealand.<br><br>Pasifika: *n*=18 (42%)<br>Māori: *n*=15 (35%)<br>NZE: *n*=7 (16%)<br>Other: *n*=3 (7%) | Observational cross-sectional.<br><br>Screening for IDA was offered in the context of a routine consultation, excluding infants with anything other than a trivial illness. | <u>Indices</u><br>Hb, MCV, SF<br><br><u>Cut-offs</u><br>Hb: <110<br>MCV: <70<br>SF: <10<br><br><u>IDA definition</u><br>Low Hb and MCV. | <u>Iron status</u><br>Mean (SD)<br>Hb: 108.8 (11.2)<br>MCV: 70.7 (7.4)<br>SF: 24.4 (28.0)<br><br>IDA: *n*=10 (23.8%)<br>Low SF only: *n*=11 30.6%)<br>Low Hb only: *n=7* (16.6%) | • Small cohort<br>• Results not reported by ethnicity<br>• Opportunistic sampling could give selection bias<br>• Inflammation and infection were not measured in blood sample or controlled for<br>• Ferritin excluded in case of a child having a minor illness |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | | | Iron therapy was offered to parents of IDA infants, and a follow-up study found improved haematological indices. | that was not identified, thus confounding the findings. |
| Rive et al. (1996) (438) | *n*=67<br>Very young children aged 6–23 months attending the paediatric acute assessment unit in South Auckland and required a blood test.<br><br>Māori or Pasifika: *n*=55 | Observational cross-sectional. | <u>Indices</u><br>Hb, MCV, SF, CRP<br><br><u>Cut-offs</u><br>Hb: <110<br>MCV: <70<br>Ferritin: <25<br><br><u>ID definition</u><br>Normal Hb with ferritin <25. Children with ferritin from 25–40 in the presence of high CRP were assumed to have latent ID.<br><br><u>IDA definition</u><br>Hb <110, MCV <70, and ferritin <25. | <u>Iron status</u><br>n (%)<br>ID: 2 (3%)<br>Latent ID: 4 (6%)<br>IDA: 14 (21%) | • Cohort were unwell hospitalised children<br>• Small sample size<br>• Ferritin cut-off high, but may be appropriate in a hospitalised group<br>• Sampling method could give selection bias<br>• Results not stratified by ethnicity |
| Wham (1996) (295) | *n*=53.<br>Very young healthy urban children aged 9–24 months in Auckland.<br><br>Children with intercurrent infections excluded. | Observational cross-sectional. | <u>Indices</u><br>SF, SI, TIBC, TS, Hb, MCV, MCH, white blood count, neutrophils, and lymphocytes.<br><br><u>ID definition</u><br>ferritin <10<br><br><u>IDA definition</u><br>Hb <110<br><br><u>Dietary data</u> | <u>Iron intake</u><br>Average iron intake was 5.1 ± 3.1 mg.<br><br><u>Iron status</u><br>ID: 13.5% (*n*=7)<br>IDA: 20% (*n*=10)<br>Median SF: 23 µg/L<br>Median Hb: 119.5 | • Small sample size<br>• Opportunistic sampling method, so selection bias may exist<br>• Ethnically non-representative<br>• Not stated if infection or inflammation was present |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | | one 24hr recall and a diet history questionnaire. | No statistically significant relationship between iron intake and iron status. | |
| Wilson et al. (1999) (441) | *n*=206<br>Very young, hospitalised children aged 9–23 months, living in Auckland. | Prospective. | <u>Indices</u><br>Hb, blood film, SF, TS<br><br><u>ID definition</u><br>Not defined in this study.<br><br><u>IDA definition</u><br>Hb <110, red cell distribution width >14.5%, and either SF <10 or TS <10%.<br><br><u>Dietary data</u><br>Obtained from subjects with IDA via an interview one week post discharge using closed questions. | <u>Dietary data</u><br>42% of children with IDA were drinking cow's milk regularly before the age of 9 months.<br><br>33% had not yet started red meat by 9 months of age.<br><br>More Māori and Pasifika children drank cow's milk regularly before 8 months of age than NZE children.<br><br><u>Iron status</u><br>IDA prevalence – *n* (%)<br>NZE: 8 (14)<br>Māori: 8 (21)<br>Pacific: 43 (43.4) | • Hospitalised unwell cohort<br>• Infection and inflammation not controlled for<br>• Dietary data could be subject to recall bias |
| Soh et al. (2002) (93) | *n*=226<br>Very young non-breastfeeding children aged 6–24 months in urban centres in the South Island. | Cross-sectional survey.<br><br>Usual intake was determined for the dietary data. | <u>Indices</u><br>SF and CRP<br><br><u>ID definition</u><br>SF ≤20<br>Inflammation: CRP ≥10<br><br><u>Dietary data</u><br>3-day non-consecutive weighed food record. | <u>Iron intake</u><br><12-months infants: 15% had iron intake below the EAR, with the majority (60%) of iron intake obtained from iron-fortified infant formula.<br><br>≥12-month children: 66% had iron intake below the EAR with | • Exclusion of breastfed children<br>• Hb was not reported so prevalence of IDA not known<br>• Cut-off for ID higher than WHO and NHANES recommendation<br>• Non-representative sample for ethnicity and mother's education<br>• Low response rate |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | | | the majority (31%) obtained from cereals.<br><br><u>Iron status</u><br>ID: 15% (*n*=37),<br>6–11.9 months: 8%<br>12–24 months: 20% | • No upper cut-off for ferritin used to indicate inflammation or infection |
| Heath et al. (2002) (91) | *n*=74<br>Very young children aged 9–24 months.<br><br>Exclusion criteria: maternal diabetes and chronic health conditions, prematurity, low birth weight. | Longitudinal.<br><br>Dietary data reported at 9, 12, 18 and 24 months of age.<br><br>Blood sample obtained from capillary heel prick. | <u>Indices</u><br>Hb, MCV, ZPP.<br><br><u>Cut-offs</u><br>Hb: <110<br>MCV: <77<br>ZPP: >80<br><br><u>Definitions</u><br>ID was defined as a low MCV and elevated ZPP.<br><br>IDA was defined as low Hb and low MCV.<br><br><u>Dietary data</u><br>Estimated 24-hr diet record once a month for the first 12 months.<br>Estimated 3-day diet record collected at 12 months, 18 months, and 24 months. | <u>Iron intake</u><br>Median ($25^{th}$, $75^{th}$ percentiles), mg/day<br>9 months: 7.0 (3.7, 10.7)<br>12 months: 4.3 (3.2, 7.1)<br>18 months: 4.9 (3.7, 6.3)<br>24 months: 4.6 (3.9, 5.8)<br><br><u>Iron status</u><br>*n* (%)<br>ID – 9 months: 6 (19), 12 months: 7 (22), 18 months: no data, 24 months: 4 (13).<br>IDA - 9 months: 4 (7), 12 months: 4 (7), 18 months: 4 (7), 24 months: 0 (0). | • No ethnic diversity – only NZE participants studied<br>• High SES participants only<br>• Small number of participants<br>• Inflammation and infection were not measured or controlled for<br>• SF was not used as a measure of iron stores<br>• Breastmilk intake was estimated rather than calculated |
| Grant et al. (2003) (293) | *n*=391.<br>Very young, hospitalised children | Prospective.<br><br>Blood sample obtained during hospitalisation. | <u>Indices</u><br>SF, SI, red cell distribution width, Hb. | <u>Iron status</u><br>ID: 73 (18.7%)<br>IDA: 220 (56.2%)<br>Iron replete: 98 (25.1%) | • Hospitalised and unwell cohort, thus not representative of the general population |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | aged 8–23 months in Auckland. | Questionnaire on previous and current feeding patterns conducted post discharge. | <u>ID definition</u><br>Abnormal values of two out of three of:<br>SF <10<br>SI <10%<br>Red cell distribution width >14.5%.<br><br><u>IDA definition</u><br>Hb <110 in addition to ID. | <u>Ethnic spread - ID</u><br>NZE: 24 (33%)<br>Māori: 19 (26%)<br>Pasifika: 26 (36%)<br>Other: 4 (5%)<br><br><u>Ethnic spread - IDA</u><br>NZE: 49 (22%)<br>Māori: 44 (20%)<br>Pasifika: 115 (52%)<br>Other: 12 (6%)<br><br>CRP measured in 313 (80%) of the sample, and after stratification by CRP ID and IDA prevalence remained.<br><br>ID and IDA higher in Pasifika, pneumonia diagnosis, low maternal meat intake during pregnancy, tea drinkers, more than three children in the house, and breastfed. | • CRP not able to be measured in all participants<br>• Not stratified further by infants aged <12 months and those over 12 months |
| Thom et al. (2003) (440) | *n*=81<br>Low birthweight infants (<2500g) aged 8–13 months.<br><br>Mean birthweight: 1800g<br>Mean gestational age: 31.6 weeks | Observational cross-sectional.<br><br>Infants classified into "high iron intake" group if iron supplemented, with the remainder in the "low iron intake" group. | <u>Indices</u><br>Hb, SF, SI, TIBC, TS, sTfR, ZPP, MCV, haematocrit.<br><br><u>Cut-offs</u><br>Hb: <110<br>SF: <10<br>SI: not stated<br>TIBC: not stated<br>TS: <10% | <u>Iron status</u><br>Total cohort *n* (%)<br>Latent ID: 27 (33)<br>IDA: 15 (19)<br><br>ID prevalence in low iron intake group was 21 (66%), and the high iron intake group was 6 (12%). | • Iron intake classified, not quantified, so true effect of iron intake may be underestimated.<br>• Inflammation and infection were not measured or controlled for<br>• Findings not applicable to the general population as study |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | Blood sample obtained via venepuncture. | sTfR: not stated<br>ZPP: not stated<br>MCV: <70 fL<br>Haematocrit: <32%<br><br><u>Definitions</u><br>Latent ID: Hb ≥110, and SF <10 or TS <10%<br><br>IDA: Hb <110 and at least one other abnormal index out of haematocrit, MCV, SF, or TS. | Infants in the low iron intake group were 13-fold more likely to develop ID | was specific for low birthweight infants. |
| Emery & Barry (2004) (434) | *n*=100<br>Māori and non-Māori neonate and mother dyads.<br><br>Premature infants, complicated pregnancies and mothers with medical conditions were excluded. | Observational cross-sectional.<br><br>Maternal venous blood samples taken the day prior to delivery.<br><br>Cord blood sample taken at the time of caesarean section. | <u>Indices</u><br>Cord Hb, cord iron, cord ferritin measured at birth.<br><br>Maternal SF, Hb and SI measured one day prior to delivery. | <u>Infant iron status</u><br>Mean cord Hb (Māori): 143.6 (*n*=20)<br>Mean cord Hb (NZE): 144.1 (*n*=73)<br>Difference: *p*=0.9<br><br>Mean cord iron (Māori): 26.7 (*n*=23)<br>Mean cord iron (NZE): 26.4 (*n*=77)<br>Difference: *p*=0.8<br><br>Mean cord SF (Māori): 26.7 (*n*=23)<br>Mean cord SF (NZE): 26.4 (*n*=76)<br>Difference: *p*=0.01 (significant)<br><br><u>Maternal iron status:</u> | • Cut-off parameters to define ID and IDA were not stated.<br>• Results for total study group were not reported<br>• No follow-up to assess changes at six months or beyond<br><br><u>Considerations:</u><br>Only mothers undergoing elective caesarean sections were included in the study. |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | | | Hb: Māori significantly lower than non-Māori ($p$=0.002).<br>SF and SI differences non-significant. | |
| Soh et al. (2004) (92) | $n$=226<br>6–24-month-old non-breastfeeding urban children. Same sample as Soh et al. (2001) | Cross-sectional observational.<br><br>Venous blood samples collected for iron status.<br><br>3-day weighed food record collected for dietary assessment. | <u>Indices</u><br>Hb, MCV, ZPP, SF, CRP.<br><br><u>Cut-offs</u><br>Hb: <110<br>MCV: ≤73 fL<br>SF: ≤10 and ≤12<br>ZPP: ≥70<br>CRP <10<br><br><u>Definitions</u><br>ID present if two or more of SF, ZPP and MCV are present.<br><br>IDA is present if in addition to the above, Hb is also low. | <u>Infant iron status</u><br>Depleted iron stores:<br>6–11.9 months: 4.2%<br>12–24 months: 16.4%<br><br>Functional ID:<br>6–11.9 months: 8.3%<br>12–24 months: 7.5%<br><br>ID:<br>6–11.9 months: 2.8%<br>12–24 months: 5.0%<br><br>IDA:<br>6–11.9 months: 5.6%<br>12–24 months: 2.5%<br>Mean values (6–11.9 months)<br>Hb: 111 ± 9<br>MCV: 77.4 ± 3.3<br>SF: 22.9 ± 2.0<br>ZPP: 50.0 ±16.6<br><br>Mean values (12–24 months)<br>Hb: 116 ± 10<br>MCV: 77.8 ± 3.8<br>SF: 16.0 ± 1.8<br>ZPP: 47.0 ± 15.7 | • CRP value of <10 is higher than the cut of used in other studies<br>• Not stated if high ferritin was used as a possible inflammation marker<br>• Study was not appropriately powered for ethnic stratification, but ethnicity is reported as a positive association with SF. |
| Grant et al. (2007) (58) | $n$=324 | Observational cross-sectional. | <u>Indices</u><br>Hb, SF, MCV, TS. | <u>Iron status</u><br>Median ($5^{th}$, $95^{th}$ percentile) | • Insufficiently powered for full ethnic examination |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | Very young children aged 6–23 months living in Auckland.<br><br>Māori: 18%<br>Pasifika: 24%<br>NZE & other: 58% | Random sampling method used.<br><br>Venous blood sample obtained for iron status. | <u>Cut-offs</u><br>Hb: <110<br>SF: <10<br>MCV: <73 fL<br>TS: <10%<br>CRP: >4<br><br><u>Definitions</u><br>ID present if two or more of SF, MCV and TS are present.<br><br>IDA is present if in addition to the above, Hb is also low. | Hb: 119 (100, 134)<br>SF: 20 (4, 57)<br>TS (%): 16.2 (5.4, 31.6)<br>MCV: 77 (67, 83)<br><br>ID: 14%<br>IDA: 6%<br><br><u>ID by ethnicity</u><br>Māori: 20%<br>Pasifika: 17%<br>NZE: 7%<br>Other: 27%<br><br>Multivariate analysis showed BMI and no milk formula were risk factors for ID.<br><br>Ethnicity was associated with ID. | • Prevalence estimates overlapped between Māori, Pasifika, and "other" ethnicities<br>• "Other" ethnicity included diverse nationalities |
| Wall et al. (2008) (428) | *n*=247<br>Very young children aged 6–23 months living in Auckland.<br><br>Same sample as Grant et al. (2007) | Observational cross-sectional, stratified by ethnicity.<br><br>Random sampling method used.<br><br>Venous blood sample obtained for iron status. | <u>Indices</u><br>Hb, SF, MCV, TS.<br><br><u>Cut-offs</u><br>Hb: <110<br>SF: <10<br>MCV: <73<br>TS: <10%<br><br><u>Definitions</u><br>ID present if two or more of SF, MCV and TS are present.<br><br>IDA present if in addition to the above Hb is low. | <u>Iron intake: 6–11 months</u><br>Median energy (kJ/kg): 325 (242, 389)<br>Median iron (mg/day): 8.3 (4.7, 10.8)<br>Median vitamin C (mg/day): 90 (42, 125)<br><br><u>Iron intake: 12–23 months</u><br>Median energy (kJ/kg): 337 (284, 394)<br>Median iron (mg/day): 6.3 (4.3, 8.8)<br>Median vitamin C (mg/day): 67 (41, 119) | • Iron status measures not reported in this paper<br>• Confounding factors with ethnicity not controlled for due to the use of a univariate analysis<br>• No association found between ethnicity and ID in the same sample in a previous paper using a multivariate analysis. |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | | <u>Dietary data</u><br>Two day weighed diet record | <u>Iron status</u><br>• Risk for ID was associated with iron intake.<br>• Pasifika children had more iron per day but an increased prevalence of ID.<br>• Fewer associations between dietary factors and iron status in Māori and Pasifika children compared to NZE | |
| Szymlek-Gay et al. (2009) (439) | *n*=225<br>Very young urban children aged 12–20 months living in the South Island.<br><br>Exclusion criteria:<br>Hb <105, or<br>HB <110 and SF ≤12 | Randomised controlled trial.<br><br>20-week trial. Children randomised into one of three groups:<br>1. Red meat (*n*=90)<br>2. Fortified milk (*n*=45)<br>3. Placebo control (*n*=90)<br><br>Blood samples collected at baseline and 20 weeks for Hb, SF, sFtR, and CRP.<br><br>Three-day weighed diet record taken at baseline, four weeks, and 18 weeks. | <u>Indices</u><br>Hb, MCV, ZPP, SF, CRP.<br>sTfR was used to calculate body iron.<br><br><u>Cut-offs</u><br>Hb: ≤110<br>MCV: ≤73<br>ZPP: ≥70<br>SF: ≤12<br>CRP: ≥10<br><br><u>Definitions</u><br>Depleted iron stores were defined by SF ≤12.<br><br>Iron-deficient erythropoiesis was defined by ≥2 abnormal values for SF, MCV, and ZPP, with normal Hb. | <u>Iron intake</u><br>Geometric mean (95% CI) at baseline in mg/day:<br>Meat group: 4.4 (4.1, 4.9)<br>Fortified milk group: 4.2 (3.7, 4.7)<br>Control group: 4.8 (4.3, 5.3)<br><br><u>Iron status</u><br>Geometric mean (95% CI)<br>Baseline:<br>Hb - meat group: 117.3 (114.7, 119.9), fortified milk group: 119.3 (116.1, 122.4), control group: 117.9 (115.3, 120.5).<br><br>SF - across all groups was 21.7 (19.7, 23.9).<br><br>sTfR - meat group: 7.0 (6.6, 7.4), fortified milk group: 6.6 (6.1, 7.1), control group: 6.9 (6.5, 7.3). | • Inadequate number of participants in fortified milk group<br>• Breast-fed infants excluded from analysis as breastmilk consumption not quantified<br>• Proportion of infants with ID and IDA only shown with a figure – numbers not given.<br>• Infants with ID and IDA excluded so not able to see any possible changes with this.<br>• Lack of ethnic diversity in sample |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | | IDA is present if in addition to iron-deficient erythropoiesis, Hb is also low. | Body iron (mg/kg) - meat group: 3.7 (2.7, 4.6), fortified milk group: 3.9 (2.8, 5.0), control group: 4.3 (3.4, 5.2).<br><br>Post intervention:<br>SF increased significantly in the fortified milk group ($p$<0.001), did not change in the meat group, and decreased non-significantly in the control group ($p$=0.063). | |
| Morton et al. (2014) (435) | $n$=113<br>Neonate and mother dyads enrolled in the GUiNZ study living in South Auckland. | Observational cross-sectional.<br><br>Cord blood sample collected at the time of birth. | <u>Indices</u><br>Hb, SF, MCV, SI, TIBC, CRP<br><br><u>Cut-offs</u><br>ID: Cord SF <75<br>IDA: Hb <130<br><br><u>Exclusion</u><br>CRP: >5<br>SF: >370 | <u>Iron status</u><br>Mean cord Hb: 160±17 ($n$=103)<br>Mean cord MCV: 109±5 ($n$=103)<br>Mean SI: 25.6±9 ($n$=113)<br>Mean TIBC: 40±8 ($n$=103)<br>Mean TS (%): 63±3<br>Median SF: 135 (88–180)<br><br><u>ID prevalence</u><br>ID (SF<35): 7%<br>Latent ID (SF 35–75): 13%<br>Iron replete (SF 76–370): 80%<br><br><u>Anaemia</u><br>2% infants were anaemic, but neither had ID.<br><br><u>Ethnic spread</u><br>Significant difference in Hb between Māori/Pasifika and all other ethnicities ($p$=0.05). No statistical significance for SF. | • Number of Māori infants (10.7%) does not reflect that of the general population<br>• Reporting of ethnic spread is prior to accounting for lost blood samples, so the true proportions not known.<br>• No follow-up to assess changes in iron status at 6 months or beyond |

| Study | Participants | Design | Iron intake and status measures | Main findings | Limitations |
|---|---|---|---|---|---|
| | | | | | |
| Daniels et al. (2018) (87) | *n*=206<br>Infants living in Dunedin, recruited at birth and followed till 12 months of age.<br><br>Premature infants excluded. | Randomised controlled trial.<br><br>Infants randomised into control (*n*=101) or the BLISS group (*n*=105).<br><br>Both groups received standard post-natal midwifery and Plunket care. The BLISS group received 8 additional visits from 0–9 months providing education on BLW modified to increase iron intake.<br><br>Outcome measures were iron intake at 7 and 12 months of age, and iron status at 12 months of age. | <u>Indices</u><br>Hb, SF, sTfR, body iron (mg/kg), CRP, AGP.<br><br><u>Cut-offs</u><br>Iron depleted: SF <15<br>ID: Body iron <0 mg/kg, Hb ≥110<br>IDA: Body iron <0 mg/kg, Hb <110<br><br><u>Exclusion</u><br>CRP: >5<br>AGP: >1<br><br><u>Dietary data</u><br>3-day weighed food record at 7 months and 12 months. | <u>Median Iron intake</u><br>7 months:<br>Control: 2.7 (1.3, 6.9)<br>BLISS: 3.0 (1.5, 7.3)<br><br>12 months:<br>Control: 5.3 (3.1, 8.4)<br>BLISS: 4.7 (3.1, 7.3)<br><br>Differences at both age points were non-significant.<br><br><u>Iron status (12 months)</u><br>Control group:<br>Iron depleted: 5%<br>ID: 7%<br>IDA: 5%<br><br>BLISS group:<br>Iron depleted: 3%<br>ID: 7%<br>IDA: 7%<br>Differences were non-significant. | • Not able to be stratified by ethnicity<br>• Not enough participants for statistical power<br>• Cut-off used for Hb extrapolated from adults and possibly too high for this age-group.<br>• Breastmilk intake estimated |

[a]AGP expressed as g/L, CRP expressed as mg/L, Hb concentration expressed in g/L, MCV expressed as fl, SF concentration expressed in µg/L, SI expressed as µmol/L, ZPP concentration expressed in µmol/mol haem.

Abbreviations: AGP – $\alpha_1$-acid glycoprotein; CRP – C-reactive protein; GUiNZ – Growing Up in New Zealand; Hb – haemoglobin; ID – iron deficiency; IDA – iron-deficiency anaemia; MCH – mean cell haemoglobin; MCV - mean corpuscular volume; NZE – New Zealand European; SF – serum ferritin; SI – serum iron; ST – serum transferrin; sTfR – serum transferrin receptor; TIBC – total iron-binding capacity; TS – transferrin saturation; ZPP – zinc protoporphyrin

[b]Mean expressed as mean (±SD); median expressed as median (IQR)

Of these studies, six investigate hospitalised infants which potentially presents a confounding factor of inflammation and infection (293, 429, 436-438, 441). A further two used inappropriate cut-off points for determining iron status: <105 for Hb (430, 431) which is much lower than the generally accepted cut-off of ≤108–110. Two studies did not adequately control for inflammation and infection (91, 432), and two more looked at neonatal iron status without follow-up (434, 435). One study was exclusively investigating the infants of Cambodian refugees (433), while another focussed on low birth weight infants (440). This leaves four observational studies and two RCTs that assess infant iron status using generally accepted parameters for iron indices and control for infection. One of the observational studies uses the same data set as another, so has been excluded from the summary below.

The differing indices used to define iron status makes comparison of these studies challenging. Wham (1996) looked at a small sample size of 53 children aged 9–24 months living in Auckland, and found that 13.5% had ID, and 20% had IDA (295). Inflammatory markers were not measured or controlled for in this study which could have resulted in an inaccurate rate of ID. Heath et. al. (2002) investigated iron intake and status of 74 children aged 9–24 months, and determined that 19% of 9–month-old infants and 22% of 12–month-old infants had ID. The rate of IDA was lower, with 7% of both 9– and 12–month old infants (91). However, this small cohort were not ethnically nor socio-economically diverse, and infection and inflammation were not controlled for. In 2004, Soh et. al. examined the iron status of 226 children aged 6–24 months in urban South Island centres, and categorised iron status into depleted iron stores, functional ID, ID, and IDA (92). In this cohort, 2.8% (6–11 months) and 5.0% (12–24 months) had ID. The prevalence of IDA in the 6–11–month age group was 5.6% and 2.5% in the 12–24–month age group. The overall group had a prevalence of 4.3% for ID and 3.5% for IDA using the cut-off of <10 mg/L for serum ferritin. CRP was controlled for as a confounding variable in this study, however the cut-off used was 10 mg/L, whereas the generally accepted cut-off in both other studies and the established reference range is >5 mg/L (442). This may have resulted in more children with concurrent infection being included in the analysis which has what effect on the findings. Grant et. al. (2007) found that in a cohort of 324 children aged 6–23 months in Auckland that 14% had ID and 6% had IDA (58). This study also controlled for infection using CRP, but used a conservative cut-off of >4 mg/L.

An RCT by Syzmlek-Gay (2009) sought to examine the impact of increased red meat or fortified milk on infant iron status at 20 weeks of age (439). The geometric mean baseline SF across all groups was 21.7 µg/L, suggesting borderline iron stores in the cohort. Additionally, infants with a baseline Hb of

<105 g/L or Hb <110 g/L accompanied by SF of ≤12 µg/L at baseline were excluded from the study. Neither intervention impacted on the prevalence of sub-optimal iron status, however both the red meat group and the fortified milk group had a significantly higher serum ferritin than the control group at 20 weeks ($p$<0.001 and $p$<0.033, respectively). The prevalence of ID was reported in figures only.

For the BLISS study, an RCT by Daniels et. al. (2018) designed a modified version of BLW with an aim to increase the intake of iron-rich foods by infants following this protocol. Infants living in Dunedin ($n$=206) were randomised into the modified BLW (or BLISS) group, and the other were a control group. Iron intake was measured at 7 and 12 months of age, and iron status was measured at 12 months of age. The BLISS group had a higher iron intake (3 mg/day) at 7 months than the control group (2.7 mg/day), but both intakes are well below the RDI of 11 mg/day. At 12 months, the control group had a higher iron intake (5.3 mg/day) than the BLISS group (4.7 mg/day). The differences were non-significant at all points. Regarding iron status, 7% of both the BLISS and control group had ID at 12 months, while 5% of the control group had IDA as compared to 7% of the BLISS group. These differences were also non-significant. Infection and inflammation were controlled for using CRP and AGP with appropriate cut-offs of >5 mg/day and >1 g/L, respectively. Baseline iron status was not recorded, so it is possible that one group may have had a lower or higher baseline iron overall than the other group.

**Iron Status in Māori and Pasifika Infants**

Of the studies described in table 12 in the previous section the iron status of Māori infants has been examined to varying degrees from as early as 1963 (58, 293, 428, 429, 434, 436). Pasifika infants have also been included in some of these studies (58, 293, 428, 430), but overall, robust up to date data is lacking for both Māori and Pasifika infants. Varying cut-offs and lack of control for infection and inflammation in many of these studies confound the findings, but what remains consistent amongst these studies is that Māori and Pasifika infants have a significantly higher rate of ID and IDA than NZE infants. One of the studies in the previous sect by Grant et. al. was adequately powered to stratify by ethnicity, with the results summarised in table 13. The cut-offs used in this study for IDA was Hb <110 g/L, plus two or more of ferritin <10 µg/L, MCV <73 fL, transferrin saturation <10%. Infants were excluded if CRP was >4 mg/L.

**Table 13.** Ethnic differences in iron status in NZ

| Ethnic group (n=324) | Iron deficiency present | |
|---|---|---|
| | Yes (*n*=49) | No (*n*=275) |
| New Zealand European (n=107) | 7 (7%) | 100 (93%) |
| Māori (n=85) | 17 (20%) | 68 (80%) |
| Pacific Island (n=106) | 18 (17%) | 88 (83%) |
| Other (n=26) | 7 (27%) | 19 (73%) |
| Total | 49 (14%) | 279 (86%) |

Note: Adapted from Grant CC, Wall CR, Brunt D, Crengle S, Scragg R. Population prevalence and risk factors for iron deficiency in Auckland, New Zealand. Journal of paediatrics and child health. 2007;43(7-8):532-8.

These findings are not unexpected considering that Māori and Pasifika infants bear a much higher burden of disease in infancy than NZE infants, with iron metabolism profoundly affected by inflammation via hepcidin. This is supported by a 2008 observational study by Wall et. al., using the same sample as Grant et. al., (2007) where dietary data in relation to the iron status of infants has been examined (428). The findings showed that there were fewer associations between dietary factors and iron status in Māori and Pasifika infants compared to NZE infants. In fact, Pasifika infants had a higher dietary intake of iron than the NZE infants. Another non-dietary factor impacting the iron status of Māori and Pasifika infants is the prevalence of a unique α-thalassemia trait. This variant of thalassemia occurs due to either damaged or missing genes for haemoglobin, resulting reduced levels of haemoglobin. Observational studies estimate the prevalence of α-thalassemia amongst Māori and Pasifika at 16.3–20% (389-392). Thus, non-dietary causes of ID and IDA in Māori and Pasifika infants are important to consider and require further research.

References

1. Tiatia J. Pacific cultural competencies: A literature review: Ministry of Health Wellington, New Zealand; 2008.
2. Statistics N. Longer Life, Better Health? Trends in health expectancy in New Zealand, 1996–2006. Wellington: Statistics New Zealand; 2009.
3. Kahukura T. Māori health chart book 2015: Wellington: Ministry of Health; 2010.
4. Anderson I, Robson B, Connolly M, Al-Yaman F, Bjertness E, King A, et al. Indigenous and tribal peoples' health (The Lancet–Lowitja Institute Global Collaboration): a population study. The Lancet. 2016;388(10040):131-57.
5. Came H. Sites of institutional racism in public health policy making in New Zealand. Social Science & Medicine. 2014;106:214-20.
6. Came H, Griffith D. Tackling racism as a "wicked" public health problem: enabling allies in anti-racism praxis. Social Science & Medicine. 2018;199:181-8.
7. Byrd WM, Clayton LA. Racial and ethnic disparities in healthcare: A background and history. Unequal treatment: Confronting Racial and Ethnic Disparities in Health Care. Washington, DC: National Academies Press; 2003. p. 455-527.
8. Rodney W. How Europe Underdeveloped Africa: Verso Books; 2018.
9. Alvarez AN, Liang CT, Neville HA. The cost of racism for people of color: Contextualizing experiences of discrimination: JSTOR; 2016.
10. Paradies Y. Colonisation, racism and indigenous health. Journal of Population Research. 2016;33(1):83-96.
11. Humpage L, Fleras A. Intersecting discourses: Closing the gaps, social justice and the Treaty of Waitangi. Social Policy Journal of New Zealand. 2001:37-54.
12. Hill S, Sarfati D, Blakely T, Robson B, Purdie G, Chen J, et al. Survival disparities in Indigenous and non-Indigenous New Zealanders with colon cancer: the role of patient comorbidity, treatment and health service factors. Journal of Epidemiology & Community Health. 2010;64(2):117-23.
13. Cram F. Measuring Māori children's wellbeing: A discussion paper. MAI Journal. 2019;8(1):16-32.
14. Cram F. Measuring Māori children's wellbeing. MAI Journal. 2019;8(1):16-32.
15. Glover M, Manaena-Biddle H, Waldon J, Cunningham C. Te whaangai uu, te reo o te aratika: Māori women and breastfeeding. Auckland, Massey University; 2008.
16. McBride-Henry K, Clendon J. Breastfeeding in New Zealand from colonisation until the year 1980: An historical review. New Zealand College of Midwives Journal. 2010(43).
17. Rata A. Te pītau o te tuakiri: Affirming Māori identities and promoting wellbeing in state secondary schools. 2012.
18. Abel S, Park J, Tipene-Leach D, Finau S, Lennan M. Infant care practices in New Zealand: A cross-cultural qualitative study. Social Science & Medicine. 2001;53(9):1135-48.
19. Laukau SH. Weaning practices in rural and urban Vava'u, Tonga Island. Pacific Health Dialog. 1994;1(2):22-6.
20. Tapera R, Harwood M, Anderson A. A qualitative Kaupapa Māori approach to understanding infant and young child feeding practices of Māori and Pacific grandparents in Auckland, New Zealand. Public Health Nutrition. 2017;20(6):1090-8.
21. Kidd J, Came H, Herbert S, McCreanor T. Māori and Tauiwi nurses' perspectives of anti-racism praxis: findings from a qualitative pilot study. AlterNative: An International Journal of Indigenous Peoples. 2020;16(4):387-94.
22. World Health Organization and UNICEF. Global strategy for infant and young child feeding: World Health Organization; 2003.
23. Ministry of Health. Food and Nutrition Guidelines for Healthy Infants and Toddlers (aged 0–2). Wellington, New Zealand: Ministry of Health; 2012.

24. Cichero J. Introducing solid foods using baby-led weaning vs. spoon-feeding: A focus on oral development, nutrient intake and quality of research to bring balance to the debate. Nutrition Bulletin. 2016;41(1):72-7.
25. Evans MS, Dunn KM. Pre-feeding skills. 2ª ed. Texas: Pro-Ed; 2000.
26. Gisel EG. Effect of food texture on the development of chewing of children between six months and two years of age. Developmental Medicine & Child Neurology. 1991;33(1):69-79.
27. Warren J. An update on complementary feeding. Nursing Children and Young People. 2018;30(6):38-47.
28. Rapley G. Baby-led weaning. Maternal and Infant Nutrition and Nurture: Controversies and Challenges. London: Quay Books; 2005. p. 275-98.
29. Cameron SL, Taylor RW, Heath ALM. Development and pilot testing of Baby-Led Introduction to Solids - a version of Baby-Led Weaning modified to address concerns about iron deficiency, growth faltering and choking. BMC Pediatrics. 2015;15(99):(26 August 2015)-(26 August ).
30. Rapley G, Murkett T. Baby-led weaning: Helping your baby to love good food: Random House; 2008.
31. Cameron SL, Heath A-LM, Taylor RW. Healthcare professionals' and mothers' knowledge of, attitudes to and experiences with, baby-led weaning: a content analysis study. British Medical Journal Open. 2012;2(6).
32. Fewtrell M, Bronsky J, Campoy C, Domellöf M, Embleton N, Mis NF, et al. Complementary feeding: a position paper by the European Society for Paediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN) Committee on Nutrition. Journal of Pediatric Gastroenterology and Nutrition. 2017;64(1):119-32.
33. D'Auria E, Bergamini M, Staiano A, Banderali G, Pendezza E, Penagini F, et al. Baby-led weaning: what a systematic review of the literature adds on. Italian Journal of Pediatrics. 2018;44.
34. Beauregard JL, Bates M, Cogswell ME, Nelson JM, Hamner HC. Nutrient content of squeeze pouch foods for infants and toddlers sold in the United States in 2015. Nutrients. 2019;11(7):1689.
35. Company N. Oh, Baby! Trends in the Baby Food and Diaper Markets Around the World. 2015.
36. Theurich MA. Perspective: novel commercial packaging and devices for complementary feeding. Advances in Nutrition. 2018;9(5):581-9.
37. Ventura AK, Worobey J. Early influences on the development of food preferences. Current Biology. 2013;23(9):R401-R8.
38. World Health Organization. The optimal duration of exclusive breastfeeding: Report of an expert consultation, Geneva, Switzerland, March 28-30, 2001. Geneva, Switzerland: Department of Nutrition for Health and Development (NHD) and Department of Child and Adolescent Health and Development (CAH); 2001.
39. Cameron SL, Heath A-LM, Taylor RW. How feasible is baby-led weaning as an approach to infant feeding? A review of the evidence. Nutrients. 2012;4(11):1575-609.
40. Brown A, Lee M. An exploration of experiences of mothers following a baby-led weaning style: developmental readiness for complementary foods. Maternal & Child nutrition. 2013;9(2):233-43.
41. EFSA Panel on Dietetic Products N, Allergies. Scientific Opinion on the appropriate age for introduction of complementary feeding of infants. European Food Safety Authority Journal. 2009;7(12):1423.
42. Roberts K, Rudolf M. A healthy start: a best practice handbook for health and early years practitioners: Henry; 2017.
43. Rudolph CD, Link DT. Feeding disorders in infants and children. Pediatric Clinics. 2002;49(1):97-112.
44. Hiiemae KM, Palmer J. Food transport and bolus formation during complete feeding sequences on foods of different initial consistency. Dysphagia. 1999;14(1):31-42.

45. Mishellany A, Woda A, Labas R, Peyron M-A. The challenge of mastication: preparing a bolus suitable for deglutition. Dysphagia. 2006;21(2):87-94.
46. Durvasula VS, O'Neill AC, Richter GT. Oropharyngeal dysphagia in children: mechanism, source, and management. Otolaryngologic Clinics of North America. 2014;47(5):691-720.
47. Rapata H, Heath A-LM, Wall C, Taylor R, Te Morenga L. Māori first foods: a Māori centred approach to understanding infant complementary feeding practices within Māori whānau. Kōtuitui: New Zealand Journal of Social Sciences Online. 2021:1-16.
48. Jones HM, Barber CC, Nikora LW, Middlemiss W. Māori child rearing and infant sleep practices. New Zealand Journal of Psychology. 2017;46(3):30-7.
49. Glasgow A, Rameka LK. Māori and Pacific traditional caregiving practices: Voices from the community. 2016.
50. Rush E, Rusk I. Food security for Pacific peoples in New Zealand: a report for the Obesity Action Coalition: Obesity Action Coalition; 2009.
51. Malolo M, Matenga-Smith To, Hughes R. The Staples We Eat. Noumea, New Caledonia; 1999.
52. Morton H. Becoming Tongan: An ethnography of childhood. Honolulu, USA: University of Hawai'i Press; 1996.
53. Lepowsky MA. Food taboos, malaria and dietary change: infant feeding and cultural adaptation on a Papua New Guinea Island. Ecology of Food and Nutrition. 1985;16(2):105-26.
54. Holmes W, Hoy D, Lockley A, Thammavongxay K, Bounnaphol S, Xeuatvongsa A, et al. Influences on maternal and child nutrition in the highlands of the northern Lao PDR. Asia Pacific Journal of Clinical Nutrition. 2007;16(3).
55. Radbill SX. Infant feeding through the ages. Clinical Pediatrics. 1981;20(10):613-21.
56. Steinkuller JS, Chan K, Rinehouse SE. Prechewing of food by adults and streptococcal pharyngitis in infants. The Journal of Pediatrics. 1992;120(4 Pt 1):563-4.
57. Oliver M, Rush E, Schluter P, Sundborn G, Iusitini L, Tautolo E-S, et al. An exploration of physical activity, nutrition, and body size in Pacific children. Pacific Health Dialog. 2011;17(2):176-87.
58. Grant CC, Wall CR, Brunt D, Crengle S, Scragg R. Population prevalence and risk factors for iron deficiency in Auckland, New Zealand. Journal of Paediatrics and Child Health. 2007;43(7-8):532-8.
59. Howe LD, Ellison-Loschmann L, Pearce N, Douwes J, Jeffreys M, Firestone R. Ethnic differences in risk factors for obesity in New Zealand infants. Journal of Epidemiology and Community Health. 2015;69(6):516-22.
60. Rapley G. Baby-led weaning: transitioning to solid foods at the baby's own pace. Community Practitioner. 2011;84(6):20.
61. Arden MA, Abbott RL. Experiences of baby-led weaning: trust, control and renegotiation. Maternal & Child Nutrition. 2015;11(4):829-44.
62. Brown A, Lee M. Early influences on child satiety-responsiveness: the role of weaning style. Pediatric Obesity. 2015;10(1):57-66.
63. Hanindita MH, Widjaja NA, Irawan R, Hidajat B. Comparison between baby led weaning and traditional spoon-feeding on iron status and growth in breastfed infants. Carpathian Journal of Food Science and Technology. 2019;5(11):96-100.
64. Morison BJ, Taylor RW, Haszard JJ, Schramm CJ, Williams Erickson L, Fangupo LJ, et al. How different are baby-led weaning and conventional complementary feeding? A cross-sectional study of infants aged 6-8months. British Medical Journal Open. 2016;6(5).
65. Siddiqui A. Object size as a determinant of grasping in infancy. The Journal of Genetic Psychology. 1995;156(3):345-58.
66. Von Hofsten C. Mastering reaching and grasping: The development of manual skills in infancy. Advances in Psychology. 61: Elsevier; 1989. p. 223-58.
67. Witherington DC. The development of prospective grasping control between 5 and 7 months: A longitudinal study. Infancy. 2005;7(2):143-61.

68. Townsend E, Pitchford NJ. Baby knows best? The impact of weaning style on food preferences and body mass index in early childhood in a case–controlled sample. British Medical Journal Open. 2012;2(1).
69. Cameron SL, Taylor RW, Heath A-LM. Parent-led or baby-led? Associations between complementary feeding practices and health-related behaviours in a survey of New Zealand families. British Medical Journal Open. 2013;3(12).
70. Brown A. No difference in self-reported frequency of choking between infants introduced to solid foods using a baby-led weaning or traditional spoon-feeding approach. Journal of Human Nutrition and Dietetics. 2018;31(4):496-504.
71. Plunket. Introducing Solids 2022 [Available from: https://www.plunket.org.nz/caring-for-your-child/feeding/solids/introducing-solid-foods/#stage-one-solids-around-six-months.
72. Ministry of Health. Food and Nutrition Guidelines for Healthy Infants and Toddlers (Aged 0-2): A background paper (4th Ed) - Partially Revised December 2012). Wellington, New Zealand: Ministry of Health; 2008.
73. Brown A, Lee M. Maternal control of child feeding during the weaning period: differences between mothers following a baby-led or standard weaning approach. Maternal and Child Health Journal. 2011;15(8):1265-71.
74. Carnell S, Wardle J. Measuring behavioural susceptibility to obesity: validation of the child eating behaviour questionnaire. Appetite. 2007;48(1):104-13.
75. Dogan E, Yilmaz G, Caylan N, Turgut M, Gokcay G, Oguz MM. Baby-led complementary feeding: Randomized controlled study. Pediatrics International. 2018;60(12):1073-80.
76. Fu X, Conlon CA, Haszard JJ, Beck KL, von Hurst PR, Taylor RW, et al. Food fussiness and early feeding characteristics of infants following Baby-Led Weaning and traditional spoon-feeding in New Zealand: An internet survey. Appetite. 2018;130:110-6.
77. Jones SW, Lee M, Brown A. Spoonfeeding is associated with increased infant weight but only amongst formula-fed infants. Maternal & Child Nutrition. 2020;16(3):e12941.
78. Kahraman A, Gümüş M, Binay Yaz Ş, Başbakkal Z. Baby-led weaning versus traditional weaning: The assessment of nutritional status in early childhood and maternal feeding practices in Turkey. Early Child Development and Care. 2020;190(5):615-24.
79. Taylor RW, Williams SM, Fangupo LJ, Wheeler BJ, Taylor BJ, Daniels L, et al. Effect of a Baby-Led Approach to Complementary Feeding on Infant Growth and Overweight A Randomized Clinical Trial. Journal of the American Medical Association Pediatrics. 2017;171(9):838-46.
80. Owen CG, Whincup PH, Orfei L, Chou Q-A, Rudnicka AR, Wathern AK, et al. Is body mass index before middle age related to coronary heart disease risk in later life? Evidence from observational studies. International Journal of Obesity. 2009;33(8):866-77.
81. Hannon TS, Rao G, Arslanian SA. Childhood obesity and type 2 diabetes mellitus. Pediatrics. 2005;116(2):473-80.
82. Russell-Mayhew S, McVey G, Bardick A, Ireland A. Mental health, wellness, and childhood overweight/obesity. Journal of Obesity. 2012;2012.
83. Scholder SvHK, Smith GD, Lawlor DA, Propper C, Windmeijer F. The effect of fat mass on educational attainment: examining the sensitivity to different identification strategies. Economics & Human Biology. 2012;10(4):405-18.
84. Matheson EM, King DE, Everett CJ. Healthy lifestyle habits and mortality in overweight and obese individuals. The Journal of the American Board of Family Medicine. 2012;25(1):9-15.
85. Grant CC, Wall CR, Yates R, Crengle S. Nutrition and indigenous health in New Zealand. Journal of Paediatrics and Child Health. 2010;46(9):479-82.
86. Craig E, Jackson C, Han D, Grimwood K, Committee NS. Monitoring the Health of New Zealand Children and Young People: Indicator Handbook. 2007.
87. Daniels L, Taylor RW, Williams SM, Gibson RS, Fleming EA, Wheeler BJ, et al. Impact of a modified version of baby led weaning on iron intake and status: a randomised controlled trial. British Medical Journal Open. 2018;8(6).

88. Daniels L, Taylor RW, Williams SM, Gibson RS, Samman S, Wheeler BJ, et al. Modified Version of Baby-Led Weaning Does Not Result in Lower Zinc Intake or Status in Infants: A Randomized Controlled Trial. Journal of the Academy of Nutrition and Dietetics. 2018;118(6).
89. Alpers B, Blackwell V, Clegg ME. Standard v. baby-led complementary feeding: a comparison of food and nutrient intakes in 6–12-month-old infants in the UK. Public Health Nutrition. 2019;22(15):2813-22.
90. Fairweather-Tait SJ. Iron nutrition in the UK: getting the balance right. Proceedings of the Nutrition Society. 2004;63(4):519-28.
91. Heath ALM, Reeves Tuttle C, Simons MS, Cleghorn CL, Parnell WR. Longitudinal study of diet and iron deficiency anaemia in infants during the first two years of life. Asia Pacific Journal of Clinical Nutrition. 2002;11(4):251-7.
92. Soh P, Ferguson EL, McKenzie JE, Homs M, Gibson RS. Iron deficiency and risk factors for lower iron stores in 6–24-month-old New Zealanders. European Journal of Clinical Nutrition. 2004;58(1):71-9.
93. Soh P, Ferguson EL, McKenzie JE, Skeaff S, Parnell W, Gibson RS. Dietary intakes of 6–24-month-old urban South Island New Zealand children in relation to biochemical iron status. Public Health Nutrition. 2002;5(2):339-46.
94. Malagelada C, Barba I, Accarino A, Molne L, Mendez S, Campos E, et al. Cognitive and hedonic responses to meal ingestion correlate with changes in circulating metabolites. Neurogastroenterology & Motility. 2016;28(12):1806-14.
95. Gal D. Let hunger Be your guide? Being hungry before a meal is associated with healthier levels of postmeal blood glucose. Journal of the Association for Consumer Research. 2016;1(1):15-24.
96. Gowey MA, Chandler-Laney P. Children's food and satiety responsiveness in association with post-prandial glucose following a standardized liquid meal. Clinical Obesity. 2018;8(1):39-42.
97. Joyce JL, Zimmer-Gembeck MJ. Parent feeding restriction and child weight. The mediating role of child disinhibited eating and the moderating role of the parenting context. Appetite. 2009;52(3):726-34.
98. Lee Y, Mitchell DC, Smiciklas-Wright H, Birch LL. Diet quality, nutrient intake, weight status, and feeding environments of girls meeting or exceeding recommendations for total dietary fat of the American Academy of Pediatrics. Pediatrics. 2001;107(6):e95-e.
99. Farrow C, Galloway A, Fraser K. Sibling eating behaviours and differential child feeding practices reported by parents. Appetite. 2009;52(2):307-12.
100. Galloway AT, Fiorito L, Lee Y, Birch LL. Parental pressure, dietary patterns, and weight status among girls who are “picky eaters”. Journal of the American Dietetic Association. 2005;105(4):541-8.
101. Ventura AK, Birch LL. Does parenting affect children's eating and weight status? International Journal of Behavioral Nutrition and Physical Activity. 2008;5(1):1-12.
102. Benton D. Role of parents in the determination of the food preferences of children and the development of obesity. International Journal of Obesity. 2004;28(7):858-69.
103. Savage JS, Hohman EE, Marini ME, Shelly A, Paul IM, Birch LL. INSIGHT responsive parenting intervention and infant feeding practices: randomized clinical trial. International Journal of Behavioral Nutrition and Physical Activity. 2018;15(1):1-11.
104. DiSantis KI, Hodges EA, Johnson SL, Fisher JO. The role of responsive feeding in overweight during infancy and toddlerhood: a systematic review. International Journal of Obesity. 2011;35(4):480-92.
105. Thompson AL, Wasser H, Nulty A, Bentley ME. Feeding style profiles are associated with maternal and infant characteristics and infant feeding practices and weight outcomes in African American mothers and infants. Appetite. 2021;160:105084.
106. Silva GA, Costa KA, Giugliani ER. Infant feeding: beyond the nutritional aspects. Jornal De Pediatria. 2016;92:2-7.

107. Adams S. Leadership Lessons From A Baby Food Disruptor 2013 [Available from: https://www.forbes.com/sites/susanadams/2013/07/31/leadership-lessons-from-a-baby-food-disruptor/?sh=5d8b631c5c15.
108. Finn K, Lenighan Y, Eldridge A, Kineman B, Pac S. Pouch Use Among Infants Does Not Impact Exposure to Other Forms of Fruits and Vegetables: Data from the Feeding Infants and Toddlers Study (FITS) 2016. Current Developments in Nutrition. 2020;4(Supplement_2):982-.
109. Balderrama J, Robbins R, Sweitzer S, Byrd-Williams C, Roberts-Gray C, Hoelscher D, et al. Prevalence of food squeeze pouches in infant and toddler packed lunches at early childcare centers. Journal of the Academy of Nutrition and Dietetics. 2017;117(9, Suppl.):A93-A.
110. Tedstone A, Nicholas J, MacKinlay B, Knowles B, Burton J, Owtram G, et al. Foods and drinks aimed at infants and young children: evidence and opportunities for action: June 2019. 2019.
111. Garcia AL, Curtin L, Ronquillo JD, Parrett A, Wright CM. Changes in the UK baby food market surveyed in 2013 and 2019: the rise of baby snacks and sweet/savoury foods. Archives of Disease in Childhood. 2020;105(12):1162-6.
112. Katiforis I, Fleming EA, Haszard JJ, Hape-Cramond T, Taylor RW, Heath A-LM. Energy, Sugars, Iron, and Vitamin B12 Content of Commercial Infant Food Pouches and Other Commercial Infant Foods on the New Zealand Market. Nutrients. 2021;13(2):657.
113. Koletzko B, Hirsch NL, Jewell JM, Caroli M, Breda JRDS, Weber M. Pureed fruit pouches for babies: child health under squeeze. Journal of Pediatric Gastroenterology and Nutrition. 2018;67(5):561-3.
114. Koletzko B, Bührer C, Ensenauer R, Jochum F, Kalhoff H, Lawrenz B, et al. Complementary foods in baby food pouches: position statement from the Nutrition Commission of the German Society for Pediatrics and Adolescent Medicine (DGKJ, eV). Molecular and Cellular Pediatrics. 2019;6(1):1-5.
115. Moumin NA, Green TJ, Golley RK, Netting MJ. Are the nutrient and textural properties of Australian commercial infant and toddler foods consistent with infant feeding advice? British Journal of Nutrition. 2020;124(7):754-60.
116. Klerks M, Roman S, Haro-Vicente JF, Bernal MJ, Sanchez-Siles LM. Healthier and more natural reformulated baby food pouches: will toddlers and their parents sensory accept them? Food Quality and Preference. 2022;99:104577-.
117. Beauregard JL, Bates M, Cogswell ME, Nelson JM, Hamner HC. Nutrient content of squeeze pouch foods for infants and toddlers sold in the United States in 2015. Nutrients. 2019;11(7):1689-.
118. Moding KJ, Ferrante MJ, Bellows LL, Bakke AJ, Hayes JE, Johnson SL. Nutritional content and ingredients of commercial infant and toddler food pouches compared with other packages available in the United States. Nutrition Today. 2019;54(6):305-12.
119. Padarath S, Gerritsen S, Mackay S. Nutritional aspects of commercially available complementary foods in New Zealand Supermarkets. Nutrients. 2020;12(10):2980.
120. Hutchinson J, Rippin H, Threapleton D, Jewell J, Kanamäe H, Salupuu K, et al. High sugar content of European commercial baby foods and proposed updates to existing recommendations. Maternal & Child Nutrition. 2021;17(1):e13020.
121. Lundkvist E, Sjostrom ES, Lundberg R, Silfverdal SA, West CE, Domellof M. Fruit pouch consumption and dietary patterns related to BMIz at 18 months of age. Nutrients. 2021;13(7):2265-.
122. Williams I, Maslin K. An analysis of squeezable pouch foods for infants and toddlers available in the UK. Journal of Human Nutrition and Dietetics. 2021;34(Suppl. S1):40-.
123. Harris LE, Chan JC. Infant feeding practices. American Journal of Diseases of Children. 1969;117(4):483-92.
124. Jones K, Pringle E, Taylor K, Young W. Infant feeding in coeliac disease. Gut. 1964;5(3):248.
125. Challacombe D. The incidence of coeliac disease and early weaning. Archives of Disease in Childhood. 1983;58(5):326.

126. Grimshaw K, Allen K, Edwards C, Beyer K, Boulay A, Van Der Aa L, et al. Infant feeding and allergy prevention: a review of current knowledge and recommendations. A EuroPrevall state of the art paper. Allergy. 2009;64(10):1407-16.
127. Wright CM, Parkinson K, Drewett R. Why are babies weaned early? Data from a prospective population based cohort study. Archives of Disease in Childhood. 2004;89(9):813-6.
128. Wilson AC, Forsyth JS, Greene SA, Irvine L, Hau C, Howie PW. Relation of infant diet to childhood health: seven year follow up of cohort of children in Dundee infant feeding study. British Medical Journal. 1998;316(7124):21-5.
129. Armentia A, Banuelos C, Arranz M, Del Villar V, Martín-Santos JM, Gil FM, et al. Early introduction of cereals into children's diets as a risk-factor for grass pollen asthma. Clinical & Experimental Allergy. 2001;31(8):1250-5.
130. Huh SY, Rifas-Shiman SL, Taveras EM, Oken E, Gillman MW. Timing of solid food introduction and risk of obesity in preschool-aged children. Pediatrics. 2011;127(3):e544-e51.
131. Kramer MS, Kakuma R. The optimal duration of exclusive breastfeeding. Protecting Infants Through Human Milk2004. p. 63-77.
132. Ministry of Health. Healthy Eating Guidelines for New Zealand Babies and Toddlers (0-2 years old). Wellington, New Zealand: Ministry of Health; 2021.
133. Ferreira SS, Marchioni DML, Wall CR, Gerritsen S, Teixeira JA, Grant CC, et al. Prevalence and maternal determinants of early and late introduction of complementary foods: results from the Growing up in New Zealand cohort study. British Journal of Nutrition. 2022:1-12.
134. Gontijo de Castro T, Gerritsen S, Wall CR, Grant C, Teixeira JA, Marchioni DM, et al. Infant Feeding in New Zealand: Adherence to Food and Nutrition Guidelines among the Growing Up in New Zealand Cohort. Ministry of Social Development; 2018. Report No.: 1988541484.
135. The Ministry of Health. Tier 1 Statistics 2019/2020: New Zealand Health Survey. Wellington: Ministry of Health; 2020.
136. Prasad N, Trenholme AA, Huang QS, Duque J, Grant CC, Newbern EC. Respiratory virus-related emergency department visits and hospitalizations among infants in New Zealand. The Pediatric Infectious Disease Journal. 2020;39(8):e176-e82.
137. Forsyth JS, Ogston SA, Clark A, Florey C, Howie PW. Relation between early introduction of solid food to infants and their weight and illnesses during the first two years of life. British Medical Journal. 1993;306(6892):1572-6.
138. Organization WH. Vitamin and mineral requirements in human nutrition: World Health Organization; 2004.
139. Dewey KG, Brown KH. Update on technical issues concerning complementary feeding of young children in developing countries and implications for intervention programs. Food and Nutrition Bulletin. 2003;24(1):5-28.
140. Koletzko B, Bhatia J, Bhutta ZA, Cooper P, Makrides M, Uauy R, et al. Pediatric nutrition in practice: Karger Medical and Scientific Publishers; 2015.
141. National Health and Medical Research Council. Nutrient reference values for Australia and New Zealand: including recommended dietary intakes. Version 1.1 updated March 2017 ed: National Health and Medical Research Council; 2006.
142. Maberly GF, Trowbridge F, Yip R, Sullivan K, West C. Programs against micronutrient malnutrition: ending hidden hunger. Annual Review of Public Health. 1994;15(1):277-301.
143. Grant CC, Wall CR, Gibbons MJ, Morton SM, Santosham M, Black RE. Child nutrition and lower respiratory tract disease burden in New Zealand: a global context for a national perspective. Journal of Paediatrics and Child Health. 2011;47(8):497-504.
144. Jin Y, Coad J, Zhou SJ, Skeaff S, Benn C, Kim N, et al. Mother and Infant Nutrition Investigation in New Zealand (MINI Project): Protocol for an observational longitudinal cohort study. Journal of Medical Internet Research Protocols. 2020;9(8):e18560.
145. Camara-Martos F, Amaro-López M. Influence of dietary factors on calcium bioavailability. Biological Trace Element Research. 2002;89(1):43-52.

146. Morgan JB, Williams P, Foote KD, Marriott LD. Do mothers understand healthy eating principles for low-birth-weight infants? Public Health Nutrition. 2006;9(6):700-6.
147. D'Souza S, Lakhani P, Waters H, Boardman K, Cinkotai K. Iron deficiency in ethnic minorities: associations with dietary fibre and phytate. Early Human Development. 1987;15(2):103-11.
148. EFSA Panel on Dietetic Products N, Allergies. Scientific Opinion on nutrient requirements and dietary intakes of infants and young children in the European Union. European Food Safety Authority Journal. 2013;11(10):3408.
149. Truswell AS. Recommended Nutrient Intakes: Australian Readings: Australian Professional Publications; 1990.
150. Health N, Council MR. Recommended dietary intakes for use in Australia. Australian Government Publishing Service; 1991. Report No.: 0644131802.
151. Intakes SCotSEoDR, Medicine Io, Board N. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride: Dietary Reference Intakes (Pap; 1997.
152. Thiamin R. Dietary reference intakes for thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin, and choline. 1998.
153. Krinsky NI, Beecher G, Burk R, Chan A, Erdman jJ, Jacob R, et al. Dietary reference intakes for vitamin C, vitamin E, selenium, and carotenoids. Institute of Medicine. 2000;19:95-185.
154. Russell RM, Beard JL, Cousins RJ, Dunn JT, Ferland G, Hambidge KM, et al. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC, USA: National Academies Press; 2001.
155. Food and Nutrition Board: Institute of Medicine. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC, USA: National Academy Press; 2002.
156. Electrolytes IoMPoDRIf, Water. DRI, dietary reference intakes for water, potassium, sodium, chloride, and sulfate: National Academy Press; 2004.
157. Values GBPoDR, Health GBDo. Dietary Reference Values for Food Energy and Nutrients for the United Kingdom: Report of the Panel on Dietary Reference Values of the Committee on Medical Aspects of Food Policy: HM Stationery Office; 1991.
158. Caballero B, Trugo LC, Finglas PM. Encyclopedia of food sciences and nutrition: Academic; 2003.
159. Medicine Io. Dietary Reference Intakes for Thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin and choline. Washington, DC: National Academies Press; 2000.
160. Institute of Medicine Food and Nutrition Board. Dietary reference intakes for thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin, and choline. Washington, DC: National Academies Press; 1999.
161. Garby L, Lammert O. Within-subjects between-days-and-weeks variation in energy expenditure at rest. Human Nutrition Clinical Nutrition. 1984;38(5):395-7.
162. Butte N. Energy requirements of infants: Background paper prepared for the joint FAO/WHO/UNU Expert Consultation on Energy in Human Nutrition. FAO Roma; 2001.
163. Butte NF, Wong WW, Hopkinson JM, Heinz CJ, Mehta NR, Smith EOB. Energy requirements derived from total energy expenditure and energy deposition during the first 2 y of life. The American Journal of Clinical Nutrition. 2000;72(6):1558-69.
164. Casale M, Von Hurst PR, Beck KL, Shultz S, Kruger MC, O'Brien W, et al. Lean mass and body fat percentage are contradictory predictors of bone mineral density in pre-menopausal Pacific Island women. Nutrients. 2016;8(8):470.
165. Rush E, Goedecke J, Jennings C, Micklesfield L, Dugas L, Lambert E, et al. BMI, fat and muscle differences in urban women of five ethnicities from two countries. International Journal of Obesity. 2007;31(8):1232-9.

166. Rush EC, Freitas I, Plank LD. Body size, body composition and fat distribution: comparative analysis of European, Maori, Pacific Island and Asian Indian adults. British Journal of Nutrition. 2009;102(4):632-41.
167. Simmons D. Relationship between maternal glycaemia and birth weight in glucose-tolerant women from different ethnic groups in New Zealand. Diabetic Medicine. 2007;24(3):240-4.
168. Rush E, Gao W, FUNAKI-TAHIFOTE M, Ngamata R, MATENGA-SMITH TI, Cassidy M, et al. Birth weight and growth trajectory to six years in Pacific children. International Journal of Pediatric Obesity. 2010;5(2):192-9.
169. University UN, Organization WH. Human Energy Requirements: Report of a Joint FAO/WHO/UNU Expert Consultation: Rome, 17-24 October 2001: Food & Agriculture Org.; 2004.
170. Shrimpton R, Victora CG, de Onis M, Lima RC, Blossner M, Clugston G. Worldwide timing of growth faltering: implications for nutritional interventions. Pediatrics. 2001;107(5):e75-e.
171. Black RE, Allen LH, Bhutta ZA, Caulfield LE, De Onis M, Ezzati M, et al. Maternal and child undernutrition: global and regional exposures and health consequences. The Lancet. 2008;371(9608):243-60.
172. Waterlow J. Classification and definition of protein-energy malnutrition. World Health Organization; 1976. Report No.: 0512-3038 Contract No.: 62.
173. Lupton JR, Brooks J, Butte NF, Caballero B, Flatt JP, Fried SK. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington DC, USA: The National Academy Press; 2002. p. 589-768.
174. Williams CM, editor Dietary fatty acids and human health. Annales De Zootechnie; 2000: EDP Sciences.
175. Albert CM, Hennekens CH, O'Donnell CJ, Ajani UA, Carey VJ, Willett WC, et al. Fish consumption and risk of sudden cardiac death. Journal of the American Medical Association. 1998;279(1):23-8.
176. Albert CM, Campos H, Stampfer MJ, Ridker PM, Manson JE, Willett WC, et al. Blood levels of long-chain n–3 fatty acids and the risk of sudden death. New England Journal of Medicine. 2002;346(15):1113-8.
177. Burr ML, Gilbert J, Holliday Ra, Elwood P, Fehily A, Rogers S, et al. Effects of changes in fat, fish, and fibre intakes on death and myocardial reinfarction: diet and reinfarction trial (DART). The Lancet. 1989;334(8666):757-61.
178. Dallongeville J, Yarnell J, Ducimetière P, Arveiler D, Ferrières J, Montaye Ml, et al. Fish consumption is associated with lower heart rates. Circulation. 2003;108(7):820-5.
179. Djoussé L, Pankow JS, Eckfeldt JH, Folsom AR, Hopkins PN, Province MA, et al. Relation between dietary linolenic acid and coronary artery disease in the National Heart, Lung, and Blood Institute Family Heart Study. The American Journal of Clinical Nutrition. 2001;74(5):612-9.
180. Dolecek TA. Epidemiological evidence of relationships between dietary polyunsaturated fatty acids and mortality in the multiple risk factor intervention trial. Proceedings of the Society for Experimental Biology and Medicine. 1992;200(2):177-82.
181. Hu FB, Stampfer MJ, Manson JE, Rimm EB, Wolk A, Colditz GA, et al. Dietary intake of α-linolenic acid and risk of fatal ischemic heart disease among women. The American Journal of Clinical Nutrition. 1999;69(5):890-7.
182. Pischon T, Hankinson SE, Hotamisligil GkS, Rifai N, Willett WC, Rimm EB. Habitual dietary intake of n-3 and n-6 fatty acids in relation to inflammatory markers among US men and women. Circulation. 2003;108(2):155-60.
183. World Health Organization. Diet, nutrition and the prevention of chronic diseases. World Health Organisation Technical Report Series. 2003;916(i-viii):1-149.
184. Gibbons A. Solving the brain's energy crisis. Science. 1998;280(5368):1345-7.
185. Sunehag AL, Haymond MW, Schanler RJ, Reeds PJ, Bier DM. Gluconeogenesis in very low birth weight infants receiving total parenteral nutrition. Diabetes. 1999;48(4):791-800.

186. Fretham SJ, Carlson ES, Georgieff MK. The role of iron in learning and memory. Advances in Nutrition. 2011;2(2):112-21.
187. Saarinen UM, Siimes MA, Dallman PR. Iron absorption in infants: High bioavailability ofbreast milk iron as indicated by the extrinsic tag method of iron absorption and by the concentration of serum ferritin. The Journal of Pediatrics. 1977;91(1):36-9.
188. Organization WH. Iron deficiency anemia. Assessment, prevention and control. A guide for programme managers. Geneva: World Health Organisation; 2011.
189. Monsen ER. Iron nutrition and absorption: dietary factors which impact iron bioavailability. Journal of the American Dietetic Association. 1988;88(7):786-90.
190. Dewey KG, Domellöf M, Cohen RJ, Landa Rivera L, Hernell O, Lonnerdal B. Iron supplementation affects growth and morbidity of breast-fed infants: results of a randomized trial in Sweden and Honduras. The Journal of Nutrition. 2002;132(11):3249-55.
191. Frassinetti S, Bronzetti GL, Caltavuturo L, Cini M, Della Croce C. The role of zinc in life: a review. Journal of Environmental Pathology, Toxicology and Oncology. 2006;25(3).
192. Bray TM, Bettger WJ. The physiological role of zinc as an antioxidant. Free Radical Biology and Medicine. 1990;8(3):281-91.
193. Bettger WJ, O'Dell BL. A critical physiological role of zinc in the structure and function of biomembranes. Life Sciences. 1981;28(13):1425-38.
194. Krebs NF, Miller LV, Michael Hambidge K. Zinc deficiency in infants and children: a review of its complex and synergistic interactions. Paediatrics and International Child Health. 2014;34(4):279-88.
195. Kambe T, Fukue K, Ishida R, Miyazaki S. Overview of inherited zinc deficiency in infants and children. Journal of Nutritional Science and Vitaminology. 2015;61(Supplement):S44-S6.
196. Sandström B, Lönnerdal B. Promoters and antagonists of zinc absorption. Zinc in Human Biology: Springer; 1989. p. 57-78.
197. Sandström B, Cederblad Å, Lönnerdal B. Zinc absorption from human milk, cow's milk, and infant formulas. American Journal of Diseases of Children. 1983;137(8):726-9.
198. Golden BE, Golden MH. Plasma zinc, rate of weight gain, and the energy cost of tissue deposition in children recovering from severe malnutrition on a cow's milk or soya protein based diet. The American Journal of Clinical Nutrition. 1981;34(5):892-9.
199. Sandström B, Keen CL, Lönnerdal B. An experimental model for studies of zinc bioavailability from milk and infant formulas using extrinsic labeling. The American Journal of Clinical Nutrition. 1983;38(3):420-8.
200. Abrams SA. Calcium absorption in infants and small children: methods of determination and recent findings. Nutrients. 2010;2(4):474-80.
201. Koo W. Osteopenia and rickets of prematurity. Fetal and Neonatal Physiology. 1998:2335-49.
202. Koo WK, Warren L. Calcium and bone health in infants. Neonatal Network. 2003;22(5):23-37.
203. Kohlenberg-Mueller K, Raschka L. Calcium balance in young adults on a vegan and lactovegetarian diet. Journal of Bone and Mineral Metabolism. 2003;21(1):28-33.
204. Larsson CL, Johansson GK. Dietary intake and nutritional status of young vegans and omnivores in Sweden. The American Journal of Clinical Nutrition. 2002;76(1):100-6.
205. New SA. Do vegetarians have a normal bone mass? Osteoporosis International. 2004;15(9):679-88.
206. Heaney RP, Recker RR, Stegman MR, Moy AJ. Calcium absorption in women: relationships to calcium intake, estrogen status, and age. Journal of Bone and Mineral Research. 1989;4(4):469-75.
207. Heaney RP, Recker RR, Weaver CM. Absorbability of calcium sources: the limited role of solubility. Calcified Tissue International. 1990;46(5):300-4.
208. Miller J, Smith D, Flora L, Slemenda C, Jiang X, Johnston Jr C. Calcium absorption from calcium carbonate and a new form of calcium (CCM) in healthy male and female adolescents. The American Journal of Clinical Nutrition. 1988;48(5):1291-4.

209. Smith KT, Heaney RP, Flora L, Hinders SM. Calcium absorption from a new calcium delivery system (CCM). Calcified Tissue International. 1987;41(6):351-2.
210. Heaney RP, Saville PD, Recker RR. Calcium absorption as a function of calcium intake. The Journal of Laboratory and Clinical Medicine. 1975;85(6):881-90.
211. Heaney R, Recker R, Hinders S. Variability of calcium absorption. The American Journal of Clinical Nutrition. 1988;47(2):262-4.
212. Atkinson S, Alston-Mills B, Lönnerdal B, Neville MC. Major minerals and ionic constituents of human and bovine milks. Handbook of Milk Composition: Elsevier; 1995. p. 593-622.
213. Abrams SA, Wen J, Stuff JE. Absorption of calcium, zinc, and iron from breast milk by five-to seven-month-old infants. Pediatric Research. 1997;41(3):384-90.
214. Specker BL, Beck A, Kalkwarf H, Ho M. Randomized trial of varying mineral intake on total body bone mineral accretion during the first year of life. Pediatrics. 1997;99(6):e12-e.
215. Gudas LJ. Cellular biology and biochemistry of the retinoids. The Retinoids. 1994:443-520.
216. Katz D, Drzymala M, Turton J, Hicks R, Hunt R, Palmer L, et al. Regulation of accessory cell function by retinoids in murine immune responses. British Journal of Experimental Pathology. 1987;68(3):343.
217. Trechsel U, Evêquoz V, Fleisch H. Stimulation of interleukin 1 and 3 production by retinoic acid in vitro. Biochemical Journal. 1985;230(2):339-44.
218. Zhao Z, Ross AC. Retinoic acid repletion restores the number of leukocytes and their subsets and stimulates natural cytotoxicity in vitamin A-deficient rats. The Journal of Nutrition. 1995;125(8):2064-73.
219. Morriss-Kay G, Sokolova N. Embryonic development and pattern formation. Federation of American Societies for Experimental Biology Journal. 1996;10(9):961-8.
220. Sommer A, West KP. Vitamin A deficiency: health, survival, and vision: Oxford University Press, USA; 1996.
221. Glasziou P, Mackerras D. Vitamin A supplementation in infectious diseases: a meta-analysis. British Medical Journal. 1993;306(6874):366-70.
222. Debelo H, Novotny JA, Ferruzzi MG. Vitamin A. Advances in Nutrition. 2017;8(6):992-4.
223. EFSA Panel on Food Additives Nutrient Sources added to Food. Scientific Opinion on the re-evaluation of mixed carotenes (E 160a (i)) and beta-carotene (E 160a (ii)) as a food additive. European Food Safety Authority Journal. 2012;10(3):2593.
224. Food and Nutrition Board: Institute of Medicine. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium and Carotenoids: National Academies Press; 2000.
225. Black MM. Effects of vitamin B12 and folate deficiency on brain development in children. Food and Nutrition Bulletin. 2008;29(2_suppl1):S126-S31.
226. Dror DK, Allen LH. Effect of vitamin B12 deficiency on neurodevelopment in infants: current knowledge and possible mechanisms. Nutrition Reviews. 2008;66(5):250-5.
227. Doscherholmen A, McMahon J, Ripley D. Vitamin B12 absorption from eggs. Proceedings of the Society for Experimental Biology and Medicine. 1975;149(4):987-90.
228. Kimura N, Fukuwatari T, Sasaki R, Hayakawa F, Shibata K. Vitamin intake in Japanese women college students. Journal of Nutritional Science and Vitaminology. 2003;49(3):149-55.
229. Watanabe F. Vitamin B12 sources and bioavailability. Experimental Biology and Medicine. 2007;232(10):1266-74.
230. Baghurst K, Record S, Leppard P. Red meat consumption in Australia: intakes, nutrient contribution and changes over time. Australian Journal of Nutrition and Dietetics. 2000;57(4 Suppl.).
231. Ministry of Health. NZ Food NZ Children: Key results of the 2002 National Children's Nutrition Survey. Wellington: Ministry of Health; 2003.
232. Dagnelie P-C, van Staveren WA, van den Berg H. Vitamin B-12 from algae appears not to be bioavailable. The American Journal of Clinical Nutrition. 1991;53(3):695-7.
233. Sarari AS, Farraj MA, Hamoudi W, Essawi TA. Helicobacter pylori, a causative agent of vitamin B12 deficiency. The Journal of Infection in Developing Countries. 2008;2(05):346-9.

234. Roumeliotis N, Dix D, Lipson A. Vitamin B12 deficiency in infants secondary to maternal causes. Canadian Medical Association Journal. 2012;184(14):1593-8.
235. Antony AC. Vegetarianism and vitamin B-12 (cobalamin) deficiency. The American Journal of Clinical Nutrition. 2003;78(1):3-6.
236. Specker BL, Miller D, Norman EJ, Greene H, Hayes K. Increased urinary methylmalonic acid excretion in breast-fed infants of vegetarian mothers and identification of an acceptable dietary source of vitamin B-12. The American Journal of Clinical Nutrition. 1988;47(1):89-92.
237. Gazdik Z, Zitka O, Petrlova J, Adam V, Zehnalek J, Horna A, et al. Determination of vitamin C (ascorbic acid) using high performance liquid chromatography coupled with electrochemical detection. Sensors. 2008;8(11):7097-112.
238. Frei B. Ascorbic acid protects lipids in human plasma and low-density lipoprotein against oxidative damage. The American Journal of Clinical Nutrition. 1991;54(6):1113S-8S.
239. Harris ED, Percival SS. A role for ascorbic acid in copper transport. The American Journal of Clinical Nutrition. 1991;54(6):1193S-7S.
240. Hallberg L. The role of vitamin C in improving the critical iron balance situation in women. International Journal for Vitamin and Nutrition research Supplement 1985;27:177-87.
241. Johnston CS, Meyer CG, Srilakshmi J. Vitamin C elevates red blood cell glutathione in healthy adults. The American Journal of Clinical Nutrition. 1993;58(1):103-5.
242. Halpner AD, Handelman GJ, Belmont CA, Harris JM, Blumberg JB. Protection by vitamin C of oxidant-induced loss of vitamin E in rat hepatocytes. The Journal of Nutritional Biochemistry. 1998;9(6):355-9.
243. Stokes P, Melikian V, Leeming R, Portman-Graham H, Blair J, Cooke W. Folate metabolism in scurvy. The American Journal of Clinical Nutrition. 1975;28(2):126-9.
244. Johnston CS, Cox SK. Plasma-saturating intakes of vitamin C confer maximal antioxidant protection to plasma. Journal of the American College of Nutrition. 2001;20(6):623-7.
245. Sánchez-Moreno C, Dashe JF, Scott T, Thaler D, Folstein MF, Martin A. Decreased levels of plasma vitamin C and increased concentrations of inflammatory and oxidative stress markers after stroke. Stroke. 2004;35(1):163-8.
246. Hunt C, Chakravorty N, Annan G, Habibzadeh N, Schorah C. The clinical effects of vitamin C supplementation in elderly hospitalised patients with acute respiratory infections. International Journal for Vitamin and Nutrition Research. 1994;64(3):212-9.
247. Hemilä H, Chalker E. Vitamin C can shorten the length of stay in the ICU: a meta-analysis. Nutrients. 2019;11(4):708.
248. Hemilä H, Chalker E. Vitamin C may reduce the duration of mechanical ventilation in critically ill patients: a meta-regression analysis. Journal of Intensive Care. 2020;8(1):1-9.
249. Tveden-Nyborg P, Lykkesfeldt J. Does vitamin C deficiency result in impaired brain development in infants? Redox Report. 2009;14(1):2-6.
250. Goldsmith GA. Human requirements for vitamin C and its use in clinical medicine. Annals of the New York Academy of Sciences. 1961;92(1):230-45.
251. McLaren DS. A colour atlas of nutritional disorders. London: Wolfe Medical Publications; 1992.
252. Uğur H, Çatak J, Mızrak ÖF, Çebi N, Yaman M. Determination and evaluation of in vitro bioaccessibility of added vitamin C in commercially available fruit-, vegetable-, and cereal-based baby foods. Food Chemistry. 2020;330:127166.
253. Kallner A, Hartmann D, Hornig D. Steady-state turnover and body pool of ascorbic acid in man. The American Journal of Clinical Nutrition. 1979;32(3):530-9.
254. Olson JA, Hodges RE. Recommended dietary intakes (RDI) of vitamin C in humans. The American Journal of Clinical Nutrition. 1987;45(4):693-703.
255. Irwin MI, Hutchins BK. A conspectus of research on vitamin C requirements of man. The Journal of Nutrition. 1976;106(6):821-79.

256. Brown K, Dewey K, Allen L. Complementary feeding of young children in developing countries: a review of current scientific knowledge. Geneva: World Health Organisation; 1998.
257. Ortiz-Andrellucchi A, Henríquez-Sánchez P, Sánchez-Villegas A, Pena-Quintana L, Mendez M, Serra-Majem L. Dietary assessment methods for micronutrient intake in infants, children and adolescents: a systematic review. British Journal of Nutrition. 2009;102(S1):S87-S117.
258. Burrows TL, Martin RJ, Collins CE. A systematic review of the validity of dietary assessment methods in children when compared with the method of doubly labeled water. Journal of the American Dietetic Association. 2010;110(10):1501-10.
259. Bingham SA, Gill C, Welch A, Day K, Cassidy A, Khaw K, et al. Comparison of dietary assessment methods in nutritional epidemiology: weighed records v. 24 h recalls, food-frequency questionnaires and estimated-diet records. British Journal of Nutrition. 1994;72(4):619-43.
260. Judd AL, Beck KL, McKinlay C, Jackson A, Conlon CA. Validation of a Complementary Food Frequency Questionnaire to assess infant nutrient intake. Maternal & Child Nutrition. 2020;16(1):e12879.
261. Willett W, Lenart E. Folic acid and neural tube defects. MONOGRAPHS IN EPIDEMIOLOGY AND BIOSTATISTICS. 2013;40(3):468-86.
262. Smithers G, Gregory JR, Bates CJ, Prentice A, Jackson LV, Wenlock R. The National Diet and Nutrition Survey: young people aged 4–18 years. Nutrition Bulletin. 2000;25(2):105-11.
263. Raper N, Perloff B, Ingwersen L, Steinfeldt L, Anand J. An overview of USDA's dietary intake data system. Journal of Food Composition and Analysis. 2004;17(3-4):545-55.
264. Moshfegh A, Goldman J, Lacomb R, Perloff B, Cleveland L. Research results using the new automated multiple-pass method. Federation of American Societies for Experimental Biology Journal. 2001;15(4):A278-A.
265. Dao MC, Subar AF, Warthon-Medina M, Cade JE, Burrows T, Golley RK, et al. Dietary assessment toolkits: an overview. Public Health Nutrition. 2019;22(3):404-18.
266. Kittisakmontri K, Lanigan J, Sangcakul A, Tim-Aroon T, Meemaew P, Wangaueattachon K, et al. Comparison of 24-hour recall and 3-day food records during the complementary feeding period in Thai infants and evaluation of plasma amino acids as markers of protein intake. Nutrients. 2021;13(2):653.
267. Trolle E, Amiano P, Ege M, Bower E, Lioret S, Brants H, et al. Feasibility of repeated 24-h dietary recalls combined with a food-recording booklet, using EPIC-Soft, among preschoolers. European journal of clinical nutrition. 2011;65(1):S84-S6.
268. Vossenaar M, Solomons NW, Monterrosa E, van Zutphen KG. Nutrient density as a dimension of dietary quality: Sight Life; 2018. 172-6 p.
269. Solomons N, Vossenaar M. Nutrient density in complementary feeding of infants and toddlers. European Journal of Clinical Nutrition. 2013;67(5):501-6.
270. Hotz C, Gibson R. Complementary feeding practices and dietary intakes from complementary foods amongst weanlings in rural Malawi. European Journal of Clinical Nutrition. 2001;55(10):841-9.
271. Faber M. Complementary foods consumed by 6–12-month-old rural infants in South Africa are inadequate in micronutrients. Public Health Nutrition. 2005;8(4):373-81.
272. Campos R, Hernandez L, Soto-Mendez MJ, Vossenaar M, Solomons NW. Contribution of complementary food nutrients to estimated total nutrient intakes for rural Guatemalan infants in the second semester of life. Asia Pacific Journal of Clinical Nutrition. 2010;19(4):481-90.
273. Friel JK, Hanning RM, Isaak CA, Prowse D, Miller AC. Canadian infants' nutrient intakes from complementary foods during the first year of life. BMC Pediatrics. 2010;10(1):1-11.
274. James K F, Corinne A I, Rhona H, Angela M. Complementary food consumption of Canadian infants. The Open Nutrition Journal. 2009;3(1).
275. Déré K, Djohan Y, Koffi K, Manhan K, Niamké A, Tiahou G. Individual dietary diversity score for diabetic and hypertensive patients in cote d'Ivoire. International Journal of Nutrition. 2016;2(1):38-47.

276. Korohina E, editor Food Frequency Questionnaire: Is It Time for a Re-Vamp? A Kaupapa Māori Critique of Dietary Recall and Assessment Tools. Medical Sciences Forum; 2022: MDPI.
277. Daniels L, Heath A-LM, Williams SM, Cameron SL, Fleming EA, Taylor BJ, et al. Baby-Led Introduction to SolidS (BLISS) study: a randomised controlled trial of a baby-led approach to complementary feeding. BMC Pediatrics. 2015;15.
278. Erickson LW, Taylor RW, Haszard JJ, Fleming EA, Daniels L, Morison BJ, et al. Impact of a Modified Version of Baby-Led Weaning on Infant Food and Nutrient Intakes: The BLISS Randomized Controlled Trial. Nutrients. 2018;10(6).
279. Castro TGd, Gerritsen S, Wall C, Grant C, Teixeira JA, Marchioni DM, et al. Infant Feeding in New Zealand: adherence to Food and Nutrition Guidelines among the Growing up in New Zealand Cohort. Wellington, NZ: Ministry of Social Development.; 2018.
280. Lopez A, Cacoub P, Macdougall IC, Peyrin-Biroulet L. Iron deficiency anaemia. The Lancet. 2016;387(10021):907-16.
281. Bruner AB, Joffe A, Duggan AK, Casella JF, Brandt J. Randomised study of cognitive effects of iron supplementation in non-anaemic iron-deficient adolescent girls. The Lancet. 1996;348(9033):992-6.
282. Lozoff B, Jimenez E, Smith JB. Double burden of iron deficiency in infancy and low socioeconomic status: a longitudinal analysis of cognitive test scores to age 19 years. Archives of Pediatrics & Adolescent Medicine. 2006;160(11):1108-13.
283. Beard JL. Why Iron Deficiency Is Important in Infant Development. The Journal of Nutrition. 2008;138(12):2534-6.
284. Mantadakis E, Chatzimichael E, Zikidou P. Iron deficiency anemia in children residing in high and low-income countries: risk factors, prevention, diagnosis and therapy. Mediterranean Journal of Hematology and Infectious Diseases. 2020;12(1).
285. Domellof M, Braegger C, Campoy C, Colomb V, Decsi T, Fewtrell M, et al. Iron Requirements of Infants and Toddlers. Journal of Pediatric Gastroenterology and Nutrition. 2014;58(1):119-29.
286. Daly A, MacDonald A, Aukett A, Williams J, Wolf A, Davidson J, et al. Prevention of anaemia in inner city toddlers by an iron supplemented cows' milk formula. Archives of Disease in Childhood. 1996;75(1):9-16.
287. Gunnarsson BS, Thorsdottir I, Palsson G. Iron status in 2-year-old Icelandic children and associations with dietary intake and growth. European Journal of Clinical Nutrition. 2004;58(6):901-6.
288. Archer NM, Brugnara C. Diagnosis of iron-deficient states. Critical Reviews in Clinical Laboratory Sciences. 2015;52(5):256-72.
289. Berglund S, Westrup B, Domellof M. Iron Supplements Reduce the Risk of Iron Deficiency Anemia in Marginally Low Birth Weight Infants. Pediatrics. 2010;126(4):E874-E83.
290. Iannotti LL, Tielsch JM, Black MM, Black RE. Iron supplementation in early childhood: health benefits and risks. The American Journal of Clinical Nutrition. 2006;84(6):1261-76.
291. Male C, Persson LA, Freeman V, Guerra A, van't Hof MA, Haschke F, et al. Prevalence of iron deficiency in 12-mo-old infants from 11 European areas and influence of dietary factors on iron status (Euro-Growth study). Acta Paediatrica. 2001;90(5):492-8.
292. Park K, Kersey M, Geppert J, Story M, Cutts D, Himes JH. Household food insecurity is a risk factor for iron-deficiency anaemia in a multi-ethnic, low-income sample of infants and toddlers. Public Health Nutrition. 2009;12(11):2120-8.
293. Grant C, Wall C, Wilson C, Taua N. Risk factors for iron deficiency in a hospitalized urban New Zealand population. Journal of Paediatrics and Child Health. 2003;39(2):100-6.
294. Quezada-Pinedo HG, Cassel F, Duijts L, Muckenthaler MU, Gassmann M, Jaddoe VW, et al. Maternal iron status in pregnancy and child health outcomes after birth: a systematic review and meta-analysis. Nutrients. 2021;13(7):2221.
295. Wham C. Dietary iron intake and iron status of young children. Asia Pacific Journal of Clinical Nutrition. 1996;5:196-200.

296. Davidsson L, Kastenmayer P, Yuen M, Lönnerdal B, Hurrell RF. Influence of lactoferrin on iron absorption from human milk in infants. Pediatric Research. 1994;35(1):117-24.
297. Collings R, Harvey LJ, Hooper L, Hurst R, Brown TJ, Ansett J, et al. The absorption of iron from whole diets: a systematic review. The American Journal of Clinical Nutrition. 2013;98(1):65-81.
298. Lane DJ, Richardson DR. The active role of vitamin C in mammalian iron metabolism: much more than just enhanced iron absorption! Free Radical Biology and Medicine. 2014;75:69-83.
299. Lynch SR, Cook JD. Interaction of vitamin C and iron. Annals of the New York Academy of Sciences. 1980;355(1):32-44.
300. WHO, UNICEF, UNU. Iron Deficiency Anemia: Assessment, Prevention, and Control—A Guide for Program Managers 2001 [Available from: http://www.who.int/nutrition/publications/micronutrients/anaemia_iron_deficiency/WHO_NHD_01.3/en/index.html.
301. World Health Organization. Haemoglobin concentrations for the diagnosis of anaemia and assessment of severity. World Health Organisation; 2011 2011.
302. Grant C, Prestidge T, Noel H. Iron Deficiency Auckland, New Zealand2016 [cited 2022 8/2/2022]. Available from: https://starship.org.nz/guidelines/iron-deficiency/.
303. Cullis JO, Fitzsimons EJ, Griffiths WJ, Tsochatzis E, Thomas DW, Haematology BSf. Investigation and management of a raised serum ferritin. British Journal of Haematology. 2018;181(3):331-40.
304. World Health Organization. Prevention, and Control: A guide for programme managers. 2001.
305. Organization WH. WHO guideline on use of ferritin concentrations to assess iron status in populations: World Health Organization; 2020.
306. Diana A, Haszard JJ, Purnamasari DM, Nurulazmi I, Luftimas DE, Rahmania S, et al. Iron, zinc, vitamin A and selenium status in a cohort of Indonesian infants after adjusting for inflammation using several different approaches. British Journal of Nutrition. 2017;118(10):830-9.
307. Suchdev PS, Namaste SM, Aaron GJ, Raiten DJ, Brown KH, Flores-Ayala R, et al. Overview of the biomarkers reflecting inflammation and nutritional determinants of anemia (BRINDA) project. Advances in Nutrition. 2016;7(2):349-56.
308. Mast AE, Blinder MA, Gronowski AM, Chumley C, Scott MG. Clinical utility of the soluble transferrin receptor and comparison with serum ferritin in several populations. Clinical Chemistry. 1998;44(1):45-51.
309. Beguin Y, Lampertz S, De Groote D, Igot D, Malaise M, Fillet G. Soluble CD23 and other receptors (CD4, CD8, CD25, CD71) in serum of patients with chronic lymphocytic leukemia. Leukemia. 1993;7(12):2019-25.
310. Huebers HA, Beguin Y, Pootrakul P, Einspahr D, Finch CA. Intact transferrin receptors in human plasma and their relation to erythropoiesis. Blood, The Journal of the American Society of Hematology. 1990;75(1):102-7.
311. Infusino I, Braga F, Dolci A, Panteghini M. Soluble transferrin receptor (sTfR) and sTfR/log ferritin index for the diagnosis of iron-deficiency anemia a meta-analysis. American Journal of Clinical Pathology. 2012;138(5):642-9.
312. Braga F, Infusino I, Dolci A, Panteghini M. Soluble transferrin receptor in complicated anemia. Clinica Chimica Acta. 2014;431:143-7.
313. Punnonen K, Irjala K, Rajamäki A. Serum transferrin receptor and its ratio to serum ferritin in the diagnosis of iron deficiency. Blood, The Journal of the American Society of Hematology. 1997;89(3):1052-7.
314. Cook JD, Flowers CH, Skikne BS. The quantitative assessment of body iron. Blood, The Journal of the American Society of Hematology. 2003;101(9):3359-63.
315. Cogswell ME, Looker AC, Pfeiffer CM, Cook JD, Lacher DA, Beard JL, et al. Assessment of iron deficiency in US preschool children and nonpregnant females of childbearing age: National Health

and Nutrition Examination Survey 2003–2006. The American Journal of Clinical Nutrition. 2009;89(5):1334-42.
316. Thompson WG, Meola T, Lipkin M, Freedman ML. Red cell distribution width, mean corpuscular volume, and transferrin saturation in the diagnosis of iron deficiency. Archives of Internal Medicine. 1988;148(10):2128-30.
317. Labbé RF, Dewanji A. Iron assessment tests: transferrin receptor vis-a-vis zinc protoporphyrin. Clinical Biochemistry. 2004;37(3):165-74.
318. Das S, Philip KJ. Evaluation of iron status: Zinc protoporphyrin vis-a-vis bone marrow iron stores. Indian Journal of Pathology and Microbiology. 2008;51(1):105.
319. Jolobe O. Prevalence of hypochromia (without microcytosis) vs microcytosis (without hypochromia) in iron deficiency. Clinical & Laboratory Haematology. 2000;22(2):79-80.
320. Mann J, Truswell AS. Essentials of Human Nutrition: Oxford University Press; 2017.
321. Cusick SE, Georgieff MK. The role of nutrition in brain development: the golden opportunity of the “first 1000 days”. The Journal of Pediatrics. 2016;175:16-21.
322. Lozoff B. Iron deficiency and child development. Food and Nutrition Bulletin. 2007;28(4_suppl4):S560-S71.
323. Brotanek JM, Halterman JS, Auinger P, Flores G, Weitzman M. Iron deficiency, prolonged bottle-feeding, and racial/ethnic disparities in young children. Archives of Pediatrics & Adolescent Medicine. 2005;159(11):1038-42.
324. Idjradinata P, Pollitt E. Reversal of developmental delays in iron-deficient anaemic infants treated with iron. The Lancet. 1993;341(8836):1-4.
325. Lozoff B, Brittenham GM, Viteri FE, Wolf AW, Urrutia JJ. The effects of short-term oral iron therapy on developmental deficits in iron-deficient anemic infants. The Journal of Pediatrics. 1982;100(3):351-7.
326. Lozoff B, Brittenham GM, Wolf AW, McClish DK, Kuhnert PM, Jimenez E, et al. Iron deficiency anemia and iron therapy effects on infant developmental test performance. Pediatrics. 1987;79(6):981-95.
327. Lozoff B, Wolf AW, Jimenez E. Iron-deficiency anemia and infant development: effects of extended oral iron therapy. The Journal of Pediatrics. 1996;129(3):382-9.
328. Walter T, Kovalskys J, Stekel A. Effect of mild iron deficiency on infant mentaldevelopment scores. The Journal of Pediatrics. 1983;102(4):519-22.
329. Walter T, De Andraca I, Chadud P, Perales CG. Iron deficiency anemia: adverse effects on infant psychomotor development. Pediatrics. 1989;84(1):7-17.
330. McCann JC, Ames BN. An overview of evidence for a causal relation between iron deficiency during development and deficits in cognitive or behavioral function. The American Journal of Clinical Nutrition. 2007;85(4):931-45.
331. Stelle I, Kalea AZ, Pereira DI. Iron deficiency anaemia: experiences and challenges. Proceedings of the Nutrition Society. 2019;78(1):19-26.
332. Lozoff B, Beard J, Connor J, Felt B, Georgieff M, Schallert T. Long-lasting neural and behavioral effects of iron deficiency in infancy. Nutrition Reviews. 2006;64(suppl_2):S34-S43.
333. Roncagliolo M, Garrido M, Walter T, Peirano P, Lozoff B. Evidence of altered central nervous system development in infants with iron deficiency anemia at 6 mo: delayed maturation of auditory brainstem responses. The American Journal of Clinical Nutrition. 1998;68(3):683-90.
334. Li Y, Wang H, Wang W. The effect of iron deficiency anemia on the auditory brainstem response in infant. Chinese Medical Journal. 1994;74(6):367-9, 92.
335. Algarin C, Peirano P, Garrido M, Lozoff B, editors. Iron deficient anemic infants have decreased rapid eye movements density in active sleep. Sleep; 2003: Amer Academy Sleep Medicine Academy.
336. Burden M, Armony-Sivan R, Westerlund A, editors. Preliminary findings from an ERP study of memory processing in iron deficient infants. International Conference on Information Systems; 2004.

337. Abrams J, Jacobson S, Lozoff B, Nelson C. EEG correlates of iron deficiency in infancy. Pediatric Research. 2005;57:1287.
338. Palti H, Pevsner B, Adler B. Does anemia in infancy affect achievement on developmental and intelligence tests? Human Biology. 1983:183-94.
339. Wasserman G, Graziano J, Factor-Litvak P, Popovac D, Morina N, Musabegovic A, et al. Consequences of lead exposure and iron supplementation on childhood development at age 4 years. Neurotoxicology and Teratology. 1994;16(3):233-40.
340. Wasserman G, Graziano J, Factor-Litvak P, Popovac D, Morina N, Musabegovic A, et al. Independent effects of lead exposure and iron deficiency anemia on developmental outcome at age 2 years. The Journal of Pediatrics. 1992;121(5):695-703.
341. Hurtado EK, Claussen AH, Scott KG. Early childhood anemia and mild or moderate mental retardation. The American Journal of Clinical Nutrition. 1999;69(1):115-9.
342. Lozoff B, Jimenez E, Hagen J, Mollen E, Wolf AW. Poorer behavioral and developmental outcome more than 10 years after treatment for iron deficiency in infancy. Pediatrics. 2000;105(4):e51-e.
343. Cantwell RJ, Cleveland WW. The long term neurological sequelae of anemia in infancy. Pediatric Research. 1974;8(4):342-.
344. Palti H, Meijer A, Adler B. Learning achievement and behavior at school of anemic and non-anemic infants. Early Human Development. 1985;10(3-4):217-23.
345. Antunes H. Iron Deficiency Anaemia in Infants - A Prospective Neurodevelopment Evaluation. Faculty of Medicine, University of Portugal; 2004.
346. Baker RD, Greer FR. Diagnosis and prevention of iron deficiency and iron-deficiency anemia in infants and young children (0–3 years of age). Pediatrics. 2010;126(5):1040-50.
347. van Lamoen A. Addressing the literacy crisis in Aotearoa New Zealand. 2022.
348. Clark A, Huang T. Survey of adult skills: Results for Auckland: Research and Evaluation Unit, Auckland Council; 2018.
349. Jones M, Satherley P. Maori Adults' Literacy, Numeracy and Problem Solving Skills. Wellington, NZ: Ministry of Education; 2018.
350. Leung A, Chan KW. Iron deficiency anemia. Advances in Pediatrics. 2001;48:385-408.
351. Kleinman RE. Pediatric nutrition handbook. Actividad Dietética. 2009;13(1):46.
352. Choi J, Kim C, Pai S. Erythropoietic activity and soluble transferrin receptor level in neonates and maternal blood. Acta Paediatrica. 2000;89(6):675-9.
353. Preziosi P, Prual A, Galan P, Daouda H, Boureima H, Hercberg S. Effect of iron supplementation on the iron status of pregnant women: consequences for newborns. The American Journal of Clinical Nutrition. 1997;66(5):1178-82.
354. Oski FA. Iron deficiency in infancy and childhood. New England Journal of Medicine. 1993;329(3):190-3.
355. Rao R, Georgieff M. Perinatal aspects of iron metabolism. Acta Paediatrica. 2002;91:124-9.
356. Van Rheenen P, Brabin BJ. Late umbilical cord-clamping as an intervention for reducing iron deficiency anaemia in term infants in developing and industrialised countries: a systematic review. Annals of Tropical Paediatrics. 2004;24(1):3-16.
357. Bothwell TH. Overview and mechanisms of iron regulation. Nutrition Reviews. 1995;53(9):237-45.
358. Boccio JR, Iyengar V. Iron deficiency. Biological Trace Element Research. 2003;94(1):1-31.
359. Jiang T, Jeter JM, Nelson SE, Ziegler EE. Intestinal blood loss during cow milk feeding in older infants: quantitative measurements. Archives of Pediatrics & Adolescent Medicine. 2000;154(7):673-8.
360. Thorsdottir I, Gunnarsson BS, Atladottir H, Michaelsen K, Palsson G. Iron status at 12 months of age—effects of body size, growth and diet in a population with high birth weight. European Journal of Clinical Nutrition. 2003;57(4):505-13.

361. Perry GS, Byers T, Yip R, Margen S. Iron nutrition does not account for the hemoglobin differences between blacks and whites. The Journal of Nutrition. 1992;122(7):1417-24.
362. Domellöf M, Lönnerdal B, Dewey KG, Cohen RJ, Rivera LL, Hernell O. Sex differences in iron status during infancy. Pediatrics. 2002;110(3):545-52.
363. Trumbo P, Yates AA, Schlicker S, Poos M. Dietary reference intakes. Journal of the American Dietetic Association. 2001;101(3):294-.
364. Cutland CL, Lackritz EM, Mallett-Moore T, Bardají A, Chandrasekaran R, Lahariya C, et al. Low birth weight: Case definition & guidelines for data collection, analysis, and presentation of maternal immunization safety data. Vaccine. 2017;35(48Part A):6492.
365. Berglund S, Domellöf M. Meeting iron needs for infants and children. Current Opinion in Clinical Nutrition & Metabolic Care. 2014;17(3):267-72.
366. Newborn Services Clinical Practice Committee. Ferrous Sulphate 2018 [Available from: https://starship.org.nz/guidelines/ferrous-sulphate/.
367. Kassebaum NJ, Jasrasaria R, Naghavi M, Wulf SK, Johns N, Lozano R, et al. A systematic analysis of global anemia burden from 1990 to 2010. Blood, The Journal of the American Society of Hematology. 2014;123(5):615-24.
368. De-Regil LM, Jefferds MED, Sylvetsky AC, Dowswell T. Intermittent iron supplementation for improving nutrition and development in children under 12 years of age. Cochrane Database of Systematic Reviews. 2011(12).
369. Neuberger A, Okebe J, Yahav D, Paul M. Oral iron supplements for children in malaria-endemic areas. Cochrane Database of Systematic Reviews. 2016(2).
370. Koletzko B, Baker S, Cleghorn G, Neto UF, Gopalan S, Hernell O, et al. Global standard for the composition of infant formula: recommendations of an ESPGHAN coordinated international expert group. Journal of Pediatric Gastroenterology and Nutrition. 2005;41(5):584-99.
371. Anyon C, Clarkson K. Cows' milk: a cause of iron-deficiency anaemia in infants. The New Zealand Medical Journal. 1971;74(470):24-5.
372. Elin RJ, Wolff SM, Finch CA. Effect of induced fever on serum iron and ferritin concentrations in man. Blood, The Journal of the American Society of Hematology. 1977;49(1):147-53.
373. Blrgegård G, Hålxgren R, Killander A, Strömberg A, Venge P, Wide L. Serum ferritin during infection: a longitudinal study. Scandinavian Journal of Haematology. 1978;21(4):333-40.
374. Konijn AM. Iron metabolism in inflammation. Bailliere's Clinical Haematology. 1994;7(4):829-49.
375. Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. New England Journal of Medicine. 1999;340(6):448-54.
376. Weiss G, Widner B, Zoller H, Schobersberger W, Fuchs D. Immune response and iron metabolism. British Journal of Anaesthesia. 1998;81:6-9.
377. Kotze M, Van Velden D, Van Rensburg S, Erasmus R. Pathogenic mechanisms underlying iron deficiency and iron overload: New insights for clinical application. Electronic Journal of the International Federation of Clinical Chemistry and Laboratory Medicine. 2009;20(2):108.
378. Fraser A, Scragg R, Metcalf P, McCullough S, Yeates N. Prevalence of Helicobacter pylori infection in different ethnic groups in New Zealand children and adults. Australian and New Zealand Journal of Medicine. 1996;26(5):646-51.
379. Chung B, Chaston T, Marks J, Srai SK, Sharp PA. Hepcidin decreases iron transporter expression in vivo in mouse duodenum and spleen and in vitro in THP-1 macrophages and intestinal Caco-2 cells. The Journal of Nutrition. 2009;139(8):1457-62.
380. Bayele HK, Srai SKS. Genetic variation in hepcidin expression and its implications for phenotypic differences in iron metabolism. Haematologica. 2009;94(9):1185.
381. Roe MA, Collings R, Dainty JR, Swinkels DW, Fairweather-Tait SJ. Plasma hepcidin concentrations significantly predict interindividual variation in iron absorption in healthy men. The American Journal of Clinical Nutrition. 2009;89(4):1088-91.

382. Kroot JJ, Hendriks JC, Laarakkers CM, Klaver SM, Kemna EH, Tjalsma H, et al. (Pre) analytical imprecision, between-subject variability, and daily variations in serum and urine hepcidin: implications for clinical studies. Analytical Biochemistry. 2009;389(2):124-9.
383. Young MF, Glahn RP, Ariza-Nieto M, Inglis J, Olbina G, Westerman M, et al. Serum hepcidin is significantly associated with iron absorption from food and supplemental sources in healthy young women. The American Journal of Clinical Nutrition. 2009;89(2):533-8.
384. Beutler E, Felitti VJ, Koziol JA, Ho NJ, Gelbart T. Penetrance of 845G→ A (C282Y) HFE hereditary haemochromatosis mutation in the USA. The Lancet. 2002;359(9302):211-8.
385. Adams PC, Reboussin DM, Barton JC, McLaren CE, Eckfeldt JH, McLaren GD, et al. Hemochromatosis and iron-overload screening in a racially diverse population. New England Journal of Medicine. 2005;352(17):1769-78.
386. Kang W, Barad A, Clark AG, Wang Y, Lin X, Gu Z, et al. Ethnic differences in iron status. Advances in Nutrition. 2021;12(5):1838-53.
387. Weatherall D. Fortnightly review: The thalassaemias. British Medical Journal. 1997;314(7095):1675.
388. Giardine B, Borg J, Viennas E, Pavlidis C, Moradkhani K, Joly P, et al. Updates of the HbVar database of human hemoglobin variants and thalassemia mutations. Nucleic Acids Research. 2014;42(D1):D1063-D9.
389. Trent R, Mickleson K, Yakas J, Hertzberg M. Population genetics of the globin genes in Polynesians. Hemoglobin. 1988;12(5-6):533-7.
390. Trent R, Mickleson K, Wilkinson T, Yakas J, Bluck R, Dixon M, et al. α Globin gene rearrangements in polynesians are not associated with malaria. American Journal of Hematology. 1985;18(4):431-3.
391. Yenchitsomanus P, Summers K, Board P, Bhatia K, Jones G, Johnston K, et al. Alpha-thalassemia in Papua New Guinea. Human Genetics. 1986;74(4):432-7.
392. Kirch PV. Peopling of the Pacific: A holistic anthropological perspective. Annual Review of Anthropology. 2010;39(1):131-48.
393. Ems T, St Lucia K, Huecker MR. Biochemistry, iron absorption. StatPearls StatPearls Publishing, Treasure Island; 2021.
394. Hurrell R, Egli I. Iron bioavailability and dietary reference values. The American Journal of Clinical Nutrition. 2010;91(5):1461S-7S.
395. Bezwoda W, Bothwell, TH, Charlton, RW, Torrance, JD, Macphail, AP, Derman, DP, Mayet F. Relative dietary importance of haem and non-haem iron. South African Medical Journal. 1983;64(14):552-6.
396. Carpenter CE, Mahoney AW. Contributions of heme and nonheme iron to human nutrition. Critical Reviews in Food Science & Nutrition. 1992;31(4):333-67.
397. Cook JD, Monsen ER. Vitamin C, the common cold, and iron absorption. The American Journal of Clinical Nutrition. 1977;30(2):235-41.
398. Moustarah F, Mohiuddin SS. Dietary iron. StatPearls: StatPearls Publishing, Treasure Island; 2022.
399. Allen L, Ahluwalia N. Improving iron status through diet: The application of knowledge concerning dietary iron bioavailability in human populations: Opportunities for Micronutrient Interverntions (OMNI) Project; 1997.
400. Cook JD, Watson SS, Simpson KM, Lipschitz DA, Skikne BS. The effect of high ascorbic acid supplementation on body iron stores. 1984. p. 721-6.
401. Monsen E, Labbe R, Lee W, Finch C, Momcilovic B. Iron balance in healthy menstruating women: effect of diet and ascorbate supplementation. Dubrovnic, Yugoslavia: Institute for Medical Research and Occupational Health, University of Zagreb; 1991.
402. Hunt JR, Gallagher SK, Johnson L. Effect of ascorbic acid on apparent iron absorption by women with low iron stores. The American Journal of Clinical Nutrition. 1994;59(6):1381-5.

403. Cook JD, Reddy MB. Effect of ascorbic acid intake on nonheme-iron absorption from a complete diet. The American Journal of Clinical Nutrition. 2001;73(1):93-8.
404. Taylor PG, Martinez-Torres C, Romano EL, Layrisse M. The effect of cysteine-containing peptides released during meat digestion on iron absorption in humans. The American Journal of Clinical Nutrition. 1986;43(1):68-71.
405. Lynch SR, Hurrell RF, Dassenko SA, Cook JD. The effect of dietary proteins on iron bioavailability in man. Mineral Absorption in the Monogastric GI Tract. 249. Boston, MA: Springer; 1989. p. 117-32.
406. Björn-Rasmussen E, Hallberg L. Effect of animal proteins on the absorption of food iron in man. Annals of Nutrition and Metabolism. 1979;23(3):192-202.
407. Armah SM, Carriquiry A, Sullivan D, Cook JD, Reddy MB. A complete diet-based algorithm for predicting nonheme iron absorption in adults. The Journal of Nutrition. 2013;143(7):1136-40.
408. Reddy MB, Hurrell RF, Cook JD. Meat consumption in a varied diet marginally influences nonheme iron absorption in normal individuals. The Journal of Nutrition. 2006;136(3):576-81.
409. Björn-Rasmussen E. Iron absorption from wheat bread. Annals of Nutrition and Metabolism. 1974;16(2):101-10.
410. Hallberg L. Bioavailability of dietary iron in man. Annual Review of Nutrition. 1981;1(1):123-47.
411. Brune M, Rossander L, Hallberg L. Iron absorption and phenolic compounds: importance of different phenolic structures. European Journal of Clinical Nutrition. 1989;43(8):547-57.
412. Disler P, Lynch S, Charlton R, Torrance J, Bothwell T, Walker R, et al. The effect of tea on iron absorption. Gut. 1975;16(3):193-200.
413. Rossander L, Hallberg L, Björn-Rasmussen E. Absorption of iron from breakfast meals. The American Journal of Clinical Nutrition. 1979;32(12):2484-9.
414. El Gharras H. Polyphenols: food sources, properties and applications–a review. International Journal of Food Science & Technology. 2009;44(12):2512-8.
415. Lönnerdal B. Calcium and iron absorption-mechanisms and public health relevance. International Journal for Vitamin and Nutrition Research. 2010;80(45):293-9.
416. Ejezie F, Nwagha U, Ikekpeazu E, Ozoemena O, Onwusi E. Assessment of iron content of breast milk in preterm and term mothers in enugu urban. Annals of Medical and Health Sciences Research. 2011;1(1):85-90.
417. Domellöf M. Iron requirements, absorption and metabolism in infancy and childhood. Current Opinion in Clinical Nutrition & Metabolic Care. 2007;10(3):329-35.
418. Legrand D, Elass E, Carpentier M, Mazurier J. Lactoferrin. Cellular and Molecular Life Sciences. 2005;62(22):2549-59.
419. Arnold R, Brewer M, Gauthier J. Bactericidal activity of human lactoferrin: sensitivity of a variety of microorganisms. Infection and Immunity. 1980;28(3):893-8.
420. Lönnerdal B. Iron in human milk and cow's milk—effects of binding ligands on bioavailability. Iron Metabolism in Infants: CRC Press; 2020. p. 87-107.
421. Ashida K, Sasaki H, Suzuki YA, Lönnerdal B. Cellular internalization of lactoferrin in intestinal epithelial cells. Biometals. 2004;17(3):311-5.
422. Lönnerdal B, Bryant A. Absorption of iron from recombinant human lactoferrin in young US women. The American Journal of Clinical Nutrition. 2006;83(2):305-9.
423. Hernell O, Lönnerdal B. Iron status of infants fed low-iron formula: no effect of added bovine lactoferrin or nucleotides. The American Journal of Clinical Nutrition. 2002;76(4):858-64.
424. King Jr JC, Cummings GE, Guo N, Trivedi L, Readmond BX, Keane V, et al. A double-blind, placebo-controlled, pilot study of bovine lactoferrin supplementation in bottle-fed infants. Journal of Pediatric Gastroenterology and Nutrition. 2007;44(2):245-51.
425. Domellöf M, Braegger C, Campoy C, Colomb V, Decsi T, Fewtrell M, et al. Iron requirements of infants and toddlers. Journal of pediatric gastroenterology and nutrition. 2014;58(1):119-29.

426. Zealand FSAN. Australia New Zealand Food Standards Code—Standard 2.9.2—Food for Infants [12 August 2022]. Available from: https://www.legislation.gov.au/Details/F2017C00334.
427. Aggett PJ, Agostoni C, Axelsson I, Bresson J-L, Goulet O, Hernell O, et al. Iron metabolism and requirements in early childhood: do we know enough?: a commentary by the ESPGHAN Committee on Nutrition. Journal of Pediatric Gastroenterology and Nutrition. 2002;34(4):337-45.
428. Wall CR, Brunt DR, Grant CC. Ethnic variance in iron status: is it related to dietary intake? Public Health Nutrition. 2009;12(9):1413-21.
429. Akel R, Frankish J, Powles C, Tyler K, Watt J, Weston H, et al. Anaemia in Maori and European infants and children on admission to hospital. A co-operative survey from six New Zealand hospitals. The New Zealand Medical Journal. 1963;62:29-33.
430. Anyon C. Normal haemoglobin values in urban Polynesian infants: the possible deleterious influence of artificial feeding. The New Zealand Medical Journal. 1976;84(578):474-6.
431. Anyon C, Desmond F. Normal haematological values in New Zealand Eruopean infants. The New Zealand Medical Journal. 1974;80(527):383.
432. Crampton P, Farrell A, Tuohy P. Iron deficiency anaemia in infants. The New Zealand Medical Journal. 1994;107(972):60-1.
433. Dickson N, Morison I. Iron deficiency in infants of Cambodian refugees. The New Zealand Medical Journal. 1992;105(929):83-4.
434. Emery D, Barry D. Comparison of Maori and non-Maori maternal and fetal iron parameters. The New Zealand Medical Journal. 2004;117(1195).
435. Morton SB, Saraf R, Bandara DK, Bartholomew K, Gilchrist CA, Atatoa Carr P, et al. Maternal and perinatal predictors of newborn iron status. The New Zealand Medical Journal. 2014;127(1402):62-77.
436. Moyes C, O'Hagan L, Armstrong C. Anaemia in Maori infants--a persisting problem. The New Zealand Medical Journal. 1990;103(883):53-.
437. Poppe M. Iron deficient children. The New Zealand Medical Journal. 1993;106(963):392-.
438. Rive S, Blacklock H, Hawkins R, Mitchell E. Anaemia in children aged 6 to 23 months attending the paediatric acute assessment unit in south Auckland. The New Zealand Medical Journal. 1996;109(1016):58-.
439. Szymlek-Gay EA, Ferguson EL, Heath A-LM, Gray AR, Gibson RS. Food-based strategies improve iron status in toddlers: a randomized controlled trial. The American Journal of Clinical Nutrition. 2009;90(6):1541-51.
440. Thom R, Parnell W, Broadbent R, Heath AL. Predicting iron status in low birthweight infants. Journal of Paediatrics and Child Health. 2003;39(3):173-6.
441. Wilson C, Grant CC, Wall CR. Iron deficiency anaemia and adverse dietary habits in hospitalised children. The New Zealand Medical Journal. 1999;112(1089):203-6.
442. Thurnham DI, Mburu AS, Mwaniki DL, De Wagt A. Micronutrients in childhood and the influence of subclinical inflammation. Proceedings of the Nutrition Society. 2005;64(4):502-9.

# Chapter 3 | Upoko Tuatoru

## Methods

*This chapter describes the methods and study procedures used to answer the research question*

The study design and data collection are described in the following section. Further details of the methods for the overall FFNZ study are reported elsewhere (1). First Foods NZ was an observational cross-sectional study which was registered with the Australian New Zealand Clinical Trials Registry, ACTRN12620000459921. Ethical approval was granted by the Health and Disability Ethics Committees New Zealand (19/STH/151) and was funded in May 2019 by the Health Research Council (HRC) of New Zealand (grant 19/172). Data collection was conducted between July 2020 and February 2022 by an inter-disciplinary team consisting of registered dietitians, a speech language therapist, and human nutritionists.

**First Foods NZ Study Design**

The wider FFNZ study aimed to examine the food intake and health of 625 infants aged 7.0-<10 months of age, from a range of sociodemographic areas and ethnicities. The main wider study aim was to compare infants who use baby food pouches with those who do not, alongside those who use BLW and those using TSF. At the same time, data on nutrient intake, nutrition status, iron status, eating behaviours, anthropometry, dental health, breastmilk intake, and feeding and swallowing difficulties was collected for this age group in general.

Recruiting a representative sample was not deemed feasible, as traditional methods such as electoral roll and door knocking would be unlikely to yield sufficient infants in the narrow age band required by this study. Therefore, we aimed to target a broad range of socio-economic areas and ethnicities when recruiting, and using research team members who are experienced working with a wide range of groups.

The age range was chosen as it is close to the time where infants are introduced to CF, with the narrow age range allowing for the rapidly changing diet in infancy. The sample size was based on the wider study aim of comparing BMI z-score and plasma ferritin concentration in infants following baby-led weaning and traditional spoon-feeding. This was chosen as there is a lack of data on these measures in infants who use baby food pouches.

The present study specifically investigates Māori and Pasifika infants, alongside all other infants. Infant complementary feeding practices (method of feeding, such as BLW and TSF, pouch use, timing of introduction of complementary foods, types of first foods offered, prevalence of traditional cultural foods), nutrient intake and nutrient density of complementary food, and iron status.

**Study Participants**

A total of 625 infants aged 7.0 - 10.0 months were recruited from two urban centres in NZ: Tāmaki-Makaurau (Auckland) and Ōtepoti (Dunedin). Participants were excluded if the adult respondent was unable to speak English or if the infant had been part of a nutrition intervention study. Participants were recruited through advertisements and word-of-mouth, whilst avoiding special interest groups, such as baby-led weaning groups. To ensure ethnic diversity recruitment was targeted in suburbs with higher densities of Māori and Pasifika whānau (families). An information sheet was given to all participants and written informed consent was obtained.

Participants were grouped by total ethnicity, with any participants who did not identify as either Māori or Pasifika categorised into a single 'others' groups. This allows the focus of this study to remain on Māori and Pasifika infants. Ethnicities in the 'others' category included New Zealand European, Southeast Asian, East Asian, South Asian, European, and MELAA (Middle Eastern, Latin American, and African).

**Demographic data**

Demographic data was collected via questionnaire which encompassed age, ethnicity, sex, household deprivation, (NZ Deprivation Index 2018 (2)), caregiver education, work status, parity, childcare use, the number of children and adults living in the household, and whether the infant was a premature or full-term at birth. Mesh block data was used to determine the level of deprivation based on the NZ Deprivation Index. Infant ethnicity was self-determined by the caregiver. Respondents were asked "what ethnic group does your child belong to?". Checkbox answer options mirrored that of the 2018 New Zealand census, and participants were able to select from the following list: "NZ European", "Māori", "Samoan", "Cook Island Māori", "Tongan", "Niuean", "Chinese", "Indian", and "other, e.g., Dutch, Japanese, Tokelauan". If the participant selected the "other" option, they were prompted to enter free text to describe their ethnicity.

**Complementary feeding practices data**

Feeding practices were self-reported by the caregiver via questionnaire, with a researcher present to clarify any questions. Caregivers were asked about the timing of the introduction of CFs, pouch use, and method of feeding (TSF, BLW, or partial BLW) at the time of CF introduction, at six months, and currently. Infants were classified as TSF if "spoon-fed by an adult" or "mostly spoon-fed by an adult, some baby feeding themselves" was selected. Infants are classified as partial BLW if "about half spoon-fed by an adult and half baby feeding themselves" was selected. Infants were classified as

BLW if “baby feeding themselves” and “mostly baby feeding themselves, some spoon feeding by an adult” was selected.

Participants were asked about the frequency of PF, use of CIF pouches versus re-usable home-filled pouches, and the prevalence of infants feeding directly from the pouch nozzle versus from a spoon. Infants were designated as either ‘frequent pouch users’ (5 or more pouches per week), ‘sometimes’ (1-4 pouches per week), ‘infrequently’ (less than 4 pouches per week), or ‘never’. The way in which infants fed from pouches was categorised as “always via the nozzle”, “mostly via the nozzle”, “both via the nozzle and spoon”, “mostly via spoon”, and “always via spoon”. The prevalence of infants feeding directly from the pouch nozzle was stratified by frequency of use.

Data on the first food offered, texture of the first food, and traditional foods and practices were collected via the same questionnaire completed by the caregiver. Participants were invited to expand upon their answer with free text. Participants were also asked if they offered their infant baby rice, reasons for not offering (if applicable), and frequency of baby rice consumption (if applicable).

**Dietary data**

Dietary data was obtained from two non-consecutive multiple-pass 24-hour recalls collected by trained interviewers at the first and second appointments. The recalls were collected on different days of the week, which captured between-day variation in dietary intake. The 24-hour recalls captured everything the infant ate and drank from midnight to midnight.

Caregivers were asked to take photographs of the eating surface, including the offered food and beverages, both before and after each meal and snack the day before the 24-hour recall was conducted. Text message reminders were sent the day before as a reminder to do this. The participants were able to use either their own smartphone or a study camera to capture these photographs, with the quality of the photos not being important as they were intended to be used as prompts to help the caregiver remember what the infant ate.

The first pass of the 24-hour recall involved obtaining a “quick list” of all the things the infant ate and drank the previous day. Amounts and brands were not required at this stage as this was to help serve as a prompt for the second pass. Various verbal prompts were used, starting with asking the adult respondent the following:

*"So, we're going to be thinking about what[baby's name]had yesterday–[insert day that was yesterday]. Let's start right at the beginning–from midnight then we'll go through the morning, afternoon, and evening. What was the first thing [baby's name] ate or drank?"*

Other verbal prompts used during the first pass were:

- *"Sometimes people forget to tell us about drinks and snacks when we do this list."*
- *"How much water did [baby's name] drink yesterday?"*
- *"Did[baby's name]have any[more]breastmilk or formula yesterday?"*
- *"Did [baby's name] have any other drinks like milk or juice yesterday?"*
- *"How about any other snacks, like muesli bars, crackers, sweets, or desserts?"*
- *"Were there any other meals or snacks that [baby's name] ate yesterday that someone else gave [him/her]?"*
- *"Do you have any information about what[baby's name] ate or drank yesterday while in their care?*

Stage 2, or the second pass of the 24-hour recall involved recording a detailed list of what the infant ate and drank, quantities, time, method of feeding if a pouch (e.g., nozzle or spoon, self-fed or adult-fed), and two-tier location of eating (specific, e.g., highchair, and broad, e.g., home). The amount of food both offered and consumed was also recorded. If the infant consumed food or drink while in the care of another adult or at childcare, a "Foods Fed by Other Adults" form was completed.

Stage 3, or the third pass of the 24-hour recall reviewed and probed for forgotten foods. The adult respondent was asked about time spent eating on each occasion, duration of formula or breast feeds, forgotten foods/fluids, and home-filled ouch use. The complete detailed list was carefully and slowly reviewed so that the participant had the opportunity to remember each occasion.

The completed 24-hour recall was then quality checked by another researcher, with any missing detail or inadequate information followed up on with the participant promptly.

**Biochemical data for iron status**

Local anaesthetic (Ametop Gel, Smith & Nephew, Canada) was applied to both of the infants' arms inside the elbow, and an occlusive dressing was applied. The dressing was removed and excess gel wiped off with a tissue 30-45 minutes after application, with the blood sample drawn no more than

4 – 6 hours after the Ametop was applied. Blood samples were collected by venepuncture via an EDTA vacutainer. Full blood count was measured on the day of collection by Southern Community Labs (SCL), Dunedin, and Labtests, Auckland on a Sysmex-XN20 Module® automated analyser (Sysmex, Kobe, Japan). The ferritin assay was conducted on a E602-Cobas 8000 analyser (Roche Diagnostics, USA), and was measured within seven days of collection. Remaining plasma samples were stored at -70° Celsius until consequent analysis of sTfR, C-Reactive Protein (CRP), and $\alpha_1$-acid glycoprotein (AGP) on a Hitachi Cobas C311 analyser (Roche Diagnostics, Mannheim, Germany, and Hitachi High-Technologies Corp, Tokyo, Japan) machine at the Department of Human Nutrition (University of Otago, Dunedin, NZ). Abnormal results were reviewed by the study paediatrician and a letter was sent to both the caregiver and their general practitioner as specified by the caregiver on the consent form. If no abnormal results were detected a letter was sent to the caregiver providing the infant's haemoglobin (g/L), plasma ferritin (µg/L) and haematocrit. The letter included a brief lay-language explanation of each of these indices. Ferritin was adjusted for inflammation using the BRINDA method (3), and sTfR was adjusted to be equivalent with the Flowers assay as follows (4): 1.5 x Roche sTfR + 0.35 mg/L. Body iron was calculated as follows: -[*log*10 (sTfR x 1000/ferritin) – 2.8229]/0.1207.

Cut-offs used to determine iron status are described in table 1. Infants who had low Hb in the absence of iron deficiency were included in the "iron sufficient" group.

**Table 1.** Iron status categories and haematological cut-offs

| Iron status category | Cut-off value |
|---|---|
| Iron sufficient | Plasma ferritin ≥15 µg/L[a], body iron ≥0 mg/kg |
| Iron depleted | Plasma ferritin <15 µg/L[a], body iron ≥0 mg/kg |
| Early functional iron deficiency | Body iron <0 mg/kg[b], haemoglobin ≥110 g/L[c] |
| Iron deficiency anaemia | Body iron <0 mg/kg[b], haemoglobin <110 g/L[c] |

[a] Southern Community Laboratories Ltd. (2012)
[b] Cook et al. (2003)
[c] World Health Organization (2001)

**Data sovereignty**

Māori and Pasifika data sovereignty was considered in the design of this study. Equity in health outcomes is a core component in quality healthcare, and in research the health data for Māori and Pasifika are collected then disseminated. Confidentiality must be navigated, and it is important to be Tiriti-lead (The Treaty of Waitangi). Therefore, both a Māori and Pasifika cultural

advisor were part of the team designing the wider First Foods NZ study and oversaw the protocols for data collection and management.

**Statistical analyses**

For dietary data, pouch use, BLW, TSF were estimated for the entire sample with 95% confidence intervals. The data is presented as numbers and percentages. For nutrient intake and nutrient density data, usual intake was calculated from the 24-hour recalls using the multiple source method for estimating usual dietary intake of individuals (5). Dietary data was analysed using FoodWorks (version 10, Xyris Software), using the New Zealand Food Composition database FOODfiles 2018 Version 01. For commercial foods, recipes were created in FoodWorks using the ingredient list on the packet, and then modified to match the information on the nutrient information panel. This ensured that nutrients that did not appear on the nutrient information panel were captured in the analysis. Breast and formula milk were excluded from analysis for the purposes of this study. Quantile regression was used to determine nutrient intake, allowing adjustment for infant age. Nutrient density is reported as concentration of nutrient per 418 kJ (100 kcal) of energy. Ethnicity data is reported as total response ethnicity.

The biochemical indices for iron status were described using mean, geometric mean, and median. Potential mediators were identified by assessing statistical significance in differences between ethnicities in early feeding practices. Linear regression analyses were performed to determine the suitability of using mediation analysis to explain differences in body iron between ethnic groups. This was done by first testing the relationship between ethnic group and body iron (mg/kg body weight), then testing the relationship between ethnicity and each mediating factor (introduction of complementary food at <5 months of age, consumption of red meat and / or iron-fortified baby rice at six months of age, and use of baby food pouches currently), and finally testing the relationship between each mediating factor and body iron. Where all relationships were significant ($p$<0.05) for an ethnic group, mediation analysis was performed.

All data was analysed using Stata V.17 (StataCorp) statistical software.

References

1. Taylor RW, Conlon CA, Beck KL, von Hurst PR, Te Morenga LA, Daniels L, et al. Nutritional Implications of Baby-Led Weaning and Baby Food Pouches as Novel Methods of Infant Feeding: Protocol for an Observational Study. JMIR Research Protocols. 2021;10(4):e29048.
2. Atkinson J, Salmond C, Crampton P. NZDep2018 Index of Deprivation, Interim Research Report. Wellington: University of Otago; 2019.
3. Suchdev PS, Namaste SM, Aaron GJ, Raiten DJ, Brown KH, Flores-Ayala R, et al. Overview of the biomarkers reflecting inflammation and nutritional determinants of anemia (BRINDA) project. Advances in Nutrition. 2016;7(2):349-56.
4. Cogswell ME, Looker AC, Pfeiffer CM, Cook JD, Lacher DA, Beard JL, et al. Assessment of iron deficiency in US preschool children and nonpregnant females of childbearing age: National Health and Nutrition Examination Survey 2003–2006. The American Journal of Clinical Nutrition. 2009;89(5):1334-42.
5. Harttig U, Haubrock J, Knüppel S, Boeing H. The MSM program: web-based statistics package for estimating usual dietary intake using the Multiple Source Method. European Journal of Clinical Nutrition. 2011;65(1):S87-S91.

# Chapter 4 | Upoko Tuawhā

## Complementary feeding practices of Māori, Pasifika, and other infants in Aotearoa New Zealand

*This chapter reports the findings of the First Foods New Zealand study pertaining to infant feeding practices by ethnicity.*

*This report is presented in manuscript format and will be submitted to a peer-reviewed journal for publication in the future. Online statement of contribution can be reviewed in Appendix H.*

Abstract

**Background:** Timing of complementary food (CF) introduction and methods of feeding (baby led weaning (BLW), traditional spoon-feeding (TSF), and pouch use) have the potential to impact infant growth, development, and health outcomes. Māori and Pasifika infants in Aotearoa New Zealand (NZ) carry a disproportionate burden of nutrition-related disease, but little is known about current complementary feeding practices in this group.

**Aim:** The aim of this study was to describe complementary feeding methods, first food characteristics, and the timing of CF introduction amongst Māori, Pasifika, and 'other' infants living in NZ.

**Methods:** Complementary feeding practices of infants aged 7.0–10.0 months living in Auckland and Dunedin were determined by questionnaire completed by the main caregiver. Ethnicity was reported as total response, with those who did not select either Māori or Pacific categorised into a single 'other' group. Frequency of pouch use and BLW were estimated with 95% confidence intervals.

**Results:** Of 625 infants, over half of all ethnicities introduced CF at around six months of age (56.5% of Māori, 62.2% of Pasifika, and 80.9% of 'others'). BLW prevalence increased from 11.5% of Māori, 3.7% of Pasifika, and 12.4% of 'other' infants at the time of introducing CF to 29.2% of Māori, 17.1% of Pasifika, and 27.3% of 'others' currently. Baby food pouches were used at least once by 89.3% of Māori, 85.4% of Pasifika, and 75.6% of 'other' infants. Of those who always or frequently were fed pouches, 27.1% of Māori, 25% of Pasifika, and 12% of 'other' infants always or mostly sucked directly from the nozzle. Vegetables and 'pureed' were the most common first food and texture offered respectively for all ethnic groups. At six months red meat was consumed by 54.6% of Māori infants, 63.4% of Pasifika infants, and 61.8% of 'other' infants. Approximately half had iron-fortified baby rice (Māori 57.3%, Pasifika 56.1%, 'other' 48.7%). Age-inappropriate drinks were currently given to 17.6% of Māori, 20.7% of Pasifika, and 3.8% of 'other' infants. When asked about traditional cultural foods 9.1% of Māori and 20.7% of Pasifika respondents reported offering these to their infants.

**Conclusion:** These findings provide insight into complementary feeding practices that potentially impact the health outcomes of our most vulnerable infants, in particular the timing of CF introduction, BLW, age-inappropriate drinks, and pouch use. Māori- and Pasifika-centred approaches in supporting whānau with infant feeding needs to implemented.

Introduction

Infant complementary feeding practices impact healthy growth and development in both early life and into adulthood. The World Health Organisation (WHO) recommend introducing complementary food (CF) at approximately six months of age (1). The Manatū Hauora (Ministry of Health New Zealand, (MoH)) recommend starting with half to two teaspoons of a thin smooth puree via the spoon after breast or formula feeding (2). It is then recommended that a variety of foods and textures are introduced, gradually progressing from a thin to thick puree, to mashed to chopped with the infant feeding themselves more as they grow older (2).

Complementary feeding practices in Aotearoa New Zealand (NZ) have evolved over the past two centuries. In both pre-colonial NZ and the neighbouring Pacific Islands, infants shared whānau (family) food, which was texture modified and warmed through pre-mastication. Vegetables were commonly offered as first foods for Māori infants (3), with starchy root crops, breadfruit, green banana, and coconut provided in the Pacific Islands (4). The infant's developmental cues such as sitting and grasping would inform the timing of introducing CF, and breastfeeding and wet nursing were common practices. Post-colonisation, many practices have changed. Infants are more commonly introduced to CF at an early age, commercial infant foods (CIF) are widely available, and for some Māori traditional community and social structures that enabled care of the infant by the wider whānau are not accessible (5).

In recent years there has been an explosion of baby food pouches onto the market. Baby food pouches are made of soft squeezable plastic, with nozzles attached that the infant can suck pureed food from directly. In 2015 in the United States, 56% of CIFs were packaged in pouches, and 54% in the United Kingdom in 2019 (6). A 2019 NZ study found that over half of all CIF (*n*= 103, 52.3%) were packaged into pouches (7). In both NZ and internationally, pouches have more sugar and lower levels of iron compared with equivalent CIFs not packaged into pouches (8-12). Alongside pouch use, baby-led weaning (BLW) appears to be increasing, with scarce research in NZ indicating that it appears to be a primarily Pakeha (European) phenomenon with a possibly increasing uptake by Māori whānau (13, 14).

There is very limited data describing the complementary feeding practices of infants in NZ including use of BLW and pouches, timing of introduction to CF, texture and types of first foods offered, and traditional cultural foods and practices. With a disproportionately high burden of communicable and non-communicable disease experienced by Māori and Pasifika infants and young children,

particularly respiratory illnesses, iron-deficiency, and dental caries (15, 16), there is a growing need to assess and better understand complementary feeding practices. This will help guide relevant advice to whānau that is seen as relevant and is consistent with cultural practices. The supports the principles of the Treaty of Waitangi that all New Zealanders are entitled to equal health outcomes and access to healthful traditional cultural food and practices. Therefore, this paper will describe complementary feeding methods, first foods characteristics, and the timing of CF introduction amongst Māori, Pasifika, and non-Māori/Pasifika infants living in NZ.

## Methods

Study design and data collection methods are described in brief, with further details reported elsewhere (17). This study was registered with the Australian New Zealand Clinical Trials Registry, ACTRN12620000459921. Ethical approval was granted by the Health and Disability Ethics Committee New Zealand (19/STH/151) and funding obtained from the Health Research Council (HRC) of New Zealand (grant 19/172). Data collection ran from July 2020 to February 2022. Data collection was paused during government-mandated COVID-19-related lockdowns, which occurred for two and a half weeks in August 2020, one week in February 2021, and four and a half months in August 2021 in Auckland, and for three weeks in August 2021 in Dunedin. All appropriate hygiene measures were taken to minimise risk during data collection.

### Study Design

First Foods New Zealand (FFNZ) is an observational cross-sectional study of 625 infants aged 7.0–10.0 months in NZ. This analysis aims to evaluate and compare infant feeding practices of Māori, Pasifika, and non-Māori/Pasifika infants. Infant complementary feeding practices encompass method of feeding, such as BLW and traditional spoon-feeding (TSF), pouch use, timing of introduction of CFs, and types of first foods offered. The sample size was based on the wider study aim of comparing body-mass index (BMI) z-score and plasma ferritin concentration in infants following BLW and TSF. This sample size will also allow the estimation of prevalence of frequent pouch feeding (PF) to a 95% ± 4% precision level.

### Study Participants

Infants were recruited from two urban centres in NZ: Tāmaki-Makaurau (Auckland) and Ōtepoti (Dunedin). Participants were excluded if the caregiver was unable to speak English or if the infant had been part of a nutrition intervention study. Participants were recruited from a range of ethnic groups and deprivation statuses using advertisements and word-of-mouth. When recruiting in social

media groups, special interest groups, such as BLW or infants with allergies, were avoided to minimise sampling bias. To ensure ethnic diversity recruitment was targeted in suburbs with higher proportions of Māori and Pasifika whānau. An English language information sheet was given to all participants and written informed consent was obtained from the primary caregiver.

**Data collection**

**Demographic Data**

Demographic data was collected via questionnaire which encompassed ethnicity, household deprivation, (NZ Deprivation Index 2018 (18)), caregiver education, work status, age, parity, if the infant is in childcare, the number of children and adults living in the household, and infant age, sex, and gestational age at birth. Mesh block data was used to determine the level of deprivation based on the NZ Deprivation Index. Infant ethnicity was self-determined by the caregiver. Respondents were asked "what ethnic group does your child belong to?". Checkbox answer options were "Māori", "Pacific", "New Zealand European", or "other". If the participant selected the "other" option, they were prompted to enter free text to describe their ethnicity. For the purposes of this manuscript, participants who did not select Māori or Pacific, were categorised as 'others'.

**Feeding Practices Data**

Feeding practices were self-reported by the caregiver via questionnaire, with a researcher present to clarify any questions. Caregivers were asked about the timing of the introduction of CFs, pouch use, and method of feeding (TSF, BLW, or partial BLW) at the time of CF introduction, at six months, and currently. Infants were classified as TSF if "spoon-fed by an adult" or "mostly spoon-fed by an adult, some baby feeding themselves" was selected. Infants are classified as partial BLW if "about half spoon-fed by an adult and half baby feeding themselves" was selected. Infants were classified as BLW if "baby feeding themselves" and "mostly baby feeding themselves, some spoon feeding by an adult" was selected.

Participants were asked about the frequency of PF, use of CIF pouches versus re-usable home-filled pouches, and the prevalence of infants feeding directly from the pouch nozzle versus from a spoon. Infants were designated as either 'frequent pouch users' (5 or more pouches per week), 'sometimes' (1–4 pouches per week), 'infrequently' (fewer than one pouch per week), or 'never'. The way in which infants fed from pouches was categorised as "always via the nozzle", "mostly via the nozzle", "both via the nozzle and spoon", "mostly via spoon", and "always via spoon". The prevalence of infants feeding directly from the pouch nozzle was stratified by how frequently they had pouches.

Data on the first food offered, texture of the first food, and traditional foods and practices were collected via the same questionnaire completed by the caregiver. Participants were invited to expand upon their answer with free text. Participants were also asked if they offered their infant baby rice, reasons for not offering (if applicable), and frequency of fortified baby rice consumption (if applicable).

**Statistical Analysis**

Ethnicity data is reported as total response ethnicity as many NZ infants belong to more than one ethnic group. Pouch use, BLW, TSF were estimated for the entire sample. The data is presented as numbers and percentages. Data was analysed using Stata statistical package (StataCorp).

Results

A total of 1424 infants were assessed for eligibility, and 625 eligible infants were enrolled in the study, all of whom provided written consent and completed the questionnaire. Data were analysed for all 625 infants. Within the cohort 131 infants were identified by their caregiver as Māori, and 82 as Pasifika. The remaining infants are allocated into a single 'others' group as the primary focus of this manuscript are Māori and Pasifika infants. The mean (SD) infant age was 8.4 months for Māori, 8.5 months for Pasifika, and 8.4 months for 'other' infants. Demographic characteristics are presented in table 1.

**Table 1.** Demographic characteristics (n=625)

| | Total response ethnicity[e] | | |
|---|---|---|---|
| | Māori | Pasifika | Others |
| n | 131 | 82 | 450 |
| Infant age (mo), mean (SD) | 8.4 (0.8) | 8.5 (0.9) | 8.4 (0.8) |
| Infant sex, n (%) | | | |
| Female | 57 (43.5) | 35 (42.7) | 215 (47.8) |
| Total response infant ethnicity[a], n (%) | | | |
| Māori | 131 (100) | 38 (46.3) | 0 |
| Pacific Island | 38 (29.0) | 82 (100) | 0 |
| New Zealand European | 99 (75.6) | 45 (54.9) | 372 (82.7) |
| Southeast Asian | 0 | 0 | 28 (6.2) |
| East Asian | 2 (1.5) | 3 (3.7) | 40 (8.9) |
| South Asian | 4 (3.1) | 3 (3.7) | 25 (5.6) |
| Other European | 6 (4.6) | 7 (8.5) | 49 (10.9) |
| Others | 2 (1.5) | 0 | 18 (4.0) |
| Term infant born, n (%) | | | |
| Pre-term (<37 weeks) | 13 (9.9) | 8 (9.8) | 29 (6.4) |
| Term (≥37 weeks) | 118 (91.1) | 73 (89.0) | 421 (93.6) |
| Respondent age (years), mean (SD) | 30.1 (5.5) | 30.3 (6.0) | 33.5 (4.3) |
| Highest level of education of caregiver, n (%) | | | |
| School | 34 (26.0) | 23 (28.1) | 48 (10.7) |
| Polytech or similar | 48 (36.6) | 21 (25.6) | 69 (15.3) |
| University | 48 (36.6) | 37 (45.1) | 333 (74.0) |
| Caregiver, n (%) | | | |
| Mother | 130 (99.2) | 80 (97.6) | 445 (98.9) |
| Maternal parity, n (%) | | | |
| Primiparous | 54 (41.2) | 39 (46.3) | 229 (50.9) |
| Respondent employment status, n (%) | | | |
| Employed full time | 17 (13.0) | 6 (7.3) | 52 (11.6) |
| Employed part time | 32 (24.4) | 14 (17.1) | 96 (21.3) |
| Other[b] | 82 (62.6) | 62 (75.6) | 302 (67.1) |
| Number of children living in household, n (%) | | | |
| One | 47 (35.9) | 28 (34.2) | 223 (49.6) |
| Two | 43 (32.8) | 28 (34.2) | 142 (31.6) |

| | Total response ethnicity[e] | | |
|---|---|---|---|
| | Māori | Pasifika | Others |
| Three | 21 (16.0) | 11 (13.4) | 67 (14.9) |
| Four or more | 20 (15.3) | 15 (18.3) | 17 (3.8) |
| Number of adults living in household, n (%) | | | |
| One | 11 (8.4) | 4 (4.9) | 13 (2.9) |
| Two | 92 (70.2) | 47 (57.3) | 399 (88.7) |
| Three | 14 (10.7) | 14 (17.1) | 19 (4.2) |
| Four or more | 14 (10.7) | 17 (20.7) | 19 (4.2) |
| Childcare used[c], n (%) | 27 (20.6) | 16 (19.5) | 76 (16.9) |
| Area-level deprivation[d], n (%) | | | |
| 1–3 (Low) | 27 (20.6) | 14 (17.1) | 144 (32.0) |
| 4–7 | 52 (39.7) | 35 (42.7) | 214 (47.6) |
| 8–10 (High) | 52 (39.7) | 33 (40.2) | 92 (20.4) |

[a] Gestational age - data missing for one Pasifika participant; level of education - data missing for one Māori and one Pasifika participant; no. of children living in household – data missing for one non-Māori/Pasifika participant.

[b] "Other" value includes not employed, paid and un-paid parental leave.

[c] "Childcare" refers to formal early childhood education or home-based care, but not a nanny or childcare provided by an extended family member/friend.

[d] Deprivation level defined using the New Zealand Deprivation (NZDep) indices of 2018 (18). NZDep index is area based, uses NZ census variables, and calculated for mesh blocks (geographic units containing ~100 people). "Low" refers to levels 1–3, "Medium" refers to levels 4–7, "High" refers to levels 8–10.

[e] Ethnicity is self-identified by the caregiver, and is greater than 100 due to many participants identifying as more than one ethnicity

Over half of all groups introduced CF at around six months of age, with only a very small proportion initiating complementary feeding before four months. The prevalence of timely and non-timely introduction to CF is shown in table 2.

**Table 2.** Timely and non-timely introduction to complementary food

| | Māori | Pasifika | Others |
|---|---|---|---|
| Age complementary foods introduced, mean (SD) months | 4.9 (1.1) | 4.9 (1.1) | 5.3 (0.8) |
| < 4 months, n (%) | 9 (6.9) | 8 (9.8) | 3 (<1) |
| 4.0–<5 months, n (%) | 44 (33.6) | 20 (24.4) | 75 (16.7) |
| **Total early introduction to CF[a]**, n (%) | 53 (40.5) | 28 (34.2) | 78 (17.3) |
| 5.0 – <7 months[a], n (%) | 74 (56.5) | 51 (62.2) | 364 (80.9) |
| ≥ 7 months, n (%) | 4 (3.1) | 3 (3.7) | 8 (1.8) |

[a]Timely introduction to CF as defined by the Healthy Eating Guidelines for New Zealand Babies and Toddlers (2)

Feeding practices are reported in table 3. BLW prevalence increased with age for all groups and was highest for Māori and 'others'.

**Table 3.** Infant complementary feeding practices by ethnicity (n=625)

| | Māori | Pasifika | Non-Māori/Pasifika |
|---|---|---|---|
| n | 131 | 82 | 450 |
| Complementary feeding approach when first introduced to solids, n (%) | | | |
| Baby-led weaning | 15 (11.5) | 3 (3.7) | 56 (12.4) |
| Partial baby-led weaning | 5 (3.8) | 1 (1.2) | 18 (4.0) |
| Traditional spoon-feeding | 111 (84.7) | 78 (95.1) | 376 (83.3) |
| Complementary feeding approach at 6 months, n (%) | | | |
| Baby-led weaning | 18 (13.7) | 5 (6.1) | 61 (13.6) |
| Partial baby-led weaning | 22 (16.8) | 5 (6.1) | 44 (9.8) |
| Traditional spoon-feeding | 91 (69.5) | 72 (87.8) | 345 (76.7) |
| Current complementary feeding approach, n (%) | | | |
| Baby-led weaning | 38 (29.2) | 14 (17.1) | 123 (27.3) |
| Partial baby-led weaning | 32 (24.6) | 21 (25.6) | 109 (24.2) |
| Traditional spoon-feeding | 60 (46.2) | 47 (57.3) | 218 (48.4) |
| Ever used baby-led weaning | | | |
| Don't know what it is | 27 (20.8) | 22 (26.8) | 30 (6.7) |
| Yes, all or most of the time | 23 (17.7) | 10 (12.2) | 80 (17.8) |
| Yes, some of the time | 50 (38.5) | 29 (35.4) | 206 (45.8) |
| Yes, tried it but stopped | 9 (6.9) | 4 (4.9) | 22 (4.9) |
| No | 21 (16.2) | 17 (20.7) | 112 (24.9) |

Types and textures of first CFs provided to infants are reported in table 4. Most infants had a pureed textured first food (Māori: 78.5%; Pasifika 81.7%; 'others': 81.1%). Over half of all infants had red meat at approximately six months of age (Māori: 54.6%; Pasifika 63.4%; 'others': 61.8%), and approximately half had iron-fortified baby rice at the same age (Māori: 57.3%; Pasifika 56.1%; 'others': 48.7%). Age-inappropriate drinks (drinks other than breast milk, formula, and water) were given to 17.6% of Māori infants, 20.7% of Pasifika infants and 3.8% of 'other' infants. Vegetables were the most common first food offered to the infants (Māori: 38.5%; Pasifika 35.4%; 'others':

52.0%), and traditional foods, or traditional practices for food preparation, were used with 9.1% of Māori infants and 20.7% of Pasifika infants.

**Table 4.** Types and textures of complementary foods

| | Māori | Pasifika | Non-Māori/Pasifika |
|---|---|---|---|
| Texture of first food, n (%) | | | |
| Pureed | 102 (78.5) | 67 (81.7) | 365 (81.1) |
| Mashed | 10 (7.7) | 11 (13.4) | 31 (6.9) |
| Chopped | 0 | 0 | 1 (0.2) |
| Finger food | 17 (13.1) | 3 (3.7) | 44 (9.8) |
| Other | 1 (0.8) | 1 (1.2) | 9 (2.0) |
| Baby rice consumed at ~6 months of age, n (%) | | | |
| Yes | 75 (57.3) | 46 (56.1) | 219 (48.7) |
| No, offered but baby refused | 12 (9.2) | 12 (14.6) | 47 (10.4) |
| No, I don't agree with it | 18 (13.7) | 8 (9.8) | 96 (21.3) |
| No, I don't think it's safe | 1 (0.8) | 3 (3.7) | 9 (2.0) |
| No, my baby hadn't started solids | 3 (2.3) | 2 (2.4) | 3 (0.7) |
| No, another reason | 22 (16.8) | 11 (13.4) | 76 (16.9) |
| Frequency of baby rice consumption at ~6 months of age, n (%) | | | |
| Never | 56 (42.8) | 36 (43.9) | 231 (51.3) |
| Infrequently (<4 times per month) | 5 (3.8) | 1 (1.2) | 17 (3.8) |
| Sometimes (1–4 times per week) | 25 (19.1) | 16 (19.5) | 73 (16.2) |
| Frequently (5+ times per week) | 45 (34.4) | 29 (35.4) | 129 (28.7) |
| Red meat consumed at ~6 months of age, n (%) | | | |
| Yes | 71 (54.6) | 52 (63.4) | 278 (61.8) |
| No, offered but baby refused | 9 (6.9) | 4 (4.9) | 23 (5.1) |
| No, I don't agree with it | 6 (4.6) | 2 (2.4) | 25 (5.6) |
| No, I don't think it's safe | 11 (8.5) | 8 (9.8) | 32 (7.1) |

| | Māori | Pasifika | Non-Māori/Pasifika |
|---|---|---|---|
| No, my baby hadn't started solids | 7 (5.4) | 3 (3.7) | 17 (3.8) |
| No, another reason | 26 (20.0) | 13 (15.9) | 75 (16.7) |
| Frequency of red meat consumption at ~6 months of age, n (%) | | | |
| Never | 60 (45.8) | 30 (36.6) | 172 (38.2) |
| Infrequently (<4 times per month) | 7 (5.3) | 6 (7.3) | 28 (6.2) |
| Sometimes (1–4 times per week) | 49 (37.4) | 32 (39.0) | 182 (40.4) |
| Frequently (5+ times per week) | 15 (11.5) | 14 (17.1) | 68 (15.1) |
| Other drinks currently offered, n (%) | | | |
| Cow's milk, soy milk, or other milk alternative | 7 (5.3) | 1 (1.2) | 3 (0.7) |
| Tea | 2 (1.5) | 0 | 0 |
| Fruit juices/drinks | 13 (9.9) | 13 (15.9) | 14 (3.1) |
| Coconut water | 1 (0.8) | 3 (3.7) | 0 |
| First food offered, n (%) | | | |
| Baby rice | 30 (23.1) | 24 (29.3) | 84 (18.7) |
| Fruit | 37 (28.5) | 20 (24.4) | 98 (21.8) |
| Vegetables | 50 (38.5) | 29 (35.4) | 234 (52.0) |
| Bread/cereals | 2 (1.5) | 1 (1.2) | 13 (2.9) |
| Other | 8 (6.15) | 6 (7.3) | 8 (1.8) |
| Meat | 3 (2.31) | 2 (2.4) | 13 (2.9) |
| Traditional foods or practices used when starting solids, n (%) yes | 12 (9.2) | 16 (19.5) | 26 (5.8) |
| Traditional or cultural foods are currently given to baby, n (%) yes | 12 (9.2) | 17 (20.7) | 23 (5.1) |

Prevalence of pouch use is reported in table 5. The table data shows most infants have tried a pouch, and an increasing prevalence of pouch use between being introduced to CFs and six months of age.

**Table 5.** Pouch use and ethnicity

| Use of baby food pouches, n (%)[a] | Māori | Pasifika | Others |
|---|---|---|---|
| When first introduced to solids | 51 (38.9) | 29 (35.4) | 104 (23.1) |
| At 6 months | 85 (64.9) | 48 (58.5) | 194 (43.1) |
| Ever | 117 (89.3) | 70 (85.4) | 340 (75.6) |

[a]Commercial baby food pouches with a nozzle attached

The frequency of pouch use by ethnicity at the time of starting CFs, at six months of age, and currently is shown in Figure 1. Data within the figure illustrates that pouch use increases with age and is shown as %, with full data reported in supplementary table I.

Data in figure 1 shows pouch use by ethnicity at the time of starting CFs, at six months of age, and illustrates that pouch use increases with time.

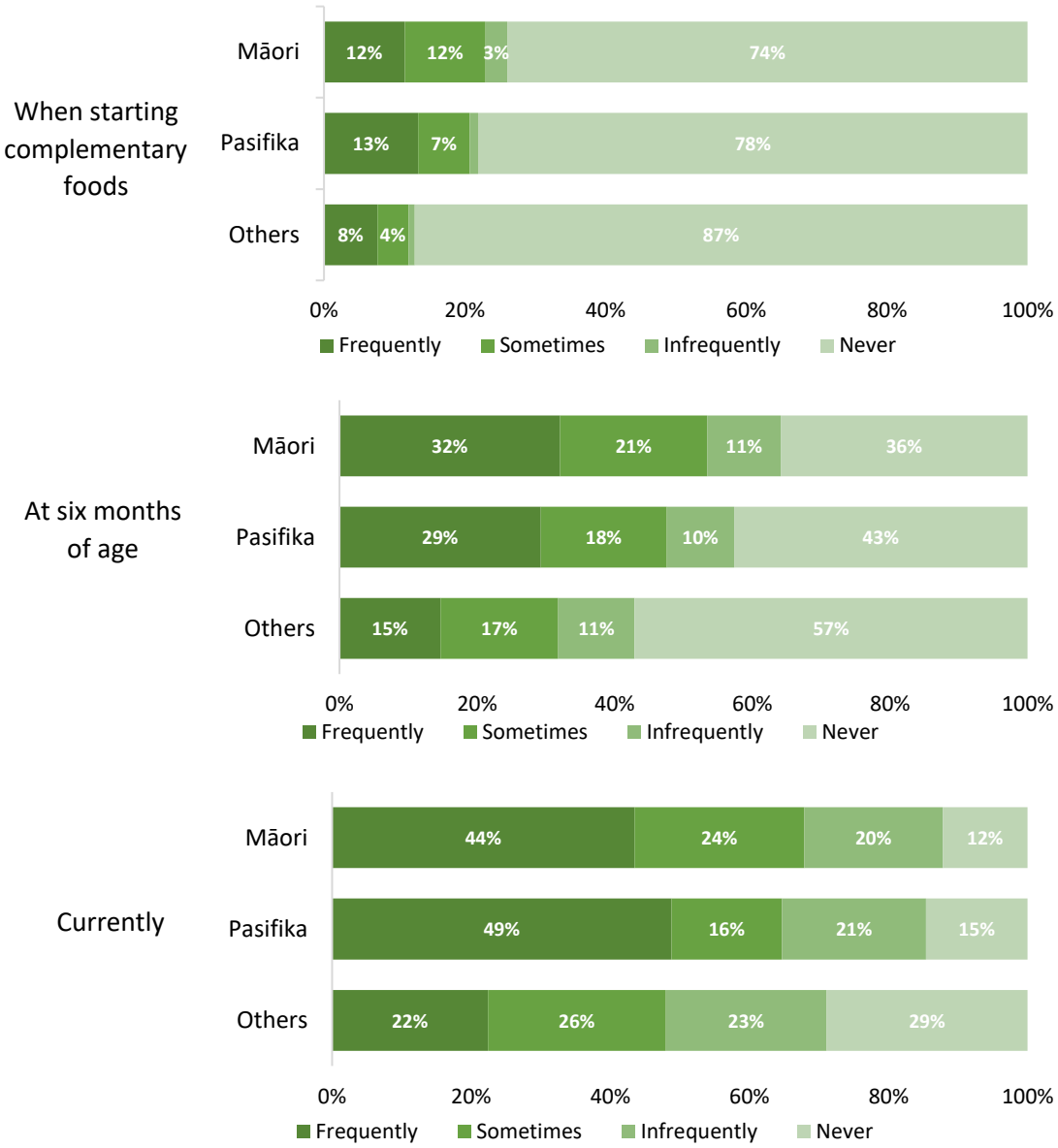



**Figure 1.**
Frequency of pouch use by ethnicity when starting complementary foods, at 6 months of age, and currently. 'Infrequently': <4 times per month; 'Sometimes': 1–4 times per week; 'Frequently': 5+ times per week. Māori: n=131; Pasifika: n=82; non-Māori/Pasifika: n=450.

The way in which pouches were fed at time of starting CFs, at six months of age, and currently is shown in figure 2. An increasing proportion of infants from all three groups were fed directly from the nozzle over time, with the percent shown in the figure and full data shown in supplementary table I.

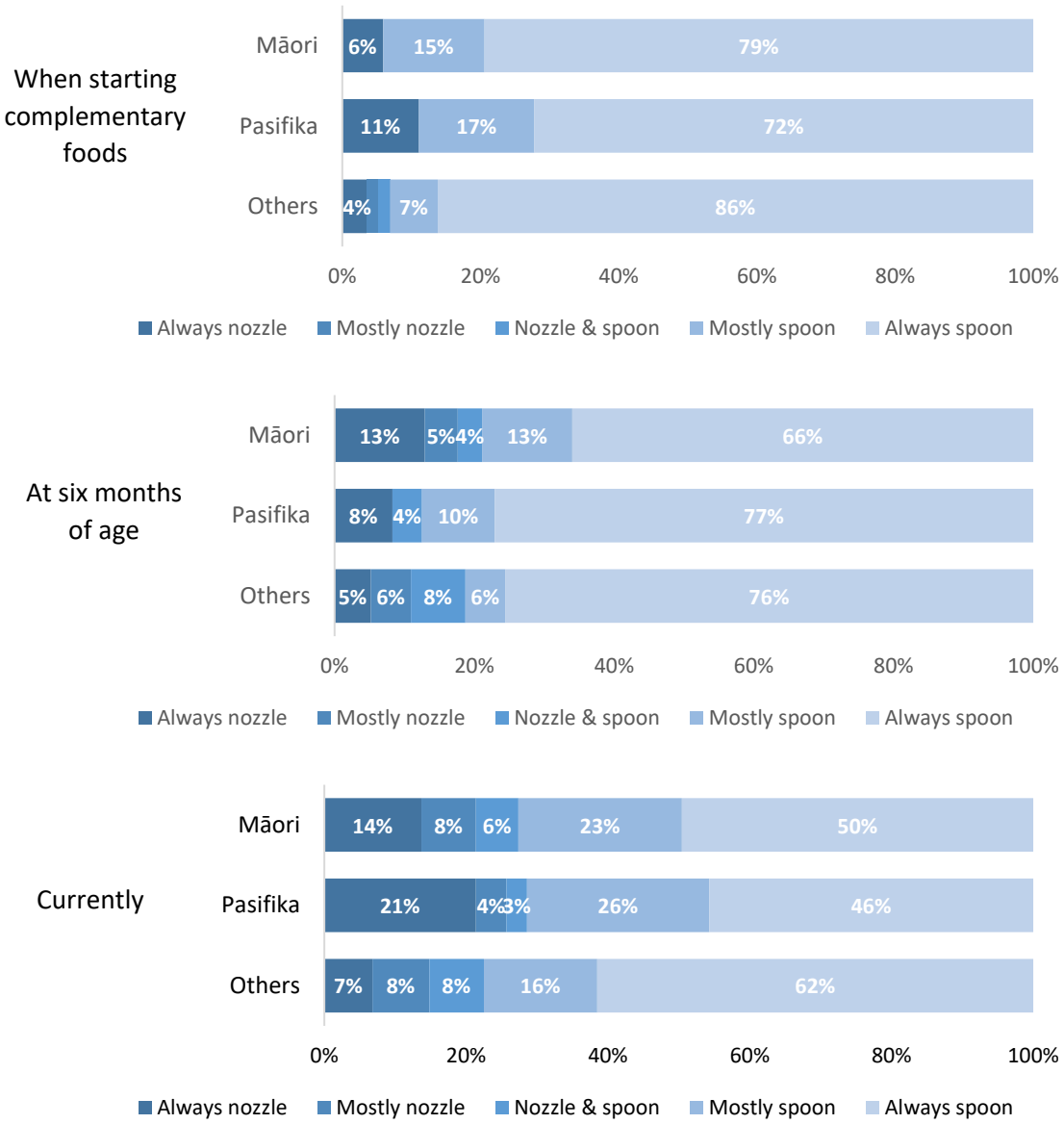



**Figure 2.** How pouches were fed by ethnicity when starting complementary foods, at 6 months, and currently.

The proportion of infants sucking the food from the nozzle stratified by how frequently pouches were used is shown in figure 3. For infants who 'frequently' had pouches, 27.1% of Māori, 25% of Pasifika, and 12% of 'others' always or mostly sucked from the nozzle. Proportion data is displayed in supplementary table II. Proportion data for the time points of "around six months of age" and "when starting solids" are shown in supplementary tables III and IV respectively.

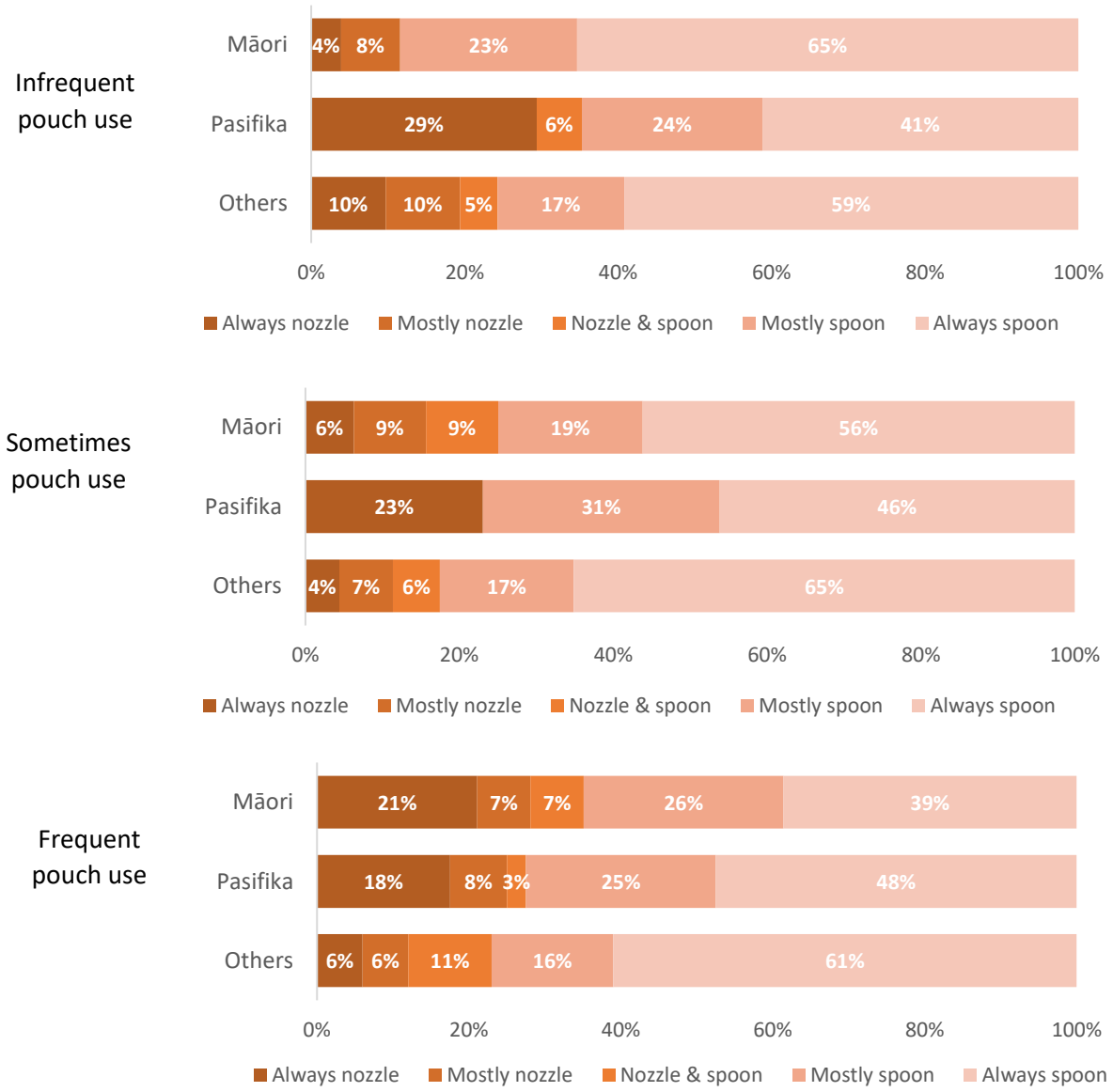



**Figure 3.** Proportion of infants currently fed by nozzle stratified by frequency of pouch use. 'Infrequently': <4 times per month; 'Sometimes': 1–4 time per week; 'Frequently': 5+ times per week.

When asked about traditional cultural infant feeding practices, six Pasifika caregivers described using ma ma, (pre-mastication) to modify the texture and temperature of the infant's food. Two Pasifika and two Māori caregivers reported using a hāngī or umu to cook food. The most common traditional food reported was taro, followed by taro leaves. Supplementary table V shows all participant responses describing cultural foods and practices.

## Discussion

The changing landscape of complementary feeding practices stands to have both positive and negative effects on child growth and development. Given that Māori and Pasifika infants bear the greatest burden of inequitable health outcomes related to nutrition in NZ it is important that research considers the equity impacts of changing feeding practices. FFNZ identified and described detailed characteristics of current infant feeding practices in Māori and Pasifika infants living in NZ, including novel infant feeding methods of BLW and PF. The timing of the introduction to CFs, characteristics of first foods offered, pouch use, and prevalence of BLW have been stratified by ethnicity to allow for an intra-ethnic evaluation. These aspects of infant feeding have the potential to impact nutrition-related health outcomes for infants.

### *Summary of findings*

The mean age of initiating complementary feeding was considerably younger than the MoH and WHO guidelines across all ethnicities. The prevalence of BLW across all ethnicities was lowest when the infants were first introduced to CFs, and highest when the infants were aged between 7 and 10.0 months. The majority of caregivers in all ethnicities had tried BLW at least once. Both prevalence and frequency of pouch use were lowest at the time of introducing CFs and highest at the current time across all ethnicities. The proportion of infants feeding directly from the nozzle increased from when CFs were first introduced to the present time. The majority of infants consumed either red meat or iron-fortified baby rice at the age of six months. The prevalence of drinks other than formula, breast milk, or water offered was low. First foods offered were predominantly vegetables for all ethnicities. There is little evidence of use of cultural foods and practices, possibly as these are the norm, and not seen as “special” practices.

### *Timing of introduction of complementary foods*

The WHO recommend that CFs are introduced when the infant is around six months of age (1), advice echoed by the MoH in NZ (19). Late introduction is associated with increased risks for food allergies, faltering growth (20, 21), iron deficiency anaemia (22), poor food acceptance (23), and later development of oral motor skills (24). Early introduction is associated with increased iron deficiency, gastrointestinal infections (20, 25, 26), and respiratory illnesses (27). Infants across all ethnicities in the present study were represented in those who were introduced to solid foods earlier than recommended. Comparisons to other studies is made challenging by the use of differing cut-offs to define food introduction as non-timely. The Growing Up in New Zealand (GUiNZ) study investigated the early introduction of CFs by ethnicity in a cohort of 5725 infants born between 2007

and 2010, and found that 57% of Māori, 46% of Pasifika, and 36% of 'other' infants started CFs at ≤4 months (28). Grant et. Al. (2007) documented time of food introduction in an ethnically diverse cohort (*n*=318) and found that 21% introduced CFs between 0 and 3 months of age, 54% between five and six months of age, and 24% at six months or older. The findings from the present study suggest that the practice of early introduction is diminishing (41% of Māori infants, 34% of Pasifika infants, and 17% of 'other' infants) but is still concerning, particularly in light of the higher burden of gastrointestinal and respiratory tract illnesses reported for Māori and Pasifika infants (27, 29-33).

*Baby-led weaning prevalence*

The prevalence of BLW was measured at three different time points: when initiating complementary feeding, at six months of age, and currently. Infants were most likely to be baby-led weaned at the 'current' time point. Infant feeding guidelines recommend the introduction of finger food between seven and nine months of age, and because BLW was defined by degree to which the infant was self-feeding, it is not clear if the increased prevalence of BLW at this time point is due to this, or a specific intention by the parent to use BLW. The rate of BLW at around six months of age may be a better indicator of the true proportion, as it is probable that a greater proportion of infants would be naturally having finger foods and self-feeding between seven and ten months than at 6 months. This study found that at 6 months of age 13.7% of Māori, 6.1% of Pasifika, and 13.6% of 'other' infants identified as being baby-led weaned. This suggests that there is a low prevalence of BLW in all ethnic groups, and although these findings may not be generalisable to the general population, it is a large cohort with diverse socio-economic backgrounds and ethnicities.

While this study provides some insight into the prevalence of BLW in NZ, this data must be interpreted with caution due to a self-selected sample from two urban centres. BLW was not referred to during the recruitment stages to help reduce bias. A small 2013 study (n=199) in NZ completed an online survey, showing that 21% of the sample reported using partial BLW, and 8% used strict BLW. A second online survey in 2018 (n=876) found that 11% of the sample followed a partial BLW approach, and 18% used strict BLW. Both studies are subject to self-selection bias with the sampling method, and the latter study had an average infant age of 17 months, introducing the potential for recall bias.

*Baby food pouch use*

Baby food pouches are relatively new to the market but have rapidly become the predominant form of packaging. Studies investigating the prevalence of pouch are scarce, and very limited research on

whether the infant feeds directly from the pouch or via a spoon was identified. In the present study pouches were used by the majority of infants across all ethnicities, with 67.9% of Māori, 64.7% of Pasifika, and 48% of 'other' infants having pouches frequently or sometimes. The proportion of infants feeding directly from the pouch increased with age in the present study. This is a troubling trend, as expert groups have advised against sucking food directly from pouches due to concerns around potential detrimental impact to dental health, nutrient and energy intake (11, 34).

Pouches in NZ are higher in total sugar than equivalent CIFs in other packaging, with more than three times as much total sugars in pouches than equivalent non-pouches (8). The higher total sugars and acid content of fruit pouches in particular increases the risk of dental caries developing. This risk is exacerbated when the product is sucked directly from the pouch nozzle, as the teeth are bathed in the food for a longer period of time than with spoon feeding or finger food (11). Increased total sugars intake may also decrease dietary diversity, prevent adequate nutrient intake through displacement of other nutrients, and can be associated with increased risk in later life of type 2 diabetes mellitus and cardiovascular disease (35-37). Infants also have an innate preference for sweet tastes, and increased exposure may reinforce this preference long term (37). Māori and Pasifika children have a high rate of dental caries and as adults a large burden of diabetes and associated co-morbidities. In light of this, it is concerning that the majority of Māori and Pasifika infants in the present study used pouches at least once a week, and that the majority of these users regularly sucked the food directly from the pouch.

Iron is another nutrient of concern with pouches. A 2021 review of pouches in NZ revealed that none were fortified with iron and contained a median of just 0.3 mg/100g (8). Dry infant cereal in NZ has mandatory minimum requirements for iron fortification of 20 mg/100g (38), however as pouch food is in wet form, it is exempt from these requirements. Additionally, meat-based pouches contain little meat, with an average of just 5–10% — even if the pouch label has beef, lamb, or pork in the title (8). This presents a particular concern given that the majority of Māori and Pasifika infants having pouches frequently or sometimes, and historically are suggested to have high rates of ID and IDA.

With pouches rapidly becoming the predominant form of packaging for CIF, further research into why there is such a discrepancy between the iron and sugar content compared with tinned, jar, or boxed CIF. The current legislation for iron fortification is only required for dry cereal-based foods, which may account for the lack of iron in pouch food, which is in wet form. There is also no minimum requirement for the amount of meat contained in a meat-based CIF which could further

impact the iron content. In regard to the higher sugar content, it is unclear whether this is related to a manufacturing process or for a desire to make the food more palatable to infants.

From a cultural perspective for Māori infants, the practice of infants self-feeding with pouches is a move away from the mātauranga Māori of traditional cultural infant feeding practices that support a strong connection to te ao Māori: infants who self-feed via the nozzle do not benefit from the close and responsive interaction of eating with and sharing the whānau meals. Similarly for Pasifika infants, the sharing of foods and eating with the 'aiga (family) stems from the role of food in togetherness, health, and tradition. However, it is important to acknowledge that the convenience of pouches meet a need and fit with the lived reality of many whānau. Ways to mitigate the negative impacts of pouch use while understanding the motivation for their use need to be considered.

*Strengths and limitations*

This is the first study to comprehensively describe infant complementary feeding practices in NZ by ethnicity. Strengths include the large sample size, and the range of ethnicities reflecting that of the greater NZ population. Limitations of the study were a self-selecting sample. The study also only included infants living in two main urban centres so may not be representative of the general population. Care was taken to ensure BLW was not mentioned during advertising and the recruitment stages to limit bias. The way in which BLW was measured at the current time point (7–10 months) may result in an over-representation of the true prevalence. BLW was determined by the amount the infant self-fed, without specifically asking if BLW was used. However, this is a time when finger foods are generally introduced, so it is not clear if the increased prevalence at this time point is due to this, or a specific intention by the parent to use BLW.

<u>Conclusion</u>

This study has provided insight into complementary feeding practices in NZ. The key areas of concern identified were the prevalence of early introduction to CF, the prevalence of pouch use, and the proportion of infants sucking directly from the pouch nozzle. CF was introduced at ≤4 months for less than half of all three groups. Pouch use was common amongst all ethnicities and increased with time. Concerningly, of infants who frequently consumed pouches fewer than half of Māori and Pasifika infants and just over half of 'other' infants were always fed pouches from a spoon. This understanding of where the greatest support for infant feeding is needed will help inform future research. This is important information for our most vulnerable populations, as under Te Tiriti o Waitangi obligations all whānau in NZ have the right to an environment where healthful traditional

cultural food and practices are readily accessible, but due to social determinants disproportionately experienced by Māori whānau and the Pasifika community these groups are over-represented in poor health outcomes. Facilitating Māori- and Pasifika-centred approaches to supporting whānau and communities with infant feeding need to be urgently implemented to better support parents and caregivers in providing the best possible nutrition to their tamariki.

References

1. World Health Organization and UNICEF. Global strategy for infant and young child feeding: World Health Organization; 2003.
2. Ministry of Health. Healthy Eating Guidelines for New Zealand Babies and Toddlers (0-2 years old). Wellington, New Zealand: Ministry of Health; 2021.
3. Rapata H, Heath A-LM, Wall C, Taylor R, Te Morenga L. Māori first foods: a Māori centred approach to understanding infant complementary feeding practices within Māori whānau. Kōtuitui: New Zealand Journal of Social Sciences Online. 2021:1-16.
4. Malolo M, Matenga-Smith To, Hughes R. The Staples We Eat. Noumea, New Caledonia; 1999.
5. Taonui R. Tribal organisation - The history of Māori social organisation: Te Ara - the Encyclopedia of New Zealand; 2005 [1 November 2022]. Available from: http://www.TeAra.govt.nz/en/tribal-organisation/page-6.
6. Garcia AL, Curtin L, Ronquillo JD, Parrett A, Wright CM. Changes in the UK baby food market surveyed in 2013 and 2019: the rise of baby snacks and sweet/savoury foods. Archives of Disease in Childhood. 2020;105(12):1162-6.
7. Padarath S, Gerritsen S, Mackay S. Nutritional aspects of commercially available complementary foods in New Zealand Supermarkets. Nutrients. 2020;12(10):2980.
8. Katiforis I, Fleming EA, Haszard JJ, Hape-Cramond T, Taylor RW, Heath A-LM. Energy, Sugars, Iron, and Vitamin B12 Content of Commercial Infant Food Pouches and Other Commercial Infant Foods on the New Zealand Market. Nutrients. 2021;13(2):657.
9. Beauregard JL, Bates M, Cogswell ME, Nelson JM, Hamner HC. Nutrient content of squeeze pouch foods for infants and toddlers sold in the United States in 2015. Nutrients. 2019;11(7):1689.
10. Koletzko B, Hirsch NL, Jewell JM, Caroli M, Breda JRDS, Weber M. Pureed fruit pouches for babies: child health under squeeze. Journal of Pediatric Gastroenterology and Nutrition. 2018;67(5):561-3.
11. Koletzko B, Bührer C, Ensenauer R, Jochum F, Kalhoff H, Lawrenz B, et al. Complementary foods in baby food pouches: position statement from the Nutrition Commission of the German Society for Pediatrics and Adolescent Medicine (DGKJ, eV). Molecular and Cellular Pediatrics. 2019;6(1):1-5.
12. Moding KJ, Ferrante MJ, Bellows LL, Bakke AJ, Hayes JE, Johnson SL. Nutritional content and ingredients of commercial infant and toddler food pouches compared with other packages available in the United States. Nutrition Today. 2019;54(6):305.
13. Cameron SL, Taylor RW, Heath A-LM. Parent-led or baby-led? Associations between complementary feeding practices and health-related behaviours in a survey of New Zealand families. British Medical Journal Open. 2013;3(12).
14. Fu X, Conlon CA, Haszard JJ, Beck KL, von Hurst PR, Taylor RW, et al. Food fussiness and early feeding characteristics of infants following Baby-Led Weaning and traditional spoon-feeding in New Zealand: An internet survey. Appetite. 2018;130:110-6.
15. Shackleton N, Broadbent JM, Thornley S, Milne BJ, Crengle S, Exeter DJ. Inequalities in dental caries experience among 4-year-old New Zealand children. Community Dentistry and Oral Epidemiology. 2018;46(3):288-96.
16. Schluter PJ, Lee M. Water fluoridation and ethnic inequities in dental caries profiles of New Zealand children aged 5 and 12–13 years: analysis of national cross-sectional registry databases for the decade 2004–2013. BMC Oral Health. 2016;16(1):1-10.
17. Taylor RW, Conlon CA, Beck KL, von Hurst PR, Te Morenga LA, Daniels L, et al. Nutritional Implications of Baby-Led Weaning and Baby Food Pouches as Novel Methods of Infant Feeding: Protocol for an Observational Study. JMIR Research Protocols. 2021;10(4):e29048.
18. Atkinson J, Salmond C, Crampton P. NZDep2018 Index of Deprivation, Interim Research Report. Wellington: University of Otago; 2019.

19. Gerritsen S, Mackay S, Jackson A. Healthy Eating Guidelines for New Zealand Babies and Toddlers (0-2 years old). 2021. Report No.: 1991007388.
20. Przyrembel H. Timing of introduction of complementary food: short-and long-term health consequences. Annals of Nutrition and Metabolism. 2012;60(Suppl. 2):8-20.
21. Butte NF, Lopez-Alarcon MG, Garza C. Nutrient adequacy of exclusive breastfeeding for the term infant during the first six months of life: World Health Organization; 2002.
22. Wang F, Liu H, Wan Y, Li J, Chen Y, Zheng J, et al. Age of complementary foods introduction and risk of anemia in children aged 4–6 years: a prospective birth cohort in China. Scientific Reports. 2017;7(1):1-8.
23. Agostoni C, Decsi T, Fewtrell M, Goulet O, Kolacek S, Koletzko B, et al. Complementary feeding: a commentary by the ESPGHAN Committee on Nutrition. Journal of Pediatric Gastroenterology and Nutrition. 2008;46(1):99-110.
24. Coulthard H, Harris G, Emmett P. Delayed introduction of lumpy foods to children during the complementary feeding period affects child's food acceptance and feeding at 7 years of age. Maternal & Child Nutrition. 2009;5(1):75-85.
25. Ministry of Health. Food and Nutrition Guidelines for Healthy Infants and Toddlers (Aged 0-2): A background paper (4th Ed) - Partially Revised December 2012). Wellington, New Zealand: Ministry of Health; 2008.
26. Dewey K. Guiding principles for complementary feeding of the breastfed child2003.
27. Wilson AC, Forsyth JS, Greene SA, Irvine L, Hau C, Howie PW. Relation of infant diet to childhood health: seven year follow up of cohort of children in Dundee infant feeding study. British Medical Journal. 1998;316(7124):21-5.
28. Ferreira SS, Marchioni DML, Wall CR, Gerritsen S, Teixeira JA, Grant CC, et al. Prevalence and maternal determinants of early and late introduction of complementary foods: results from the Growing up in New Zealand cohort study. British Journal of Nutrition. 2022:1-12.
29. Wright CM, Parkinson K, Drewett R. Why are babies weaned early? Data from a prospective population based cohort study. Archives of Disease in Childhood. 2004;89(9):813-6.
30. Armentia A, Banuelos C, Arranz M, Del Villar V, Martín-Santos JM, Gil FM, et al. Early introduction of cereals into children's diets as a risk-factor for grass pollen asthma. Clinical & Experimental Allergy. 2001;31(8):1250-5.
31. Huh SY, Rifas-Shiman SL, Taveras EM, Oken E, Gillman MW. Timing of solid food introduction and risk of obesity in preschool-aged children. Pediatrics. 2011;127(3):e544-e51.
32. Kramer MS, Kakuma R. The optimal duration of exclusive breastfeeding. Protecting Infants Through Human Milk2004. p. 63-77.
33. Grant CC, Wall CR, Brunt D, Crengle S, Scragg R. Population prevalence and risk factors for iron deficiency in Auckland, New Zealand. Journal of Paediatrics and Child Health. 2007;43(7-8):532-8.
34. Theurich MA, Fewtrell M, Baumgartner J, Perkin MR, Breda J, Wickramansinghe K, et al. Moving Complementary Feeding Forward: Report on a Workshop of the Federation of International Societies for Pediatric Gastroenterology, Hepatology and Nutrition (FISPGHAN) and the World Health Organization Regional Office for Europe. Journal of Pediatric Gastroenterology and Nutrition. 2022;75(4):411-7.
35. Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH, et al. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. Circulation. 2009;120(11):1011-20.
36. Slavin J. Beverages and body weight: challenges in the evidence-based review process of the Carbohydrate Subcommittee from the 2010 Dietary Guidelines Advisory Committee. Nutrition Reviews. 2012;70(suppl_2):S111-S20.
37. Fidler Mis N, Braegger C, Bronsky J, Campoy C, Domellöf M, Embleton ND, et al. Sugar in infants, children and adolescents: a position paper of the European society for paediatric

gastroenterology, hepatology and nutrition committee on nutrition. Journal of Pediatric Gastroenterology and Nutrition. 2017;65(6):681-96.
38. Zealand FSAN. Australia New Zealand Food Standards Code—Standard 2.9.2—Food for Infants [12 August 2022]. Available from: https://www.legislation.gov.au/Details/F2017C00334.

Supplementary Tables

**Table I.** Frequency of pouch use and method of pouch feeding (shown diagrammatically in Figure X)

| n | Māori<br>131 | Pasifika<br>82 | Non-Māori/Pasifika<br>450 |
|---|---|---|---|
| *Frequency of pouch use when starting solids, n (%)* | | | |
| Never | 97 (74.1) | 64 (78.1) | 392 (87.1) |
| Infrequently (<4 times per month) | 4 (3.1) | 1 (1.2) | 4 (0.9) |
| Sometimes (1–4 times per week) | 15 (11.5) | 6 (7.3) | 20 (4.4) |
| Frequently (5+ times per week) | 15 (11.5) | 11 (13.4) | 34 (7.6) |
| *Frequency of pouch use at 6 months, n (%)* | | | |
| Never | 47 (35.9) | 35 (42.7) | 257 (57.1) |
| Infrequently (<4 times per month) | 14 (10.7) | 8 (9.8) | 50 (11.1) |
| Sometimes (1–4 times per week) | 28 (21.4) | 15 (18.3) | 77 (17.1) |
| Frequently (5+ times per week) | 42 (32.1) | 24 (29.3) | 66 (14.7) |
| *Frequency of pouch use currently, n (%)* | | | |
| Never | 16 (12.2) | 12 (14.6) | 130 (28.9) |
| Infrequently (<4 times per month) | 26 (19.9) | 17 (20.7) | 104 (23.1) |
| Sometimes (1–4 times per week) | 32 (24.4) | 13 (15.9) | 115 (25.6) |
| Frequently (5+ times per week) | 57 (43.5) | 40 (48.8) | 101 (22.4) |
| *How pouches were fed when starting solids, n (%)* | n=34 | n=18 | n=58 |
| Always via nozzle | 2 (5.9) | 2 (11.1) | 2 (3.5) |
| Mostly via nozzle | 0 | 0 | 1 (1.7) |
| Both via nozzle and spoon | 0 | 0 | 1 (1.7) |
| Mostly via spoon | 5 (14.7) | 3 (16.7) | 4 (6.9) |

| | | | |
|---|---|---|---|
| Always via spoon | 27 (79.4) | 13 (72.2) | 50 (86.2) |
| *How pouches were fed at 6 months, n (%)* | n=85 | n=48 | n=192 |
| Always via nozzle | 11 (12.9) | 4 (8.3) | 10 (5.2) |
| Mostly via nozzle | 4 (4.7) | 0 | 11 (5.7) |
| Both via nozzle and spoon | 3 (3.5) | 2 (4.2) | 15 (7.8) |
| Mostly via spoon | 11 (12.9) | 5 (10.4) | 11 (5.7) |
| Always via spoon | 56 (65.9) | 37 (77.1) | 145 (75.5) |
| *How pouches were fed currently, n (%)* | n=117 | n=70 | n=338 |
| Always via nozzle | 16 (13.7) | 15 (21.4) | 23 (6.8) |
| Mostly via nozzle | 9 (7.7) | 3 (4.3) | 27 (8.0) |
| Both via nozzle and spoon | 7 (6.0) | 2 (2.9) | 26 (7.7) |
| Mostly via spoon | 27 (23.1) | 18 (25.7) | 54 (16.0) |
| Always via spoon | 58 (49.6) | 32 (45.7) | 208 (61.5) |

**Table II.** Frequency of pouch use and how pouches were fed by ethnicity in the last month (shown diagrammatically in Figure 1)

| | **Māori** | **Pacific** | **Others** |
|---|---|---|---|
| | **n (%)** | **n (%)** | **n (%)** |
| | n=115 | n=70 | n=318 |
| **Infrequently** | **n=26** | **n=17** | **n=103** |
| Always nozzle | 1 (3.9) | 5 (29.4) | 10 (9.7) |
| Mostly nozzle | 2 (7.7) | 0 | 10 (9.7) |
| Both nozzle & spoon | 0 | 1 (5.9) | 5 (4.9) |
| Mostly spoon | 6 (23.1) | 4 (23.5) | 17 (16.5) |
| Always spoon | 17 (65.4) | 7 (41.2) | 61 (59.2) |
| **Sometimes** | **n=32** | **n=13** | **n=115** |
| Always nozzle | 2 (6.3) | 3 (23.1) | 5 (4.4) |
| Mostly nozzle | 3 (9.4) | 0 | 8 (7.0) |
| Both nozzle & spoon | 3 (9.4) | 0 | 7 (6.1) |
| Mostly spoon | 6 (18.8) | 4 (30.8) | 20 (17.4) |
| Always spoon | 18 (56.3) | 6 (46.2) | 75 (65.2) |
| **Frequently** | **n=57** | **n=40** | **n=100** |
| Always nozzle | 12 (21.1) | 7 (17.5) | 6 (6.0) |
| Mostly nozzle | 4 (7.0) | 3 (7.5) | 6 (6.0) |
| Both nozzle & spoon | 4 (7.0) | 1 (2.5) | 11 (11.0) |
| Mostly spoon | 15 (26.3) | 10 (25.0) | 16 (16.0) |
| Always spoon | 22 (38.6) | 19 (47.5) | 61 (61.0) |

'Infrequently': <4 times per month: 'Sometimes': 1 – 4 times per week; 'Frequently': 5+ times per week.

**Table III.** Frequency of pouch use and how pouches were fed by ethnicity around six months of age (shown diagrammatically in Figure 2)

| | **Māori** | **Pacific** | **Others** |
|---|---|---|---|
| | **n (%)** | **n (%)** | **n (%)** |
| | n=84 | n=47 | n=191 |
| **Infrequently** | **n=14** | **n=8** | **n=49** |
| Always nozzle | 0 | 0 | 3 (6.1) |
| Mostly nozzle | 1 (7.1) | 0 | 4 (8.2) |
| Both nozzle & spoon | 0 | 0 | 1 (2.0) |
| Mostly spoon | 1 (7.1) | 1 (12.5) | 7 (14.3) |
| Always spoon | 12 (85.7) | 7 (87.5) | 34 (69.4) |
| **Sometimes** | **n=28** | **n=15** | **n=76** |
| Always nozzle | 3 (10.7) | 1 (6.7) | 4 (5.3) |
| Mostly nozzle | 2 (7.1) | 0 | 4 (5.3) |
| Both nozzle & spoon | 1 (3.6) | 1 (6.7) | 8 (10.5) |
| Mostly spoon | 3 (10.7) | 1 (6.7) | 2 (2.6) |
| Always spoon | 19 (67.9) | 12 (80.0) | 58 (76.3) |
| **Frequently** | **n=42** | **n=24** | **n=66** |
| Always nozzle | 7 (16.7) | 3 (12.5) | 3 (4.6) |
| Mostly nozzle | 1 (2.4) | 0 | 3 (4.6) |
| Both nozzle & spoon | 2 (4.8) | 1 (4.2) | 6 (9.1) |
| Mostly spoon | 7 (16.7) | 3 (12.5) | 1 (1.5) |
| Always spoon | 25 (59.5) | 17 (70.8) | 53 (80.3) |

'Infrequently': <4 times per month: 'Sometimes': 1 – 4 times per week; 'Frequently': 5+ times per week.

**Table IV.** Frequency of pouch use and how pouches were fed by ethnicity when starting solids (shown diagrammatically in Figure 3)

| | **Māori** | **Pacific** | **Others** |
|---|---|---|---|
| | **n (%)** | **n (%)** | **n (%)** |
| | n=34 | n=18 | n=58 |
| **Infrequently** | **n=4** | **n=1** | **n=4** |
| Always nozzle | 0 | 0 | 0 |
| Mostly nozzle | 0 | 0 | 0 |
| Both nozzle & spoon | 0 | 0 | 0 |
| Mostly spoon | 0 | 0 | 1 (25.0) |
| Always spoon | 4 (100) | 1 (100) | 3 (75.0) |
| **Sometimes** | **n=15** | **n=6** | **n=20** |
| Always nozzle | 0 | 0 | 1 (5.0) |
| Mostly nozzle | 0 | 0 | 1 (5.0) |
| Both nozzle & spoon | 0 | 0 | 0 |
| Mostly spoon | 3 (20.0) | 1 (16.7) | 2 (10.0) |
| Always spoon | 12 (80.0) | 5 (63.6) | 16 (80.0) |
| **Frequently** | **n=15** | **n=11** | **n=34** |
| Always nozzle | 2 (13.3) | 2 (18.2) | 1 (2.9) |
| Mostly nozzle | 0 | 0 | 0 |
| Both nozzle & spoon | 0 | 0 | 1 (2.9) |
| Mostly spoon | 2 (13.3) | 2 (18.2) | 1 (2.9) |
| Always spoon | 11 (73.3) | 7 (63.6) | 31 (91.2) |

'Infrequently': <4 times per month: 'Sometimes': 1 – 4 times per week; 'Frequently': 5+ times per week.

**Table V.** Māori and Pasifika cultural foods, feeding, and cooking practices (total response)

| Food | Māori, n | | Pasifika, n | |
|---|---|---|---|---|
| | When starting solids | Currently | When starting solids | Currently |
| Kumara | 1 | 0 | 2 | 3 |
| Taro | 2 | 2 | 5 | 7 |
| Taro leaves | 3 | 3 | 4 | 6 |
| Corned beef | 0 | 0 | 0 | 1 |
| Pumpkin | 1 | 0 | 1 | 0 |
| Green banana | 0 | 0 | 0 | 1 |
| Coconut cream | 1 | 1 | 1 | 1 |
| Titi (mutton bird) | 1 | 1 | 1 | 1 |
| Cassava | 0 | 1 | 1 | 1 |
| ‘Ota ika/Oka I’a (raw fish salad) | 0 | 0 | 0 | 1 |
| Kaimoana (seafood) | 1 | 0 | 1 | 3 |
| Palusami/Lu Sipi/Lu Pulu* | 0 | 0 | | 3 |
| Chop-suey | 0 | 0 | 1 | 0 |
| Koko rice** | 0 | 1 | 0 | 1 |
| **Practices / Cooking methods** | | | | |
| Karakia before eating | 1 | 0 | 1 | 0 |
| Food cooked in h**ā**ng*ī* or umu | 1 | 1 | 2 | 1 |
| Ma ma (Pre-mastication) | 1 | 0 | 6 | 0 |
| Boil-up | 1 | 1 | 1 | 1 |
| Eating together with the whānau | 1 | 0 | 1 | 0 |

*Corned beef or lamb with vegetables and coconut milk, wrapped in taro leaves and cooked.
** Samoan cocoa with rice

# Chapter 5 | Upoko Tuarima

## Energy and nutrient intake from complementary foods amongst Māori, Pasifika, and other infants in Aotearoa New Zealand

*This chapter reports the findings of the First Foods New Zealand study relating to nutrient intake from complementary foods and the nutrient density of complementary foods by ethnicity.*

*This report is presented in manuscript format and will be submitted to a peer-reviewed journal for publication in the future. Online statement of contribution can be reviewed in Appendix I.*

Abstract

**Background:** Complementary food (CF) has the critical role of filling the gap between the nutrients provided via breast or formula milk and the total requirements of the infant. Due to the high nutrient requirements of infants, CFs must be nutrient dense to fulfil nutrient requirements while meeting the energy requirements. Understanding where the nutrient gaps are in the infant's complementary diet is important to help identify interventions and future research, particularly for Māori and Pasifika infants who have a higher burden of disease than other ethnicities in New Zealand (NZ).

**Aim:** The aim of this study was to describe the nutrient intake and nutrient density from complementary foods amongst Māori, Pasifika, and 'other' infants living in NZ.

**Methods:** Data were collected from 625 infants aged 7–10.0 months in two main urban centres in NZ between July 2020 and February 2022. Usual nutrient intake from complementary food was measured from two multiple-pass 24-hour recalls using the multiple-source method. Nutrient density was calculated as the concentration of selected nutrients per 418 kJ (100 kcal) of energy. Ethnicity was reported as total response, with those who did not select either Māori or Pacific categorised into a single 'other' group.

**Results:** Energy intake increased between 7.0 and 10.0 months of age for all groups and was higher overall for boys than girls. Māori and Pasifika girls had a lower energy intake from CF than 'other' girls, and Pasifika boys had a higher energy intake from CFs than Māori and 'other' boys. Māori infants had significantly less protein intake as a percentage of total energy intake compared to 'other' infants, and both Māori and Pasifika had a significantly lower proportion of energy from fat than 'others', with 27%, 25%, and 31% respectively. The proportion of energy from carbohydrates was significantly lower for 'others' than for Māori and Pasifika. The proportion of sugar as a percentage of total energy intake was 25% for both Māori and Pasifika, and 22% for 'others'. This difference was significant between Māori and 'others', and there was a non-significant but trending difference between Pasifika and 'others'. Iron, zinc, and calcium densities were inadequate for all groups. Māori and 'others' had an iron density of 0.8 mg/418 kJ, and Pasifika 0.9 mg/418 kJ, compared to the required density of 3.0–4.5 mg/418 kJ. Zinc density was 0.5 mg/418 kJ for all groups compared to the required 1.1–16 mg/418 kJ. Calcium density was 37 mg/418 kJ for Māori, 40 mg/418 kJ for Pasifika, and 38 mg/418 kJ for 'others', compared to the required density of 74 – 105 mg/418 kJ. The desired density for vitamin A, vitamin C, and vitamin $B_{12}$ was met for all groups.

**Conclusion:** These findings provide greater understanding of the nutrient quality of the complementary diet for infants in NZ, and show a consistent inadequate nutrient density for iron,

zinc, and calcium among infants of all ethnic groups. More research to assess ways to improve nutrient density to help close the current gaps is needed.

Introduction

Complementary foods (CF) are recommended from approximately six months of age, when infants' nutritional requirements start to exceed that provided by breast milk or infant formula alone and coincide with developmental readiness for eating solid food (1, 2). The infant needs to obtain essential nutrients for healthy growth and development from a range of foods. This is also a time where food preferences and eating behaviours are shaped, with the infant having the opportunity to explore different tastes and textures. There is extensive evidence to support the relationship between nutrition from complementary food and health outcomes throughout the life cycle, particularly non-communicable diseases such as type 2 diabetes and cardiovascular disease (3, 4), and neurocognitive development related to iron status (5). Despite this, very little is known about the nutrient intake of infants from CFs in Aotearoa New Zealand (NZ).

Key nutrients infants require from CFs are energy, protein, fat, carbohydrate, fibre iron, zinc, calcium, iodine, selenium, folate, and vitamins A, $B_{12}$ and C. Other key nutrients that are not primarily obtained via the diet are fluoride and vitamin D (2). Typical assessment of the nutritional adequacy of the infant's diet involves measuring the nutrient intake from CFs and milk feeds, followed by a comparison to the estimated average requirements (EAR). However, this approach does not take into consideration energy, as the nutrient requirements stay the same regardless of energy intake. Using nutrient density (the ratio of nutrient to energy) captures energy intake as well. Developing this further, a reference nutrient density can be established, and this is referred to as the "*critical nutrient density*" (6). A diet with an adequate critical nutrient density will meet the recommended intake when the energy requirements are met, with any gaps between the nutrient density of the diet and the critical nutrient density highlighting the nutrients of concern. This is a particularly useful approach to apply to the infant's complementary diet as CFs are appropriate to manipulate (unlike breast or formula milk).

Infants have high nutrient requirements per kilogram of body weight compared to any other life stage, so CF must be nutrient dense to close the gap. Excess saturated fat and sugar from CFs on the other hand have a detrimental impact on the health of infants, is linked to increased dental caries, reduced dietary diversity, and development a sweet taste preference that can be life-long (7), and in later life increased morbidity — particularly type 2 diabetes and cardiovascular disease (7-9).

With ethnicity being the greatest predictor of nutrition-related health outcomes in NZ (10), it is important to understand where the nutrient gaps are in the infant diet – particularly for Māori and Pasifika infants, who bear the greatest burden of communicable and non-communicable disease (11, 12). Currently very little is known about the CF intake of infants in NZ, and while some insight was provided by the Baby-Led Introduction to Solids (BLISS) study indicating adequate energy, protein, fat, vitamin C, calcium, and zinc, but inadequate carbohydrate and iron from CF and milk feeds combined (13), these data come from a small control group of 77 infants and does not examine nutrients from CFs. The Growing Up In New Zealand study found that Māori and Pasifika infants had a lower adherence to feeding guidelines (14), which is suggestive of a poorer quality diet, but as data were obtained from food frequency questionnaires, nutrient density calculations are more problematic. This highlights the need to understand the nutrient intake of NZ infants by ethnicity, and the nutrient density of CFs to help better support improving the choices that parents make. To the best of the authors' knowledge this is the first study of its kind in NZ specifically to assess nutrient intake from, and density of, CFs independently of breast or formula milk. Therefore, the purpose of the present study was to investigate and describe NZ infants' nutrient intake from, and density of, CFs stratified by ethnicity.

## Methods

Study design and data collection methods are described in brief in the following section, with further details reported elsewhere (15). This observational study was registered with the Australian New Zealand Clinical Trials Registry, ACTRN12620000459921. Ethical approval was granted by the Health and Disability Ethics Committees New Zealand (19/STH/151) and was funded in May 2019 by the Health Research Council (HRC) of New Zealand (grant 19/172). Data collection ran from July 2020 to February 2022. Data collection was paused during government-mandated COVID-19-related lockdowns, which occurred for two and a half weeks in August 2020, one week in February 2021, and four and a half months in August 2021. All appropriate hygiene measures were taken to minimise risk.

### Study Design

First Foods New Zealand (FFNZ) is an observational cross-sectional study of 625 infants aged 7.0 – 10.0 months in NZ. The analysis aims to evaluate and compare nutrient intake from complementary food for Māori, Pasifika, and non-Māori/Pasifika infants. The age range was chosen as it is close to the time where infants are introduced to CF, with the narrow age range allowing for the rapidly changing diet in infancy. The sample size was based on the wider study aim of comparing BMI z-

score and plasma ferritin concentration in infants following baby-led weaning and traditional spoon-feeding (15).

**Study Participants**

A total of 625 infants aged 7.0–10.0 months were recruited from two urban centres in NZ: Tāmaki-Makaurau (Auckland) and Ōtepoti (Dunedin). Participants were excluded if the adult respondent was unable to speak English or if the infant had been part of a nutrition intervention study. Participants were recruited through advertisements and word-of-mouth, whilst avoiding special interest groups, such as baby-led weaning groups. To ensure ethnic diversity, recruitment was targeted in suburbs with higher densities of Māori and Pasifika whānau (families). An information sheet was given to all participants and written informed consent was obtained.

**Data collection**

Demographic Data

The data for this paper were collected at an appointment conducted either in the participant's home or at the University research unit, according to the participant's preference. At the first visit demographic data were collected via questionnaire which encompassed ethnicity, household deprivation, (NZ Deprivation Index 2018 (16)), maternal education, maternal work status, and childcare use. Where possible, NZ census questions were used. Mesh block data were used to determine the level of deprivation based on the NZ Deprivation Index. Infant ethnicity was self-identified by the caregiver. Respondents were asked "what ethnic group does your child belong to?". Checkbox answer options were "Māori", "Pacific", "New Zealand European", or "other". If the participant selected the "other" option, they were prompted to enter free text to describe their ethnicity. For the purposes of this paper, if the participant did not select either Māori or Pacific, they were categorised as 'others' and this is how they are referred to forthwith.

**Dietary data**

Dietary data were obtained from two non-consecutive 24-hour recalls collected by trained interviewers at the first and second appointments. The recalls were collected on different days of the week, which captured between-day variation in dietary intake. The 24-hour recalls captured everything the infant ate and drank from midnight to midnight. The caregiver was asked to take photographs of the eating surface, including the offered food and beverages, both before and after each meal and snack the day before the 24-hour recall was conducted. The photographs were then used as prompts during the 24-hour recall interview.

**Statistical Analysis**

Usual intake was calculated from the 24-hour recalls using the multiple source method for estimating usual dietary intake of individuals (17). Dietary data were analysed using FoodWorks (version 10, Xyris Software), using the New Zealand Food Composition database FOODfiles 2018 Version 01. For commercial foods, recipes were created in FoodWorks using the ingredient list on the packet, and then modified to match the information on the nutrient information panel (18). This ensured that nutrients that did not appear on the nutrient information panel were captured in the analysis. Breast and formula milk were excluded from analysis for the purposes of this study. Quantile regression was used to determine nutrient intake, allowing adjustment for infant age. Nutrient density is reported as concentration of nutrient per 418 kJ (100 kcal) of energy. Ethnicity data is reported as total response ethnicity. Data was analysed using Stata statistical package (StataCorp).

Results

A total of 1424 infants were assessed for eligibility, with 625 infants-caregiver dyads enrolled in the study, all of whom completed the questionnaire. Data were analysed for all 625 infants. Infant ethnicity is reported as total response as many infants were identified as belonging to more than one ethnicity. A total of 131 infants were identified by their caregiver as Māori, and 82 as Pasifika. A further 516 identified as New Zealand European (NZE), 105 as Asian (Southeast Asian, East Asian, and South Asian), 62 as European (other than NZE), and 20 as MELAA (Middle Eastern, Latin American, and African), and these have been allocated into a single 'others' group as the primary focus of this paper is Māori and Pasifika infants. The mean (SD) infant age was 8.4 months for Māori, 8.5 months for Pasifika, and 8.4 months for 'other' infants. Demographic characteristics are presented in table 1.

**Table 1.** Demographic characteristics (n=625)

| | Total response ethnicity[e] | | |
|---|---|---|---|
| | Māori | Pasifika | Others |
| n | 131 | 82 | 450 |
| Infant age (mo), mean (SD) | 8.4 (0.8) | 8.5 (0.9) | 8.4 (0.8) |
| Infant sex, n (%) | | | |
| Female | 57 (43.5) | 35 (42.7) | 215 (47.8) |
| Total response infant ethnicity[a], n (% | | | |
| Māori | 131 (100) | 38 (46.3) | 0 |
| Pacific Island | 38 (29.0) | 82 (100) | 0 |
| New Zealand European | 99 (75.6) | 45 (54.9) | 372 (82.7) |
| Southeast Asian | 0 | 0 | 28 (6.2) |
| East Asian | 2 (1.5) | 3 (3.7) | 40 (8.9) |
| South Asian | 4 (3.1) | 3 (3.7) | 25 (5.6) |
| Other European | 6 (4.6) | 7 (8.5) | 49 (10.9) |
| Others | 2 (1.5) | 0 | 18 (4.0) |
| Term infant born, n (%) | | | |
| Pre-term (<37 weeks) | 13 (9.9) | 8 (9.8) | 29 (6.4) |
| Term (≥37 weeks) | 118 (91.1) | 73 (89.0) | 421 (93.6) |
| Respondent age (years), mean (SD) | 30.1 (5.5) | 30.3 (6.0) | 33.5 (4.3) |
| Highest level of education of adult respondent, n (%) | | | |
| School | 34 (26.0) | 23 (28.1) | 48 (10.7) |
| Polytech or similar | 48 (36.6) | 21 (25.6) | 69 (15.3) |
| University | 48 (36.6) | 37 (45.1) | 333 (74.0) |
| Adult respondent, n (%) | | | |
| Mother | 130 (99.2) | 80 (97.6) | 445 (98.9) |
| Maternal parity, n (%) | | | |
| Primiparous | 54 (41.2) | 39 (46.3) | 229 (50.9) |
| Respondent employment status, n (%) | | | |
| Employed full time | 17 (13.0) | 6 (7.3) | 52 (11.6) |
| Employed part time | 32 (24.4) | 14 (17.1) | 96 (21.3) |
| Other[b] | 82 (62.6) | 62 (75.6) | 302 (67.1) |
| Number of children living in household, n (%) | | | |
| One | 47 (35.9) | 28 (34.2) | 223 (49.6) |
| Two | 43 (32.8) | 28 (34.2) | 142 (31.6) |
| Three | 21 (16.0) | 11 (13.4) | 67 (14.9) |
| Four or more | 20 (15.3) | 15 (18.3) | 17 (3.8) |

| | Total response ethnicity[e] | | |
|---|---|---|---|
| | Māori | Pasifika | Others |
| Number of adults living in household, n (%) | | | |
| One | 11 (8.4) | 4 (4.9) | 13 (2.9) |
| Two | 92 (70.2) | 47 (57.3) | 399 (88.7) |
| Three | 14 (10.7) | 14 (17.1) | 19 (4.2) |
| Four or more | 14 (10.7) | 17 (20.7) | 19 (4.2) |
| Childcare used[c], n (%) | 27 (20.6) | 16 (19.5) | 76 (16.9) |
| Area-level Deprivation[d], n (%) | | | |
| 1–3 (Low) | 27 (20.6) | 14 (17.1) | 144 (32.0) |
| 4–7 | 52 (39.7) | 35 (42.7) | 214 (47.6) |
| 8–10 (High) | 52 (39.7) | 33 (40.2) | 92 (20.4) |

[a] Gestational age - data missing for one Pasifika participant; level of education - data missing for one Māori and one Pasifika participant; no. of children living in household – data missing for one non-Māori/Pasifika participant.

[b] "Other" value includes no employed, paid and un-paid parental leave.

[c] "Childcare" refers to formal early childhood education or home-based care, but not a nanny or childcare provided by an extended family member/friend.

[d] Deprivation level defined using the New Zealand Deprivation (NZDep) indices of 2018 (16). NZDep index is area based, uses NZ census variables and calculated for mesh blocks (geographic units containing ~100 people). "Low" refers to levels 1–3, "Medium" refers to levels 4–7, "High" refers to levels 8–10.

[e] Ethnicity is self-identified by the caregiver

Energy intake was stratified by age (7, 8, and 9 months) and sex, with the data reported in table 2. The median energy intake from CFs increased between seven and nine months of age for all groups and was higher overall for boys than girls. There were no significant differences between Māori and 'other' infants, or Pasifika and 'other' infants in energy intake.

**Table 2.** Energy intake from complementary food by age, sex, and ethnicity (n=625)

| Energy by age and sex | | Māori Median (25th, 75th percentile) | | Pasifika Median (25th, 75th percentile) | | Others Median (25th, 75th percentile) |
|---|---|---|---|---|---|---|
| Energy (girls) kJ/day | n | | n | | n | |
| 7 months | 24 | 984 (612, 1482) | 11 | 821 (713, 1112) | 80 | 870 (613, 1418) |
| 8 months | 22 | 956 (694, 1689) | 12 | 1048 (755, 1721) | 90 | 1096 (736, 1619) |
| 9 months | 11 | 1224 (858, 2118) | 12 | 1449 (852, 2167) | 45 | 1979 (1381, 2469) |
| Energy (boys) kJ/day | | | | | | |
| 7 months | 29 | 1057 (846, 1414) | 18 | 862 (523, 1366) | 88 | 931 (473, 1282) |
| 8 months | 25 | 1583 (912, 1878) | 14 | 1362 (986, 1878) | 84 | 1320 (859, 1852) |
| 9 months | 20 | 2021 (1420, 2608) | 15 | 2310 (1546, 3140) | 62 | 1993 (1382, 2532) |

The contribution of CF to the estimated energy recommendation (EER) is shown in table 3. Māori and Pasifika girls had a much lower energy intake from CFs than 'other' girls. In contrast, Pasifika boys obtained a higher proportion of energy from CF than both Māori and 'other' boys, who were similar.

**Table 3.** Contribution of CF to EER by age and sex

| Age | Māori | Pasifika | Other | Estimated energy requirements[a] |
|---|---|---|---|---|
| Girls | | | | |
| 7 months | 39.4% | 32.9% | 34.8% | 2500 |
| 8 months | 35.4% | 40.1% | 40.6% | 2700 |
| 9 months | 40.1% | 51.8% | 70.7% | 2800 |
| Boys | | | | |
| 7 months | 37.8% | 30.8% | 33.3% | 2800 |
| 8 months | 52.8% | 45.4% | 44.0% | 3000 |
| 9 months | 65.2% | 74.5% | 64.3% | 3100 |

[a] Recommendation for total estimated energy requirements from milk feeds and complementary foods. Recommendations from National Health and Medical Research Council 2006. *Nutrient Reference Values for Australia and New Zealand: including recommended dietary intakes*, National Health and Medical Research Council (19).

Daily macronutrient intake by ethnicity is reported in table 4. Māori infants had a significantly lower protein intake as a percentage of energy from complementary foods than 'other' infants (*P*=.01). Both Māori and Pasifika infants had a significantly lower total fat intake as a percentage of total energy compared to 'other' infants *(p*<.009 for Māori, P<.001 for Pasifika). The percentage of carbohydrate contributing to total energy from CFs was significantly higher for Māori and Pasifika infants *(p*=.001 and P<.001 respectively). For sugar intake in grams per day, Pasifika infants had a significantly higher intake than 'other' infants *(p*=.04). Sugar as a percentage of total energy intake was significantly higher for Māori infants than 'other' infants *(p*=.03).

**Table 4.** Macronutrient intake and total ethnicity

| Macronutrients | Māori Median ($25^{th}$, $75^{th}$ percentile) | Pasifika Median ($25^{th}$, $75^{th}$ percentile) | Others Median ($25^{th}$, $75^{th}$ percentile) |
|---|---|---|---|
| n | 131 | 82 | 450 |
| Protein (g/day) | 11.1 (7.0, 15.5) | 11.4 (7.2, 18.9) | 11.5 (6.2, 17.9) |
| Protein (% energy) | **14.6 (11.6, 17.4)[a]** | 14.8 (12.3, 17.6) | 15.7 (12.7, 18.5) |
| Fat (g) | 9.2 (5.9, 14.6) | 8.5 (5.4, 13.9) | 9.7 (4.6, 17.1) |
| Fat (% energy) | **27.3 (22.1, 34.6)[a]** | **24.9 (19.1, 30.2)[a]** | 30.7 (23.8, 37.9) |
| Saturated Fat (g) | 3.5 (1.9, 5.3) | *3.2 (1.8, 5.5)[b]* | 3.3 (1.4, 5.9) |
| Saturated fat (% energy) | 10.0 (7.1, 13.1) | 9.1 (6.7, 12.0) | 10.1 (7.1, 13.7) |
| Carbohydrate (g) | 39.5 (23.8, 57.9) | 42.5 (26.3, 66.2) | 35 (21.8, 53.2) |
| Carbohydrate (% energy) | **54.5 (46.8, 61.7)[a]** | **56.6 (48.3, 64.1)[a]** | 49.4 (42.0, 58.3) |
| Fibre (g) | 5.4 (3.6, 7.8) | 6.2 (3.9, 8.0) | 5.6 (3.5, 8.3) |
| Sugar (g) | 16.7 (9.7, 25.4) | **18.4 (12.6, 27.8)[a]** | 16.4 (9.1, 24.2) |
| Sugar (% energy) | **24.6 (17.0, 31.2)[a]** | *24.6 (19.7, 31.7)[b]* | 22.3 (16.5, 29.6) |

[a] Significantly different from 'others' adjusted for age (p<0.05)

[b] Non-significant trend

The contribution of macronutrients to the recommended intake of protein, fat, and carbohydrate is reported in table 5. The proportion of protein from CF increased by age time for all groups.

**Table 5.** Proportion of macronutrient recommendations met by complementary food.

| Macronutrients | Māori | Pasifika | Others | Daily recommendation (AI)[a] |
|---|---|---|---|---|
| Total protein | 79.4% | 81.4% | 82.1% | 14g |
| Total fat | 30.7% | 28.3% | 32.3% | 30g |
| Total carbohydrate | 41.6% | 47.2% | 36.8% | 95g |

[a] Recommendations from National Health and Medical Research Council 2006. *Nutrient Reference Values for Australia and New Zealand: including recommended dietary intakes*, National Health and Medical Research Council (19).

Usual daily micronutrient intake from CF is shown in table 6. None of the daily recommendations for minerals were met by CF alone. Median mineral intakes were not significantly different across groups. CF contributed 34% of the estimated average requirement (EAR) for iron for Māori, 39% for Pasifika, and 36% for others. For zinc, CF contributed 64% of the EAR for both Māori and 'others', and 68% for Pasifika. Calcium intake from CF did not meet the recommendation of 140 mg/day from CFs for any of the groups. Vitamin A intake exceeded the recommendation of 244 µg/day from CF for all three groups. CF contributed 78% of the EAR for vitamin $B_{12}$ for Māori, 76% for Pasifika, and 80% for others. For vitamin C, CF contributed 69% of the EAR for Māori, 86% for Pasifika, and 75% for 'others'. There were no significant differences in vitamin or mineral intake between groups.

**Table 6.** Selected micronutrient intake and total ethnicity

| Nutrient | Total Ethnicity Māori | Pasifika | Others | Daily Recommendation [a] |
|---|---|---|---|---|
| n | 131 | 82 | 450 | |
| Iron | 2.4 (1.4, 4.1) | 2.7 (1.8, 4.6) | 2.5 (1.4, 4.1) | 7 mg [b] |
| Zinc | 1.6 (1.0, 2.3) | 1.7 (1.1, 2.7) | 1.6 (0.9, 2.6) | 2.5 mg [b] |
| Calcium | 115 (65, 175) | 123 (71, 200) | 107 (58, 195) | 140 mg from CF [c] |
| Vitamin A | 257 (141, 390) | 295 (166, 416) | 262 (143, 417) | 244 µg from CF only |
| Vitamin $B_{12}$ | 0.39 (0.20, 0.76) | 0.38 (0.21, 0.76) | 0.4 (0.16, 0.82) | 0.5µg |
| Vitamin C | 20.8 (12.7, 30.8) | 25.9 (14.8, 34.9) | 22.4 (12.7, 37.8) | 30mg |

[a] Recommendation for total intake from milk feeds and complementary foods unless stated otherwise. Recommendations from National Health and Medical Research Council 2006. *Nutrient Reference Values for Australia and New Zealand: including recommended dietary intakes*, National Health and Medical Research Council (19).

[b] EAR (estimated average requirement)

[c] AI (adequate intake, used when an EAR value is not available)

The nutrient density (grams per 418 kJ / 100 kcal of CF) was calculated for iron, zinc, calcium, vitamin A, vitamin C, and vitamin $B_{12}$, and is reported in table 7. The target nutrient density from CF as defined by the World Health Organisation using estimated energy requirements (20). Iron, zinc, and calcium density of CF falls well below the desired target for all ethnic groups.

**Table 7.** Selected micronutrient density per 418 kJ of complementary diet of infants aged 7.0–10.0 months by total ethnicity

| Nutrient | Median density (25th, 75th percentile) | | | Mean desired nutrient density for infants aged 6–8 and 9–11 months[a] | |
|---|---|---|---|---|---|
| | Māori | Pasifika | Others | 6–8 months | 9–11 months |
| Iron *(mg/418 kJ)* | 0.8 (0.6, 1.1) | 0.9 (0.7, 1.1) | 0.8 (0.7, 1.1) | 4.5[b] | 3.0[b] |
| Zinc *(mg/418 kJ)* | 0.5 (0.4, 0.6) | 0.5 (0.5, 0.6) | 0.5 (0.5, 0.6) | 1.6 | 1.1 |
| Calcium *(mg/418 kJ)* | 37 (28, 50) | 40 (29, 50) | 38 (28, 50) | 105 | 74 |
| Vitamin A *(µg RE/418kJ)* | 107 (61, 159) | 123 (70, 173) | 110 (60, 174) | 31 | 30 |
| Vitamin C *(mg/418 kJ)* | 6.6 (5.0, 10.8) | 7.3 (5.2, 10.9) | 8.0 (5.7, 11.3) | 1.5 | 1.7 |
| Vitamin $B_{12}$ *(µg/418 kJ)* | 0.13 (0.1, 0.2) | 0.12 (0.1, 0.2) | 0.15 (0.1, 0.2) | 0.07[c] | 0.08[c] |

[a] Adapted from Dewey KG, Brown KH. Update on technical issues concerning complementary feeding of young children in developing countries and implications for intervention programs. Food and Nutrition Bulletin. 2003;24(1):5-28.
[b] Assuming moderate iron bioavailability
[c] Adapted from Allen LH. B vitamins: proposed fortification levels for complementary foods for young children. The Journal of nutrition. 2003;133(9):3000S-7S.

## Discussion

Little is currently known about the nutrient intake from CF of infants in NZ. The FFNZ study has quantified energy and key nutrient intake from CF as well as the nutrient density of CF for infants living in NZ and stratified by ethnicity. Energy, protein, fat, carbohydrate, iron, zinc, calcium, vitamin A, vitamin B12, and vitamin C were analysed and reported on.

### *Summary of findings*

Energy intake from CF increased overall with age for all groups and was higher for boys than girls. Protein as a percentage of energy intake from CF was significantly lower for Māori compared to

'others'. Fat as a percentage of energy intake from CF was significantly lower for both Māori and Pasifika than 'others', whereas carbohydrate as a percentage of energy intake from CF was significantly higher. Sugar intake in grams from CF was significantly higher for Pasifika when compared to 'others', and sugar as a percentage of energy intake from CF was significantly higher for Māori than 'others'. CF contributed a low proportion of iron to the recommended intake, and over half of total requirements for zinc, vitamin B12, and vitamin C. Vitamin A requirements from CF were met, but calcium was not. There were no significant differences in vitamin or mineral intake between groups. Nutrient density of CFs was below the critical nutrient density requirement for iron, zinc, and calcium. This adds a new understanding to the existing literature on the adequacy of the complementary diet of infants.

*Energy and macronutrients*

Energy intake from CF increased by age for both boys and girls in all three groups, which mirrors findings in two Canadian studies (21, 22). However, Māori and Pasifika girls had a much lower energy intake from CF at nine months of age compared to all boys and 'other' girls. This may be due to Māori and Pasifika girls acquiring a greater proportion of their nutrients from milk feeds than CF at nine months or having less energy-dense foods. Iguacel et al. (2019) found that breast-fed infants had a lower total intake of CF compared to formula-fed infants, even after adjusting for sex, parent education level, and total food intake (23). Despite their lower kilojoule intake, Pasifika girls met the minimum energy requirements from CF suggested by Dewey (2013) for breast-fed infants aged 9 months of 1254kJ per day (24), but Māori girls were negligibly lower at 1224kJ per day.

The significant differences found between macronutrient intake as a percentage of the total energy intake has interesting implications. ''other'' infants had a significantly higher protein intake than Māori, and a significantly higher fat intake than both Māori and Pasifika, who both had a significantly higher intake of total carbohydrate than 'others'. Additionally, sugar intake expressed as a percentage of total energy intake for Māori infants and sugar intake in grams for Pasifika infants were both significantly higher than that of 'other' infants. A possible reason for this is that higher proportion of Māori and Pasifika live in deprived areas than 'other' infants, and it is probable that protein-based foods are more expensive than carbohydrate-based foods (25). Rush et. al. (2015) demonstrated that NZ whānau living in highly deprived areas replaced fruit and vegetables with cheaper energy-dense and nutrient-poor breakfast cereals and/or convenience foods (26). Wang et al. (2009) found that so-called "healthier" foods (as defined by energy, sugar, saturated fat, protein, fibre, and fruit and vegetable content) were significantly more expensive than those higher in sugar

and saturated fat, and lower in protein, fibre, and fruit and vegetables in both rural and urban NZ (27), and although this is older data considering with the recent COVID-19 pandemic and associated cost of living crisis (28) it is feasible that such cost discrepancies still exist. The potential consequences of this are a poorer quality diet for Māori and Pasifika infants, and greater nutritional deficits that likely uphold the existing high nutrition-related morbidity rates, particularly for type 2 diabetes and cardiovascular disease.

Studies examining nutrient intake from CFs have been mostly conducted in developing countries (29-36), with two studies examining nutrient intake from CF in developed countries (22, 37). A recent NZ study validating a complementary food frequency questionnaire in 9–12-month-old infants gives some insight into nutrient intake but has a small sample group and is not ethnically diverse, as this was not needed for the primary aim of the study (38). Comparisons to these studies are made challenging due to different age range cut-offs used and different methods of reporting nutrient intake. Furthermore, accurately measuring or estimating breast milk intake is very difficult, so investigating the nutrient density of CF provides the opportunity to explore the nutritional adequacy of the infant diet when milk feeds are not quantified. This provides a clearer focus of where interventions and future research can be directed, as dietary manipulations are aimed at CF, rather than breast or formula feeding in this age group.

*Nutrient density of complementary food*

CF must bridge the gap between the infant's requirements and the amount obtained from breast or formula milk. Infants have a much higher nutrient requirement by weight than any other life stage yet consume relatively little CF compared to breast milk or formula. Therefore, CFs need to be very nutrient dense, as determined by the amount of a given nutrient per 418 kJ of energy, to meet the infant's needs. Solomons & Vossenaar (2013) refer to "critical nutrient density", where the nutrient density is sufficient to provide the recommended daily intake without exceeding the average energy requirements of the infant (39). Two particularly challenging nutrients for infants to obtain are iron and zinc, due mostly to the very low concentration found in breast milk. This is reflected in the results of this study, with CFs providing infants from all ethnic groups with approximately a fifth of the target nutrient density for iron and less than half of the target density for zinc. Calcium also fell short of the target density with CFs having about half of the recommended amount. The poor nutrient density of CF consumed in this cohort suggests that many infants being weaned onto solids may be at increased risk of iron, zinc, and calcium deficiency.

Nutrient density of CF has been measured in developing countries where malnutrition is associated with approximately half of the deaths of over 10 million young children and infants annually. Density of selected vitamins and minerals have been examined in rural South Africa, Guatemala, Malawi, and the USA, with an overall consensus that iron, zinc, and calcium are common problem nutrients. Iron density of CF ranged from 1.2 mg/1000kJ in rural South African to 2.6 mg/1000kJ in Guatemala, the latter higher to that found in the present study of 2.0 mg/1000kJ for Māori and 'other' infants, and 2.1 mg/1000kJ for Pasifika infants. Zinc and calcium densities of CF in the current study are also similar to those in Malawi, Guatemala, and rural South Africa. This is a surprising finding, as it might reasonably be expected that the average NZ infant diet would contain higher amounts of bioavailable iron and zinc than that of infants in developing countries. Additionally, there are mandatory fortification requirements for iron and vitamin c in cereal-based commercial infant food in NZ. The low iron, zinc, and calcium densities reported in this study coupled with the energy intakes from CF being adequate suggest that the infants would need to consume significantly more kilojoules per day to meet the EAR for these minerals, which is not feasible. The basis of the infant's complementary diet may need to be considered as part of the solution: many infants have a cereal-rich diet, with cereals being popular, easy to use, and inexpensive first foods. Cereals tend have low energy density but have bulking properties which promote satiety and may limit the infant's capacity for other nutrient-dense foods. Investigating from which food groups infants are obtaining their energy and nutrients would give a clearer understanding of these nutritional gaps. Infants in the present study consumed higher density CF for vitamin A, vitamin C, and vitamin $B_{12}$ than those in the former studies. Details are reported in supplementary table 1.

Another perspective on the consistent and world-wide problem with iron and zinc in particular is that the recommendation for these nutrients may be higher than necessary. Quinn (2014) proposed that gradually depleting iron stores in the first six months of life is an evolutionary adaption to reduce the availability of iron for pathogens and thus reduce infection risk (40). Furthermore, an unfortified diet that is high quality, as recommended by WHO (41), is unable to close the critical nutrient gap for iron from 6 to 11 months of age, and zinc at 6–8 months for breast-fed infants (42). It is also possible that iron (but not zinc) absorption is upregulated in infants with iron deficiency (43). Given our findings of much lower iron intake than the recommended amount combined this is an area that would benefit from further research.

*Strengths and limitations*

This is the first study to describe nutrient intake and density from CF in NZ. Strengths of the study are a large, ethnically diverse sample size with a broad range of socio-demographic characteristics reflecting that of the larger NZ population. However, a limitation is that this is not a nationally generalisable cohort with infants only recruited from two main urban centres, due to the logistics of the larger study (15). Another strength is the use of two multiple-pass 24-hour recalls on non-consecutive days. These were administered by trained researchers on different days of the week so between-day variation was captured. Dietary data were obtained for all 625 participants in the cohort. A further limitation in this study was the lack of breast milk data. Although breast milk data was collected it was not able to be analysed prior to the submission of this thesis. The breast milk data will be included in the published version of this manuscript.

Conclusion

This study demonstrates a novel approach using nutrient density to understanding and assessing the nutrient adequacy and critical nutrient gaps in the infant's complementary diet. All ethnic groups had low iron, zinc, and calcium intake from CF and the nutrient density of CF was insufficient to close the nutrient gap for these minerals. There were no significant differences between ethnicities for micronutrient intake or density, however Māori had significantly less protein and fat intake than 'others', and significantly higher carbohydrate and sugar as a percentage of total energy intake. Similarly, Pasifika infants also had a significantly lower fat intake as a percentage of total energy, and higher carbohydrate and sugar than 'other' infants. Nutrient density of the CFs was shown to be inadequate for iron, zinc, and calcium for all ethnic groups. To better understand where interventions and future research could be directed to address these critical nutrient gaps it is important to understand the food groups that supply nutrients for infants. Iron availability and absorption in particular is greatly influenced by many factors, and given the low density of iron, zinc, and calcium in the CF diet of this cohort, understanding the overall make-up of the infant's diet may help guide interventions to help prevent the development of nutritional deficiencies and related health outcomes.

References

1. World Health Organization and UNICEF. Global strategy for infant and young child feeding: World Health Organization; 2003.
2. Gerritsen S, Mackay S, Jackson A. Healthy Eating Guidelines for New Zealand Babies and Toddlers (0-2 years old). 2021. Report No.: 1991007388.
3. Koletzko B, Brands B, Grote V, Kirchberg FF, Prell C, Rzehak P, et al. Long-term health impact of early nutrition: the power of programming. Annals of Nutrition and Metabolism. 2017;70(3):161-9.
4. Koletzko B, Brands B, Chourdakis M, Cramer S, Grote V, Hellmuth C, et al. The Power of Programming and the EarlyNutrition project: opportunities for health promotion by nutrition during the first thousand days of life and beyond. Annals of Nutrition and Metabolism. 2014;64(3-4):187-96.
5. Lozoff B, Georgieff MK, editors. Iron deficiency and brain development. Seminars in Pediatric Neurology; 2006: Elsevier.
6. Vossenaar M, Solomons NW, Monterrosa E, van Zutphen KG. Nutrient density as a dimension of dietary quality: Sight Life; 2018. 172-6 p.
7. Fidler Mis N, Braegger C, Bronsky J, Campoy C, Domellöf M, Embleton ND, et al. Sugar in infants, children and adolescents: a position paper of the European society for paediatric gastroenterology, hepatology and nutrition committee on nutrition. Journal of Pediatric Gastroenterology and Nutrition. 2017;65(6):681-96.
8. Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH, et al. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. Circulation. 2009;120(11):1011-20.
9. Slavin J. Beverages and body weight: challenges in the evidence-based review process of the Carbohydrate Subcommittee from the 2010 Dietary Guidelines Advisory Committee. Nutrition Reviews. 2012;70(suppl_2):S111-S20.
10. Beck K, Jones B, Ullah I, McNaughton S, Haslett S, Stonehouse W. Associations between dietary patterns, socio-demographic factors and anthropometric measurements in adult New Zealanders: an analysis of data from the 2008/09 New Zealand Adult Nutrition Survey. European Journal of Nutrition. 2018;57(4):1421-33.
11. Shackleton N, Broadbent JM, Thornley S, Milne BJ, Crengle S, Exeter DJ. Inequalities in dental caries experience among 4-year-old New Zealand children. Community Dentistry and Oral Epidemiology. 2018;46(3):288-96.
12. Schluter PJ, Lee M. Water fluoridation and ethnic inequities in dental caries profiles of New Zealand children aged 5 and 12–13 years: analysis of national cross-sectional registry databases for the decade 2004–2013. BMC Oral Health. 2016;16(1):1-10.
13. Erickson LW, Taylor RW, Haszard JJ, Fleming EA, Daniels L, Morison BJ, et al. Impact of a Modified Version of Baby-Led Weaning on Infant Food and Nutrient Intakes: The BLISS Randomized Controlled Trial. Nutrients. 2018;10(6).
14. Gontijo de Castro T, Gerritsen S, Wall CR, Grant C, Teixeira JA, Marchioni DM, et al. Infant Feeding in New Zealand: Adherence to Food and Nutrition Guidelines among the Growing Up in New Zealand Cohort. Ministry of Social Development; 2018. Report No.: 1988541484.
15. Taylor RW, Conlon CA, Beck KL, von Hurst PR, Te Morenga LA, Daniels L, et al. Nutritional Implications of Baby-Led Weaning and Baby Food Pouches as Novel Methods of Infant Feeding: Protocol for an Observational Study. JMIR Research Protocols. 2021;10(4):e29048.
16. Atkinson J, Salmond C, Crampton P. NZDep2018 Index of Deprivation, Interim Research Report. Wellington: University of Otago; 2019.
17. Harttig U, Haubrock J, Knüppel S, Boeing H. The MSM program: web-based statistics package for estimating usual dietary intake using the Multiple Source Method. European Journal of Clinical Nutrition. 2011;65(1):S87-S91.

18. Katiforis I, Fleming EA, Haszard JJ, Hape-Cramond T, Taylor RW, Heath A-LM. Energy, Sugars, Iron, and Vitamin B12 Content of Commercial Infant Food Pouches and Other Commercial Infant Foods on the New Zealand Market. Nutrients. 2021;13(2):657.
19. National Health and Medical Research Council. Nutrient reference values for Australia and New Zealand: including recommended dietary intakes. Version 1.1 updated March 2017 ed: National Health and Medical Research Council; 2006.
20. Dewey KG, Brown KH. Update on technical issues concerning complementary feeding of young children in developing countries and implications for intervention programs. Food and Nutrition Bulletin. 2003;24(1):5-28.
21. James K F, Corinne A I, Rhona H, Angela M. Complementary food consumption of Canadian infants. The Open Nutrition Journal. 2009;3(1).
22. Friel JK, Hanning RM, Isaak CA, Prowse D, Miller AC. Canadian infants' nutrient intakes from complementary foods during the first year of life. BMC Pediatrics. 2010;10(1):1-11.
23. Iguacel I, Pardo LM, Pérez MJC, Aznar LAM, Villagrasa MPS, Rodríguez-Palmero M, et al. Feeding patterns and growth trajectories in breast-fed and formula-fed infants during the introduction of complementary food. Nutrición Hospitalaria: Organo oficial de la Sociedad Española de Nutrición Parenteral y Enteral. 2019;36(4):777-85.
24. Dewey KG. The challenge of meeting nutrient needs of infants and young children during the period of complementary feeding: an evolutionary perspective. The Journal of Nutrition. 2013;143(12):2050-4.
25. Brooks RC, Simpson S, Raubenheimer D. The price of protein: combining evolutionary and economic analysis to understand excessive energy consumption. Obesity Reviews. 2010;11(12):887-94.
26. Rush E, Savila Fa, Jalili-Moghaddam S, Amoah I. Vegetables: New Zealand children are not eating enough. Frontiers in Nutrition. 2019;5:134.
27. Wang J, Williams M, Rush E, Crook N, Forouhi NG, Simmons D. Mapping the availability and accessibility of healthy food in rural and urban New Zealand–Te Wai o Rona: Diabetes Prevention Strategy. Public Health Nutrition. 2010;13(7):1049-55.
28. Statistics New Zealand. Living costs increase for all household groups Wellington, New Zealand: Statistics New Zealand; 2023 [4/2/2023]. Available from: https://www.stats.govt.nz/news/living-costs-increase-for-all-household-groups.
29. Owino V, Amadi B, Sinkala M, Filteau S, Tomkins A. Complementary feeding practices and nutrient intake from habitual complementary foods of infants and children aged 6-18 months old in Lusaka, Zambia. African Journal of Food, Agriculture, Nutrition and Development. 2008;8(1):28-47.
30. Campos R, Hernandez L, Soto-Mendez MJ, Vossenaar M, Solomons NW. Contribution of complementary food nutrients to estimated total nutrient intakes for rural Guatemalan infants in the second semester of life. Asia Pacific Journal of Clinical Nutrition. 2010;19(4):481-90.
31. Gibson R, Ferguson E, Lehrfeld J. Complementary foods for infant feeding in developing countries: their nutrient adequacy and improvement. European Journal of Clinical Nutrition. 1998;52(10):764-70.
32. Hotz C, Gibson R. Complementary feeding practices and dietary intakes from complementary foods amongst weanlings in rural Malawi. European Journal of Clinical Nutrition. 2001;55(10):841-9.
33. Abeshu MA, Lelisa A, Geleta B. Complementary feeding: review of recommendations, feeding practices, and adequacy of homemade complementary food preparations in developing countries–lessons from Ethiopia. Frontiers in Nutrition. 2016;3:41.
34. Islam MM, Khatun M, Peerson JM, Ahmed T, Mollah MAH, Dewey KG, et al. Effects of energy density and feeding frequency of complementary foods on total daily energy intakes and consumption of breast milk by healthy breastfed Bangladeshi children. The American Journal of Clinical Nutrition. 2008;88(1):84-94.

35. Lutter CK, Rivera JA. Nutritional status of infants and young children and characteristics of their diets. The Journal of Nutrition. 2003;133(9):2941S-9S.
36. Faber M. Complementary foods consumed by 6–12-month-old rural infants in South Africa are inadequate in micronutrients. Public Health Nutrition. 2005;8(4):373-81.
37. Dewey KG. Nutrition, growth, and complementary feeding of the brestfed infant. Pediatric Clinics of North America. 2001;48(1):87-104.
38. Judd AL, Beck KL, McKinlay C, Jackson A, Conlon CA. Validation of a Complementary Food Frequency Questionnaire to assess infant nutrient intake. Maternal & Child Nutrition. 2020;16(1):e12879.
39. Solomons N, Vossenaar M. Nutrient density in complementary feeding of infants and toddlers. European Journal of Clinical Nutrition. 2013;67(5):501-6.
40. Quinn EA. Too much of a good thing: evolutionary perspectives on infant formula fortification in the United States and its effects on infant health. American Journal of Human Biology. 2014;26(1):10-7.
41. Dewey K. Guiding principles for complementary feeding of the breastfed child2003.
42. Samuel A, Osendarp SJ, Ferguson E, Borgonjen K, Alvarado BM, Neufeld LM, et al. Identifying dietary strategies to improve nutrient adequacy among Ethiopian infants and young children using linear modelling. Nutrients. 2019;11(6):1416.
43. Russell RM, Beard JL, Cousins RJ, Dunn JT, Ferland G, Hambidge KM, et al. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC, USA: National Academies Press; 2001.

**Supplementary table I.** Nutrient densities per 418 kJ (100 kcal) of complementary foods consumed by infants in Malawi, Guatemala, and rural South Africa, compared to the present study

| Nutrient | FFNZ Study, 2022: median density (25th, 75th percentile) | | | Malawi, 2001[a] | Guatemala, 2010[b] | Rural South Africa, 2004[c] |
|---|---|---|---|---|---|---|
| Age (months) | 7.0 – 10.0 | | | 6 – 8 | 7 – 9 | 6 – 9 |
| | Māori | Pasifika | Others | | | |
| Iron (mg/418 kJ) | 0.8 (0.6, 1.1) | 0.9 (0.7, 1.1) | 0.8 (0.7, 1.1) | 0.8 | 1.0 | 0.5 |
| Zinc (mg/418 kJ) | 0.5 (0.4, 0.6) | 0.5 (0.5, 0.6) | 0.5 (0.5, 0.6) | 0.5 | 0.5 | 0.3 |
| Calcium (mg/418 kJ) | 37 (28, 50) | 40 (29, 50) | 38 (28, 50) | 5.0 | 42 | 22 |
| Vitamin A (µg RE/418kJ) | 107 (61, 159) | 123 (70, 173) | 110 (60, 174) | 2.0 | 53.5 | 29 |
| Vitamin C (mg/418 kJ) | 6.6 (5.0, 10.8) | 7.3 (5.2, 10.9) | 8.0 (5.7, 11.3) | - | 3.8 | 3.5 |
| Vitamin $B_{12}$ (µg/418 kJ) | 0.13 (0.1, 0.2) | 0.12 (0.1, 0.2) | 0.15 (0.1, 0.2) | - | - | 0.06 |

[a] Hotz C, Gibson R. Complementary feeding practices and dietary intakes from complementary foods amongst weanlings in rural Malawi. European Journal of Clinical Nutrition. 2001;55(10):841-9.

[b] Campos R, Hernandez L, Soto-Mendez MJ, Vossenaar M, Solomons NW. Contribution of complementary food nutrients to estimated total nutrient intakes for rural Guatemalan infants in the second semester of life. Asia Pacific Journal of Clinical Nutrition. 2010;19(4):481-90.

[c] Faber M. Complementary foods consumed by 6–12-month-old rural infants in South Africa are inadequate in micronutrients

# Chapter 6 | Upoko Tuaono

## Iron status of Māori, Pasifika, and other infants living in Aotearoa New Zealand

*This chapter reports the findings of the First Foods New Zealand study pertaining to iron status by ethnicity.*

*This report is presented in manuscript format and will be submitted to a peer-reviewed journal for publication in the future. Online statement of contribution can be reviewed in Appendix J.*

Abstract

**Background:** Iron status is one of the most prevalent problems facing infants worldwide, in both developing and developed countries. A complex interplay of both dietary and non-dietary factors affects iron absorption, and subsequently iron status.

**Aim:** The aim of this study was to describe iron status, and examine factors related to differences in iron status amongst Māori, Pasifika, and 'other' infants living in NZ.

**Methods:** Data were collected from 360 infants aged 7 to 10.0 months living in two main urban centres in NZ between July 2020 and February 2022. Haemoglobin, plasma ferritin, soluble transferrin receptor (sTfR), C-Reactive protein, and alpha-1-acid-glycoprotein were obtained from a venous blood sample. Inflammation was adjusted for using the Biomarkers Reflecting Inflammation and Nutritional Determinants of Anaemia (BRINDA) method. Body iron concentration (mg/kg body weight) was calculated using the ratio of sTfR and ferritin

**Results:** A total of 96.4% of Pasifika infants were iron sufficient, defined as body iron ≥0 mg/kg body weight and haemoglobin (Hb) ≥110 g/L, compared to 82.5% of Māori and 77.5% of 'other' infants. 'Other' infants had the highest prevalence of iron deficiency overall, with 6% categorised with iron-deficiency anaemia (IDA) (body iron <0 mg/kg, haemoglobin <110 g/L), 8% with early functional iron deficiency (body iron <0 mg/kg, haemoglobin ≥110 g/L), and 8.4% with iron depletion (ferritin <15 µg/L, body iron ≥0 mg/kg). For Māori infants, 4.7% had IDA and early functional iron deficiency respectively, and 8% were iron depleted. One (3.6%) Pasifika infant was iron depleted, and the remainder were iron sufficient. Ferritin and body iron concentration showed a non-significant higher trend for Pasifika compared to 'other' infants. Mediation analysis suggested that the difference in body iron concentration between Pasifika and 'others' was partially explained by the frequency of consumption of baby food pouches.

**Conclusion:** These findings give an up-to-date and robust understanding of the iron status of infants by ethnicity, highlighting an unexpected finding that non-Māori and non-Pasifika infants are at higher risk of poor iron status in NZ.

Introduction

Over two billion people worldwide have iron deficiency, and half of these have related anaemia (1). The majority live in low–middle income countries (2), with South Asian and African countries having the highest prevalence (3). As many as 40% of very young children in developing countries are iron deficient with and without anaemia (4), but the figure is much lower in developed countries, with 2–25% of infants in European countries having iron deficiency (5). In Aotearoa New Zealand (NZ) studies suggest 2.8 – 22% of infants aged 6 – 12 months have at least some degree of iron deficiency (6-8). Historically research has shown Māori and Pasifika infants to have poor iron status (9-13), with 3 – 65% having iron deficiency (ID) with or without anaemia. However, these studies frequently failed to control for infection, used varying indices and cut-offs outside of the generally accepted range, and some used data from hospitalised infants. A well-designed study in 2007 stratified by ethnicity and found that Māori, Pasifika, and 'other' ethnicities had a greater burden of iron deficiency than European infants (14).

Iron deficiency in infancy is associated with immune deficiencies, cognitive impairment, and delayed motor skill development, and the impact on cognition and intelligence quotient (IQ) may be lifelong. Importantly, these effects can occur when there is ID without anaemia (15, 16), and may be irreversible even with iron repletion (16, 17). Lower scores on tests of mental and motor functioning were shown at age five (18), and follow-up studies in school age and adolescents report ongoing lower scores even with iron supplementation in infancy (19-23).

Defining iron status can be complex due to varying biochemical indices, with different indices used in conjunction with each other depending on the individual's needs. Iron status can be thought of as a continuum, from replete through to iron deficiency anaemia (IDA) with varying cut-offs used worldwide to define ID and IDA. Current World Health Organisation (WHO) cut-offs drawn from expert consultations in 1987 and 2004 have determined that a ferritin value of less than 10–12 ug/L is suggestive of depleted iron stores for people of all life stages in the absence of inflammation (24). Other useful assays include soluble transferrin receptor (sTfR), serum iron, serum transferrin concentration, and transferrin saturation which is determined by calculating the ratio of serum iron to transferrin. Haemoglobin (Hb) concentration is required to diagnose anaemia, and the WHO recommends a cut-off for children aged six months to four years of less than 110 g/L. The European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN) suggest <105 g/L however (25). Regardless of whether a cut-off of 110 or 105 g/L is used, infants at the lower end are at risk of developing anaemia. Body iron (mg/kg body weight), calculated using the ratio of sTfR and

ferritin, is increasingly used to measure iron status as inflammation has less of an effect and it provides a continuous measure of iron status in populations (26).

Infant iron status is determined by dietary and non-dietary factors, beginning with maternal iron stores during pregnancy and gestational age at birth, extending through to post-natal iron losses and requirements, inflammation, hepcidin control, genetics, dietary iron, and dietary modifiers (27). Specific dietary factors may contribute to infant iron status. Consumption of iron-fortified baby rice and red meat at around six months of age are important due to term infants being born with enough iron stores for approximately six months (28), with breast versus formula feeding possibly making a difference (29-34). Varying combinations and degrees of severity of these factors uniquely influence infants' iron status.

The current prevalence of ID and IDA within different ethnic groups in NZ is not known. With the inequitable health outcomes experienced by Māori and Pasifika and the historical indication of high rates of ID with or without anaemia (ID(A)) it is important to gain a better understanding of the frequency of ID(A) in all ethnic groups and contributing factors to help better direct healthcare interventions. Therefore, the present study aimed to measure the iron status and intra-ethnic determinants of iron status of Māori, Pasifika, and other infants in NZ.

## Methods

Study design and data collection methods are described in brief, with further details reported elsewhere (35). This study was registered with the Australian New Zealand Clinical Trials Registry, ACTRN12620000459921. Ethical approval was granted by the Health and Disability Ethics Committees New Zealand (19/STH/151) and funding obtained from the Health Research Council (HRC) of New Zealand (grant 19/172). Data collection ran from July 2020 to February 2022. Data collection was paused during government-mandated COVID-19-related lockdowns, which occurred for two and a half weeks in August 2020, one week in February 2021, and four and a half months in August 2021 in Auckland, and for three weeks in August 2021 in Dunedin. All appropriate hygiene measures were taken to minimise risk during data collection.

**Study Participants**

Infants aged 7.0 – 10.0 months were recruited from two urban centres in NZ: Tāmaki-Makaurau (Auckland) and Ōtepoti (Dunedin). Participants were excluded if the caregiver was unable to speak English or if the infant had recently been part of a nutrition intervention study that may have affected their usual intake. Participants were recruited from a range of ethnic groups and levels of deprivation using advertisements and word-of-mouth, whilst avoiding special interest groups, such as baby-led weaning groups. To ensure ethnic diversity, recruitment was targeted in suburbs with higher proportions of Māori and Pasifika whānau. An information sheet was given to all caregivers and written informed consent was obtained from the primary caregiver of 625 infants.

**Data collection**

**Demographic data**

Demographic data was collected via questionnaire which encompassed age, ethnicity, sex, household deprivation, (NZ Deprivation Index 2018 (36)), caregiver education, work status, parity, childcare use, the number of children and adults living in the household, and whether the infant was a premature or full-term at birth. Mesh block data was used to determine the level of deprivation based on the NZ Deprivation Index. Infant ethnicity was self-determined by the caregiver. Respondents were asked "what ethnic group does your child belong to?". Checkbox answer options mirrored that of the 2018 New Zealand census, and participants were able to select from the following list: "NZ European", "Māori", "Samoan", "Cook Island Māori", "Tongan", "Niuean", "Chinese", "Indian", and "other, e.g., Dutch, Japanese, Tokelauan". If the participant selected the "other" option, they were prompted to enter free text to describe their ethnicity. Participants were grouped by total response for both Māori and Pasifika. For the purposes of this analysis, remaining participants were grouped together as a singular 'non-Māori, non-Pasifika' group, and will be referred to as 'other(s)' forthwith.

**Feeding Practices Data**

Feeding practices were self-reported by the caregiver via a questionnaire, with a researcher present to clarify any questions. Caregivers were asked about the timing of the introduction of complementary food and current pouch use. Infants were designated as 'frequent pouch users' if they consumed five or more pouches per week in the past month. Participants were also asked if their infant had iron-fortified baby rice or red meat at around six months of age. Milk feeding data was also collected via a questionnaire. Participants were asked if their infant was still being breast-

fed, how old the infant was when they stopped being breastfed (if applicable), if the infant had ever had infant formula, how old the infant was when they first had infant formula, and if they were still drinking infant formula.

**Biochemical assessment**

Local anaesthetic (Ametop Gel, Smith & Nephew, Canada) was applied to both of the infants' arms inside the elbow, and an occlusive dressing was applied. The dressing was removed and excess gel wiped off with a tissue 30–45 minutes after application, with the blood sample drawn no more than 4–6 hours after the Ametop was applied. Blood samples were collected by venepuncture via an EDTA vacutainer. Full blood count was measured on the day of collection by Southern Community Labs (SCL), Dunedin, and Labtests, Auckland on a Sysmex-XN20 Module® automated analyser (Sysmex, Kobe, Japan). The ferritin assay was conducted on a E602-Cobas 8000 analyser (Roche Diagnostics, USA), and was measured within seven days of collection. Remaining plasma samples were stored at -70° Celsius until consequent analysis of sTfR, C-Reactive Protein (CRP), and $\alpha_1$-acid glycoprotein (AGP) on a Hitachi Cobas C311 analyser (Roche Diagnostics, Mannheim, Germany, and Hitachi High-Technologies Corp, Tokyo, Japan) machine at the Department of Human Nutrition (University of Otago, Dunedin, NZ). Abnormal results were reviewed by the study paediatrician and a letter was sent to both the caregiver and their general practitioner as specified by the caregiver on the consent form. If no abnormal results were detected a letter was sent to the caregiver providing the infant's haemoglobin (g/L), plasma ferritin (μg/L) and haematocrit. The letter included a brief lay-language explanation of each of these indices. Ferritin was adjusted for inflammation using the BRINDA method (37), and sTfR was adjusted to be equivalent with the Flowers assay as follows (26): 1.5 x Roche sTfR + 0.35 mg/L. Body iron was calculated as follows: -[*log*10 (sTfR x 1000/ferritin) – 2.8229]/0.1207.

Cut-offs used to determine iron status are described in table 1. Infants who had low Hb in the absence of iron deficiency were included in the "iron sufficient" group.

**Table 1.** Iron status categories and haematological cut-offs

| Iron status category | Cut-off value |
|---|---|
| Iron sufficient | Plasma ferritin ≥15 μg/L[a], body iron ≥0 mg/kg |
| Iron depleted | Plasma ferritin <15 μg/L[a], body iron ≥0 mg/kg |
| Early functional iron deficiency | Body iron <0 mg/kg[b], haemoglobin ≥110 g/L[c] |
| Iron deficiency anaemia | Body iron <0 mg/kg[b], haemoglobin <110 g/L[c] |

[a] Southern Community Laboratories Ltd. (2012)
[b] Cook et al. (2003)
[c] World Health Organization (2001)

**Statistical analysis**

The biochemical indices were described using mean, geometric mean, and median. Potential mediators were identified by assessing statistical significance in differences between ethnicities in early feeding practices. Multivariable linear regression analyses were performed to determine the suitability of using mediation analysis to explain differences in body iron between ethnic groups. This was done by first testing the relationship between ethnic group and body iron (mg/kg body weight), then testing the relationship between ethnicity and each mediating factor. Potential mediating factors tested were the introduction of complementary food at <5 months of age, consumption of red meat and / or iron-fortified baby rice at six months of age, and use of baby food pouches currently. The factors that were found to be significantly different between ethnicities were then tested for the relationship with body iron. Where all relationships were significant ($p$<0.05) for an ethnic group, mediation analysis was performed. All data was analysed using Stata V.17 (StataCorp) statistical software.

Results

In total 1424 infants were assessed for eligibility. Of these, 625 were enrolled in the study and blood samples were collected from 365 infants. Of the 260 infants that did not have a blood sample taken, 116 (45%) had an unsuccessful blood draw, 92 (35%) caregivers declined the blood test, 32 (12%) were unable to attend the blood test appointment due to illnesses, and 20 (8%) were unable to attend due to government-mandated COVID-19 lockdowns. Within those who had a blood sample taken, 63 infants were Māori, 28 Pasifika, and the remaining 288 infants were European, Asian (east Asian, southeast Asian, and south Asian), or Middle Eastern, Latin American, and African (MELAA). Ethnicity is reported as total response for Māori and Pasifika. Demographic characteristics are

reported in table 2. The ethnicity portion of this table demonstrates that many participants identified as two or more ethnicities.

**Table 2.** Demographic characteristics by ethnicity (n=321)

| | Māori | Pasifika | Others[a] |
|---|---|---|---|
| n | 63 | 28 | 288 |
| Age, mean (SD) months | 8.3 (0.8) | 8.5 (0.8) | 8.3 (0.8) |
| Sex, n (%) male | 37 (58.7) | 14 (50.0) | 160 (55.6) |
| Ethnicity, n (%) | | | |
| Māori | 63 (100) | 14 (50.0) | 0 |
| Pacific | 14 (22.2) | 28 (100) | 0 |
| South Asian | 2 (3.2) | 1 (3.6) | 9 (3.1) |
| South-East Asian | 0 | 0 | 14 (4.9) |
| East Asian | 2 (3.2) | 1 (3.6) | 17 (5.9) |
| NZ European | 48 (76.2) | 20 (71.4) | 250 (86.8) |
| Other European | 5 (7.9) | 1 (3.6) | 32 (11.1) |
| Others | 0 | 0 | 12 (4.2) |
| Preterm birth, n (%)[b] | 3 (4.8) | 1 (3.6) | 21 (7.3) |
| Respondent age, mean (SD) years | 30.7 (5.8) | 31.9 (6.9) | 33.4 (4.2) |
| Primiparous, n (%) | 26 (41.3) | 15 (53.6) | 145 (50.4) |
| Respondent employment status, n (%) | | | |
| Full-time | 9 (14.3) | 2 (7.1) | 36 (12.5) |
| Part-time | 18 (28.6) | 4 (14.3) | 62 (21.5) |
| Other[c] | 36 (57.1) | 22 (78.6) | 190 (66.0) |
| Childcare[d], n (%) | 16 (25.4) | 7 (25.0) | 49 (17.0) |
| Area-level deprivation[e], n (%) | | | |
| 1–3 (Low) | 16 (25.4) | 6 (21.4) | 91 (32.6) |
| 4–7 | 25 (39.7) | 12 (42.9) | 136 (47.2) |
| 8–10 (High) | 22 (34.9) | 10 (35.7) | 61 (21.2) |

[a]'Others' was defined as infants who did not identify as Māori or Pasifika

[b]Gestational age - data missing for one Pasifika participant

[c]"Other" value includes not employed, paid and un-paid parental leave.

[d]"Childcare" refers to formal early childhood education or home-based care, but not a nanny or childcare provided by an extended family member/friend

[e]Deprivation level defined using the New Zealand Deprivation (NZDep) indices of 2018 (36). NZDep index is area based, uses NZ census variables, and calculated for mesh blocks (geographic units containing ~100 people). "Low" refers to levels 1–3, "Medium" refers to levels 4–7, "High" refers to levels 8–10.

Haematological indices are reported in table 3. There was a trend for Pasifika infants to have higher ferritin and body iron concentrations than 'others' ($p$=0.08 and 0.05 respectively).

**Table 3.** Iron and inflammatory biomarkers by total ethnicity

| | Māori | | Pasifika | | Others[a] | |
|---|---|---|---|---|---|---|
| | *n*[b] | | *n* | | *n*[b] | |
| Haemoglobin, mean (SD) g/L | 65 | 115.8 (9.3) | 28 | 117.0 (9.7) | 295 | 115.3 (8.9) |
| Plasma ferritin[c], geometric mean (95% CI) µg/L | 63 | 25.3 (21.2, 30.2) | 28 | 29.5 (24.3, 35.9)[d] | 288 | 22.5 (20.5, 24.7) |
| Soluble transferrin receptor[c], mean (SD) mg/L | 63 | 4.3 (1.4) | 28 | 4.1 (1.5) | 289 | 4.3 (1.2) |
| Body iron[e], mean (SD) mg/kg | 63 | 3.5 (3.1) | 28 | 4.3 (2.3)[d] | 288 | 3.0 (3.3) |

[a] 'Others' was defined as all those who did not identify as Māori or Pasifika
[b] Analysis of all biochemical indices were unable to be performed on all infants due to insufficient blood draws for some, therefore there are differing numbers in some categories
[c] Plasma ferritin and soluble transferrin receptor adjusted for inflammation (CRP & AGP) using the BRINDA method (38)
[d] Non-significant trend compared to 'others'
[e] Body iron calculation = -[log10(sTfRx1000/ferritin)-2.8229]/0.1207 (26)

Iron status by ethnicity is described in table 4 and shows that Pasifika infants had the lowest prevalence of any stage of iron deficiency compared to Māori and 'others'.

**Table 4.** Iron status by total response ethnicity

| | Māori (*n*=63) | Pasifika (*n*=28) | Others[a] (*n*=288) |
|---|---|---|---|
| Iron sufficient[b], n (%) | 52 (82.5) | 27 (96.4) | 219 (76.0) |
| Iron depleted[c], n (%) | 5 (8) | 1 (3.6) | 27 (9.4) |
| Early functional iron deficiency[d], n (%) | 3 (4.7) | 0 | 34 (11.8) |
| Iron deficient anaemia[e], n (%) | 3 (4.7) | 0 | 8 (2.8) |

[a] 'Others' was defined as all those who did not identify as Māori or Pasifika
[b] Defined as body iron ≥0 mg/kg, ferritin ≥15 µg/L
[c] Defined as body iron ≥0 mg/kg, ferritin <15 µg/L
[d] Defined as body iron <0 mg/kg, Hb ≥110 g/L
[e] Defined as body iron <0 mg/kg, Hb <110 g/L

Mediation factors for differences in body iron by ethnicity explained by differences in feeding practices for Pasifika compared to 'others' are reported in table 5. The relationship between body iron and Māori ethnicity was too small to satisfy the requirements for mediation. However, for Pasifika infants, frequent pouch use (defined as the infant having pouches at least five times per week) partially explains their higher body iron compared to 'other' infants. None of the other factors analysed were able to explain the difference in body iron. The prevalence of early feeding practices chosen by ethnicity are shown in supplementary table 1.

**Table 5.** Mediation of differences in body iron by ethnicity

| Potential mediators | Mean difference in body iron from 'others' | Mean difference in body iron from 'others' adjusted for potential mediator | % mediated |
|---|---|---|---|
| **Pasifika** | | | |
| Frequent pouch use | 1.24 (-0.01, 2.48) | 1.02 (-0.23, 2.26) | **17.7** |
| Early introduction to solids | 1.24 (-0.01, 2.48) | 1.22 (-0.03, 2.47) | 1.6 |
| Baby rice consumed around 6 months | 1.24 (-0.01, 2.48) | 1.26 (0.01, 2.51) | -1.6 |
| Red meat consumed around 6 months | 1.24 (-0.01, 2.48) | 1.31 (0.05, 2.56) | -5.6 |
| Currently having infant formula | 1.24 (-0.01, 2.48) | 1.18 (-0.03, 2.40) | 4.8 |

## Discussion

### *Summary of findings*

Blood samples were obtained from 365 infants in an ethnically diverse self-selected sample with the results stratified by ethnicity. Infants were categorised as having IDA, early functional ID, or ID using body iron stores (mg/kg) and Hb concentration (g/L). 'Other' infants had the highest proportion of ID with and without anaemia, followed by Māori. Pasifika infants had a low prevalence of ID, with just one infant having depleted iron stores only. The haematological indices mirrored these findings, with a trend for Pasifika infants to have a higher ferritin concentration compared to 'others', and a non-significantly higher Hb value compared to 'others' and Māori. Frequent pouch use partially explained the higher body iron in Pasifika infants compared to 'other' infants when potential mediation factors were examined, but not the consumption of iron-fortified baby rice or red meat at around six months of age, use of infant formula, or age of introduction to complementary food.

### *Biochemical indices and cut-offs*

Diagnosing ID and appropriately interpreting haematological indices in infants is challenging due to varying cut-offs and indices used worldwide. This is due in part to the lack of sufficiently validated markers and reference ranges for very young children and infants, with cut-offs extrapolated from older children being less than desirable due to physiological changes in red blood cell morphology during the huge phase of growth in the first year of life (39). The United States National Health and Nutrition Examination Surveys (NHANES) (26) and the World Health Organization (40) define low Hb as <110 g/L, a figure that has been extrapolated from older children. ESPGHAN use a lower cut-off of 105 g/L, however. Similarly, the ferritin threshold varies between studies, with the WHO advising that <12 μg/L is suggestive of low iron stores in children under 5 years of age, which is similar to that of ESPGHAN, who give a range of <10–12 μg/L. In NZ the Southern Community Laboratories have a higher cut-off of <15 μg/L for very young children, while Starship Children's Hospital (the largest paediatric tertiary hospital in NZ) use a cut-off of <10 μg/L (41). It has been suggested that increasing the ferritin cut-off in children to as high as 30 μg/L improves the diagnostic accuracy, with the sensitivity with a cut-off at 12 μg/L being 25% compared to 30 μg/L being 92% (42). Using a cut-off of 12 μg/L gave an implausibly low prevalence of iron depletion, so it was therefore decided in this study to use a cut-off of <15 μg/L.

### *Iron status*

The findings in this study are in stark contrast to previous studies which indicate that Māori and Pasifika infants tended to have higher rates of IDA and ID than other infants in NZ (9-13). Whilst

there are flaws in some older studies, such as the use of hospitalised populations (9, 11-13, 43, 44), not measuring inflammatory markers, and the use of inappropriate haematological cut-offs, there are a small number of studies that are robust and comparable in design to the present study. Grant et. al. (2007) defined ID as <10 µg/L for ferritin, and found that ID was present in 20% of the Māori infants in their cohort, 17% of Pasifika, 27% of 'other' infants, and just 7% of NZE infants (14). In comparison, the total rate of ID with and without anaemia in the present study was 21% of Māori infants, 3.6% of Pasifika, and 22% of 'others', a group comprised primarily of NZE infants. Wall et al., (2008), using the same cohort as the aforementioned study by Grant et al., (2007), found fewer associations between dietary factors and iron status in Māori and Pasifika compared to NZE infants (45), suggesting non-nutritive factors as possible causes for ID and IDA. A possible explanation for this is a historically higher rate of illness and infection in Māori and Pasifika infants, possibly exacerbated by genetic variations in the hepcidin production mediating the pathogenesis of ID caused by inflammation (46). Additionally, there is a relatively high proportion of α-thalassaemia amongst Māori and Pasifika people, at 16 – 20% of the population (47-50). This causes a microcytic hypochromic appearance of red blood cells which may be mistaken for anaemia, potentially explaining the higher proportion of infants in older studies where haemoglobin was the sole biochemical measure used to define IDA. One infant was identified as having probable α-thalassaemia in the present study and was included in the final analysis, as the definition of ID did not rely upon the appearance of the red blood cells.

*Factors mediating differences in iron status*

Frequent pouch use partially explained the higher body iron concentration in Pasifika infants compared to 'other' infants. This was unexpected due to the low iron content of pouches in NZ of just 0.3 mg/100g (51), and with meat-based pouches containing on average just 5 – 10% of meat. However, it is possible that if fruit pouches were commonly consumed the vitamin C may increase iron absorption, however this was not assessed in this study. The use of iron-fortified baby rice and / or red meat at six months of age, current infant formula use, and early introduction of complementary foods did not explain the differences in body iron. It is possible that a more complex interplay of dietary factors may partially explain the difference. Wall et al. (2008) found that Pasifika children obtained a higher proportion of iron from meat compared to NZE children (45), so it is possible that another factor that may help explain the difference is that Pasifika infants may have obtained their iron from more highly bioavailable sources such as red meat. It is also possible that the timing and quantity of calcium intake may further impact the bioavailability of dietary iron

intake, however these factors have not been evaluated in the present study due to the additional scope of work required to determine this.

*Strengths and limitations*

This is the first study since 2007 to comprehensively evaluate the iron status of NZ infants and stratify by ethnicity. A strength of this study is the diverse range of ethnicities in similar proportions to that of the wider population, however the wider study was not specifically designed to assess ethnic differences. The use of a venous blood sample, analysis and adjustment for two inflammatory markers, and subsequent use of body iron concentration as a measure of iron status gives a sensitive and reliable measure of iron status. Limitations were a high attrition rate due to a separate visit to a clinic for phlebotomy being required for Auckland participants, which may have been avoided if in-home samples were collected. This is particularly reflected in the Māori and Pasifika group, with 52% and 66% respectively not providing a blood sample. Moreover, it is possible that participants who have a higher level of health literacy are more likely to present for the required blood test and may be more motivated to seek out and implement advice for best practices of infant feeding. Therefore, this data should be interpreted with caution, and further studies investigating iron status by ethnicity that are generalisable to the population should be conducted. Another limitation was the lack of dietary and non-dietary factors relating to iron status that were not included in the analysis, particularly food sources of iron, prematurity, and blood loss.

## Conclusion

This study has provided a robust update on the iron status of infants living in NZ by ethnicity. Contrasting with previous studies, Pasifika infants had a very low rate of ID, and Māori infants had a slightly lower prevalence than 'others'. This has been an unexpected finding, with frequent pouch use being the only factor explaining the difference in body iron between Pasifika and 'other' infants. This is also a surprising finding given the low iron concentration in pouches, but if a high proportion of fruit pouches are used it may be due to the absorption enhancing action of vitamin C. The difference between Pasifika and 'others' could not be explained by current infant formula use, red meat and / or iron-fortified baby rice offered at six months, and age of introduction of complementary food, suggesting there may be a non-nutritive factor, or a more complex dietary factor that the study has not accounted for. Another possibility is that the study was not adequately powdered to detect such a difference. While these findings suggest a stronger focus for healthcare interventions is needed for Māori and other non-Pasifika ethnicities, the small group size of both Māori and Pasifika due to a high attrition rate in the study indicates further research to support

these findings would be beneficial. Additionally, further research into non-nutritive factors and absorption modifiers of iron as a possible explanation for any differences in iron status would help better direct future healthcare interventions and support in this crucial area of infant growth and development.

References

1. World Health Organization. Worldwide prevalence of anaemia 1993-2005: WHO global database on anaemia. 2008. Report No.: 9241596651.
2. Pasricha S-R, Drakesmith H, Black J, Hipgrave D, Biggs B-A. Control of iron deficiency anemia in low-and middle-income countries. Blood, The Journal of the American Society of Hematology. 2013;121(14):2607-17.
3. Rahman MM, Abe SK, Rahman MS, Kanda M, Narita S, Bilano V, et al. Maternal anemia and risk of adverse birth and health outcomes in low-and middle-income countries: systematic review and meta-analysis, 2. The American Journal of Clinical Nutrition. 2016;103(2):495-504.
4. Armitage AE, Moretti D. The importance of iron status for young children in low-and middle-income countries: a narrative review. Pharmaceuticals. 2019;12(2):59.
5. Eussen S, Alles M, Uijterschout L, Brus F, Van Der Horst-graat J. Iron intake and status of children aged 6-36 months in Europe: a systematic review. Annals of Nutrition and Metabolism. 2015;66(2-3):80-92.
6. Soh P, Ferguson EL, McKenzie JE, Homs M, Gibson RS. Iron deficiency and risk factors for lower iron stores in 6–24-month-old New Zealanders. European Journal of Clinical Nutrition. 2004;58(1):71-9.
7. Daniels L, Taylor RW, Williams SM, Gibson RS, Fleming EA, Wheeler BJ, et al. Impact of a modified version of baby led weaning on iron intake and status: a randomised controlled trial. British Medical Journal Open. 2018;8(6).
8. Heath ALM, Reeves Tuttle C, Simons MS, Cleghorn CL, Parnell WR. Longitudinal study of diet and iron deficiency anaemia in infants during the first two years of life. Asia Pacific Journal of Clinical Nutrition. 2002;11(4):251-7.
9. Akel R, Frankish J, Powles C, Tyler K, Watt J, Weston H, et al. Anaemia in Maori and European infants and children on admission to hospital. A co-operative survey from six New Zealand hospitals. The New Zealand Medical Journal. 1963;62:29-33.
10. Anyon C. Normal haemoglobin values in urban Polynesian infants: the possible deleterious influence of artificial feeding. The New Zealand Medical Journal. 1976;84(578):474-6.
11. Moyes C, O'Hagan L, Armstrong C. Anaemia in Maori infants--a persisting problem. The New Zealand Medical Journal. 1990;103(883):53-.
12. Rive S, Blacklock H, Hawkins R, Mitchell E. Anaemia in children aged 6 to 23 months attending the paediatric acute assessment unit in south Auckland. The New Zealand Medical Journal. 1996;109(1016):58-.
13. Wilson C, Grant CC, Wall CR. Iron deficiency anaemia and adverse dietary habits in hospitalised children. The New Zealand Medical Journal. 1999;112(1089):203-6.
14. Grant CC, Wall CR, Brunt D, Crengle S, Scragg R. Population prevalence and risk factors for iron deficiency in Auckland, New Zealand. Journal of Paediatrics and Child Health. 2007;43(7-8):532-8.
15. Bruner AB, Joffe A, Duggan AK, Casella JF, Brandt J. Randomised study of cognitive effects of iron supplementation in non-anaemic iron-deficient adolescent girls. The Lancet. 1996;348(9033):992-6.
16. Lozoff B, Jimenez E, Smith JB. Double burden of iron deficiency in infancy and low socioeconomic status: a longitudinal analysis of cognitive test scores to age 19 years. Archives of Pediatrics & Adolescent Medicine. 2006;160(11):1108-13.
17. World Health Organization. Guideline: intermittent iron supplementation in preschool and school-age children. Geneva: World Health Organization; 2011.
18. Lozoff B, Jimenez E, Wolf AW. Long-term developmental outcome of infants with iron deficiency. New England Journal of Medicine. 1991;325(10):687-94.
19. Cantwell RJ, Cleveland WW. The long term neurological sequelae of anemia in infancy. Pediatric Research. 1974;8(4):342-.

20. Palti H, Meijer A, Adler B. Learning achievement and behavior at school of anemic and non-anemic infants. Early Human Development. 1985;10(3-4):217-23.
21. Hurtado EK, Claussen AH, Scott KG. Early childhood anemia and mild or moderate mental retardation. The American Journal of Clinical Nutrition. 1999;69(1):115-9.
22. Lozoff B, Jimenez E, Hagen J, Mollen E, Wolf AW. Poorer behavioral and developmental outcome more than 10 years after treatment for iron deficiency in infancy. Pediatrics. 2000;105(4):e51-e.
23. Antunes H. Iron Deficiency Anaemia in Infants - A Prospective Neurodevelopment Evaluation. Faculty of Medicine, University of Portugal; 2004.
24. DeMaeyer EM, Dallman P, Gurney JM, Hallberg L, Sood S, Srikantia S, et al. Preventing and controlling iron deficiency anaemia through primary health care: a guide for health administrators and programme managers: World Health Organization; 1989.
25. Aggett PJ, Agostoni C, Axelsson I, Bresson J-L, Goulet O, Hernell O, et al. Iron metabolism and requirements in early childhood: do we know enough?: a commentary by the ESPGHAN Committee on Nutrition. Journal of Pediatric Gastroenterology and Nutrition. 2002;34(4):337-45.
26. Cogswell ME, Looker AC, Pfeiffer CM, Cook JD, Lacher DA, Beard JL, et al. Assessment of iron deficiency in US preschool children and nonpregnant females of childbearing age: National Health and Nutrition Examination Survey 2003–2006. The American Journal of Clinical Nutrition. 2009;89(5):1334-42.
27. Lozoff B, Kaciroti N, Walter T. Iron deficiency in infancy: applying a physiologic framework for prediction. The American Journal of Clinical Nutrition. 2006;84(6):1412-21.
28. Dewey KG. Nutrition, growth, and complementary feeding of the brestfed infant. Pediatric Clinics of North America. 2001;48(1):87-104.
29. Chantry CJ, Howard CR, Auinger P. Full breastfeeding duration and risk for iron deficiency in US infants. Breastfeeding Medicine. 2007;2(2):63-73.
30. Maguire JL, Salehi L, Birken CS, Carsley S, Mamdani M, Thorpe KE, et al. Association between total duration of breastfeeding and iron deficiency. Pediatrics. 2013;131(5):e1530-e7.
31. Luo R, Shi Y, Zhou H, Yue A, Zhang L, Sylvia S, et al. Anemia and feeding practices among infants in rural Shaanxi Province in China. Nutrients. 2014;6(12):5975-91.
32. Hipgrave D, Fu X, Zhou H, Jin Y, Wang X, Chang S, et al. Poor complementary feeding practices and high anaemia prevalence among infants and young children in rural central and western China. European Journal of Clinical Nutrition. 2014;68(8):916-24.
33. Altucher K, Rasmussen KM, Barden EM, Habicht J-P. Predictors of improvement in hemoglobin concentration among toddlers enrolled in the Massachusetts WIC Program. Journal of the American Dietetic Association. 2005;105(5):709-15.
34. Hopkins D, Emmett P, Steer C, Rogers I, Noble S, Emond A. Infant feeding in the second 6 months of life related to iron status: an observational study. Archives of Disease in Childhood. 2007;92(10):850-4.
35. Taylor RW, Conlon CA, Beck KL, von Hurst PR, Te Morenga LA, Daniels L, et al. Nutritional Implications of Baby-Led Weaning and Baby Food Pouches as Novel Methods of Infant Feeding: Protocol for an Observational Study. JMIR Research Protocols. 2021;10(4):e29048.
36. Atkinson J, Salmond C, Crampton P. NZDep2018 Index of Deprivation, Interim Research Report. Wellington: University of Otago; 2019.
37. Suchdev PS, Namaste SM, Aaron GJ, Raiten DJ, Brown KH, Flores-Ayala R, et al. Overview of the biomarkers reflecting inflammation and nutritional determinants of anemia (BRINDA) project. Advances in Nutrition. 2016;7(2):349-56.
38. Diana A, Haszard JJ, Purnamasari DM, Nurulazmi I, Luftimas DE, Rahmania S, et al. Iron, zinc, vitamin A and selenium status in a cohort of Indonesian infants after adjusting for inflammation using several different approaches. British Journal of Nutrition. 2017;118(10):830-9.
39. Domellof M, Dewey KG, Lonnerdal B, Cohen RJ, Hernell O. The diagnostic criteria for iron deficiency in infants should be reevaluated. The Journal of Nutrition. 2002;132(12):3680-6.

40. World Health Organization. Haemoglobin concentrations for the diagnosis of anaemia and assessment of severity. World Health Organisation; 2011 2011.
41. Grant C, Prestidge T, Noel H. Iron Deficiency Auckland, New Zealand2016 [cited 2022 8/2/2022]. Available from: https://starship.org.nz/guidelines/iron-deficiency/.
42. Mast AE, Blinder MA, Gronowski AM, Chumley C, Scott MG. Clinical utility of the soluble transferrin receptor and comparison with serum ferritin in several populations. Clinical Chemistry. 1998;44(1):45-51.
43. Grant C, Wall C, Wilson C, Taua N. Risk factors for iron deficiency in a hospitalized urban New Zealand population. Journal of Paediatrics and Child Health. 2003;39(2):100-6.
44. Poppe M. Iron deficient children. The New Zealand Medical Journal. 1993;106(963):392-.
45. Wall CR, Brunt DR, Grant CC. Ethnic variance in iron status: is it related to dietary intake? Public Health Nutrition. 2009;12(9):1413-21.
46. Bayele HK, Srai SKS. Genetic variation in hepcidin expression and its implications for phenotypic differences in iron metabolism. Haematologica. 2009;94(9):1185.
47. Trent R, Mickleson K, Yakas J, Hertzberg M. Population genetics of the globin genes in Polynesians. Hemoglobin. 1988;12(5-6):533-7.
48. Trent R, Mickleson K, Wilkinson T, Yakas J, Bluck R, Dixon M, et al. α Globin gene rearrangements in polynesians are not associated with malaria. American Journal of Hematology. 1985;18(4):431-3.
49. Yenchitsomanus P, Summers K, Board P, Bhatia K, Jones G, Johnston K, et al. Alpha-thalassemia in Papua New Guinea. Human Genetics. 1986;74(4):432-7.
50. Kirch PV. Peopling of the Pacific: A holistic anthropological perspective. Annual Review of Anthropology. 2010;39(1):131-48.
51. Katiforis I, Fleming EA, Haszard JJ, Hape-Cramond T, Taylor RW, Heath A-LM. Energy, Sugars, Iron, and Vitamin B12 Content of Commercial Infant Food Pouches and Other Commercial Infant Foods on the New Zealand Market. Nutrients. 2021;13(2):657.

**Supplementary table I.** Early feeding practices by ethnicity

| Feeding practice | Māori, n (%) | Pasifika, n (%) | 'Others', n (%) |
|---|---|---|---|
| *n* | 63 | 28 | 249 |
| Using baby-led weaning at around 6 months of age | 7 (11.1) | 1 (3.6) | 40 (16.1) |
| Frequent pouch use[a] | **24 (38.1)[c]** | **12 (42.9)[c]** | 56 (22.5) |
| Early introduction to complementary food[b] | **19 (30.2)[c]** | 7 (25.0) | 43 (17.3) |
| Iron-fortified baby rice consumed at around 6 months of age | 36 (57.1) | 15 (53.6) | 110 (44.2) |
| Red meat consumed at around 6 months of age | **29 (46.0)[c]** | 12 (42.9) | 155 (62.3) |
| Currently having infant formula | 38 (60.3) | 15 (53.6) | 127 (51.0) |

[a] Pouches are given at least five times per week
[b] Defined as introduced to complementary foods at <5 months of age
[c] Significantly different to 'others', $p<0.05$

# Chapter 7 | Upoko Tuawhitu

## Discussion and Conclusions

*This chapter provides a summary of the findings for each aim of this study, discusses the implications of those findings, and provides concluding remarks and directions for future research.*

Inequities in nutrition-related health outcomes in Aotearoa New Zealand (NZ) are predicted by ethnicity, with Māori and Pasifika bearing the greatest burden compared to other ethnicities. Little is currently known about what and how we feed infants in NZ, particularly Māori and Pasifika infants. Therefore, the primary aim of this study was to conduct an intra-ethnic analysis of infant complementary feeding practices, nutrient intake and density from complementary foods, and iron status between Māori, Pasifika, and non-Māori, non-Pasifika infants.

The first aim was to describe complementary feeding practices, presented in chapter three of this thesis. The complementary feeding practices explored by ethnicity encompassed timing of introduction to complementary foods, method of feeding (baby-led weaning versus traditional spoon-feeding), pouch use, types of first foods offered, traditional cultural foods offered, and inappropriate drinks. Infants' ethnicity was identified by their caregiver and reported as total response. Infants were categorised as Māori, Pasifika, or 'other' (non-Māori, non-Pasifika). Baby-led weaning (BLW) prevalence was lowest for all ethnicities at the time of introducing complementary foods and highest at the 'current' time (7.0–10.0 months), with the majority of caregivers reporting having tried BLW at least once. Similar proportions of Māori and 'other' infants were baby-led weaned, compared to only a very small proportion of Pasifika infants. Pouch use prevalence, frequency, and proportion of infants sucking directly from the pouch was similarly lowest at the time of introducing complementary food, and highest at the current time, with Māori and Pasifika infants having a higher prevalence of frequent pouch use and sucking directly from the pouch than 'other' infants. The majority of infants from all ethnic groups were introduced to complementary food at around six months of age, as per the Manatū Hauora – Ministry of Health (MoH) guidelines (1). However, for Māori and Pasifika, this represented 56.5% and 62.2% respectively, with 80.9% of 'others' introducing complementary foods in a timely manner. The majority of infants from all ethnic groups had red meat around six months of age, with approximately 50% of all ethnicities having iron-fortified baby rice. For all ethnicities, the most common type of first food offered was vegetables, and the most common texture for the first food offered was pureed. Age-inappropriate drinks were given to 18% of Māori, 21% of Pasifika, and 4% of 'other' infants. Caregivers were asked if traditional cultural foods were given to their infants, but there was a low response rate with just 9% of Māori and 21% of Pasifika reporting that they offered cultural foods to their infants. A possible reason for such a low response rate is that whānau do not consider their everyday foods to be different or special, and just see it as ordinary everyday food. Rephrasing this question using specific food examples, or even providing an exhaustive list of cultural foods and cooking practices may help overcome this issue in future research.

Baby-led weaning was most prevalent at the 'current' timepoint – i.e., at the time of conducting the study when the infants were aged 7.0–10.0 months, as compared to when first introducing complementary foods and/or at six months of age. However, there was a limitation in the way in which BLW was measured: infants were categorised as "BLW" or "partial BLW" according to how frequently the caregiver reported the infant as self-feeding, without specifically asking about an intention to BLW. Given that 7.0–10.0 months of age is a common time for the introduction to finger foods it may not be an accurate representation of the true prevalence of BLW. At six months of age, when finger-foods are less common, there was a low prevalence of BLW in all ethnic groups, with ~14% of both Māori and 'others' being identified as using BLW, and just 6% of Pasifika. As this is not a nationally generalisable cohort these findings cannot be extrapolated to the wider population, but with ethnically and socio-economically diverse participants it gives some insight into the use of BLW in NZ.

Key areas of concern highlighted by these findings were the prevalence of early introduction to complementary foods for Māori and Pasifika infants, and the prevalence of pouch use alongside the frequency of infants sucking food directly from the pouch nozzle. With regard to the early introduction to complementary food, the World Health Organisation (WHO) changed their recommendations to "around six months of age" in 2001 (2), and these guidelines were adopted by the Ministry of Health in NZ in 2006 (3). The prevalence of early introduction of complementary foods amongst Māori and Pasifika infants is concerning in light of their higher prevalence of gastrointestinal and respiratory tract illnesses, with displacement of breast milk by solid food prior to six months increasing the risk of illnesses (4). However, when comparing the findings in the present study to those in the Growing Up In New Zealand (GUINZ) study (2009 & 2010) (5), it appears that the proportion of infants being introduced early is declining, with a 17 % lower frequency of early introduction for Māori, 12% for Pasifika, and 19% for 'others' in the FFNZ study.

A number of potential concerns have been identified around pouch use, in particular the high sugar content compared to commercial infant food not packaged in pouches and the low iron and meat content. Pouches now make up the majority of the market share of commercial infant food and lack advisory warnings on the label around the method of feeding – presumably due to a lack of research, despite expert groups advising against sucking food directly from the pouches (6, 7). Māori and Pasifika children have a higher rate of dental caries, and as adults up to a four-fold higher rates of type 2 diabetes than NZ Europeans (8), significantly higher rates of cardiovascular disease (9-11), along with historically high rates of iron deficiency with and without anaemia (12-17). Therefore, the

increasing availability, popularity, and use of baby food pouches could have implications for these health outcomes due to the higher sugar and lower iron content. The high sugar content is a risk for both dental caries forming as well as the infant developing a sweet-taste preference that may be life-long. The low iron content may impact on iron status, which in turn can have far-reaching implications for cognitive and social development and can affect IQ in adolescence (18-22).

The second aim of this study was presented in chapter four, and examined energy and nutrient intake from complementary food, and the nutrient density of the complementary diet. This is critical information as complementary food has the important role of filling the gap between what the infant's nutrient requirements are, and what they obtain from breast milk and / or formula. Furthermore, infants have exceptionally high nutrient requirements per kilogram of body weight (23). Currently very little is known about the complementary diets of NZ infants and investigating this is essential to better understand the complexities of the infant's diet as well as helping inform future nutrition interventions and research to help close nutrient gaps.

Energy intake was stratified by sex, age, and ethnicity. Expectedly, energy intake increased between 7 and 10 months of age and was higher overall for boys than girls. Of interest, Māori and Pasifika girls had a much lower intake of energy from complementary foods compared to 'other' girls and all boys. A possible reason for this is that Māori and Pasifika girls may acquire a greater proportion of their nutrients from milk feeds than from complementary foods at nine months of age, or they may be consuming less energy-dense complementary foods. Pasifika girls still met the recommended intake of energy from complementary foods at nine months, but Māori girls were marginally lower (23). For macronutrients, 'other' infants had a significantly higher intake of total fat as a percentage of their total energy intake compared to Māori and Pasifika, and a significantly higher intake of protein as a percentage of total energy intake compared to just Māori. 'Other' infants had a significantly lower proportion of carbohydrate intake than both Māori and Pasifika infants. Pasifika infants had a significantly higher intake of sugar in grams per day compared to others, while sugar as a percentage of total energy intake was significantly higher for Māori. This may be reflective of the relative cost of the food sources, with protein-rich foods having a higher cost (24), and the Māori and Pasifika infants tended to live in more highly deprived neighbourhoods than 'other' infants. The nutrient density of complementary foods was found to be inadequate for iron, zinc, and calcium but adequate for vitamin A, vitamin $B_{12}$, and vitamin C.

The common finding of inadequate nutrient density of complementary foods for iron, zinc, and calcium for all ethnic groups is consistent with international findings in developing countries (25-27). This presents a unique opportunity to examine the quality and nutritional density of complementary food, but also raises a question about the recommendations for iron in particular – could they be too high? Given that dietary iron intake has not been strongly or consistently associated with iron status this is worth exploring further.

The method used to obtain dietary data was a multiple (three) pass 24-hour recall. This method provides less of a burden than a weighed diet record, and alongside detailed prompts at each pass for forgotten foods and fluids and the use of props this has provided a valid and reliable method to assess the nutrient intake of the infants in this study. Furthermore, the use of nutrient density to assess the infants' complementary diets has given a clearer understanding of where critical nutrient gaps lie. Furthermore, this focus on the complementary diet helps better direct where future interventions are needed given that it is appropriate to manipulate the complementary diet rather than milk feeds.

The third and final aim of this thesis was to describe infant iron status using body iron expressed as mg/kg body weight of NZ infants stratified by ethnicity. There is little recent robust data on the iron status of NZ infants, yet iron is crucial for healthy growth and development. Iron deficiency has short- and long-term effects on immune function, cognitive function (28, 29), and motor skill development (30). The most recent available data in NZ suggest that 3 – 22% of infants aged between six and 12 months have some degree of iron deficiency with or without anaemia (31-33), with that figure stretching to up to 65% when looking at just Māori and Pasifika (13-17). These figures are based on historical data however, using varying biochemical indices and cut-offs to define iron deficiency and anaemia, as well as using hospitalised infants in some cases.

Of the overall study group of 625 infants, biochemical, demographic, and feeding practice data was collected from 360, with the remaining 265 infants not providing a blood sample (Appendix D). In total, 96.4% of Pasifika infants were iron sufficient, followed by 82.5% of Māori and 77.5% of 'other' infants. In total, 6% of 'other' infants were categorised with iron-deficiency anaemia (IDA), 8% with early functional iron deficiency, and 8.4% with iron depletion. Of the Māori infants, 4.7% had IDA and early functional iron deficiency respectively, and 8% were iron depleted. One (3.6%) Pasifika infant was iron depleted.

Given historical data indicating a high prevalence of iron deficiency with and without anaemia for Māori and Pasifika infants it was surprising to find a very low prevalence of ID and IDA amongst Pasifika, and to a lesser extent Māori in this cohort. This can perhaps be explained in part by some older studies using inappropriate cut-offs to determine iron status, and in some cases hospitalised infants (13, 15-17, 34, 35). A more recent and similarly designed study still contrasts with the present study, finding that 20% of Māori infants and 17% of Pasifika were iron deficient compared to just 7% of NZE infants (12). A later study examining the same cohort determined that non-nutritive factors were likely determinants of ID and IDA (36). Notably, genetic factors can determine up to 50% of the variability in iron status (37, 38). It is possible that a higher rate of infection and inflammation in Māori and Pasifika infants (39, 40) could explain the higher rate of ID previously seen, however it is not known if this is a factor in the present study. Another consideration is the higher rate of α-thalassaemia affecting Māori and Pasifika, at 16 – 20% of the population (41-44) – the resulting microcytic hypochromic red blood cells may have been interpreted as anaemia in older studies.

When mediating factors were analysed to explain the ethnic difference in iron status it was found that frequent pouch consumption by Pasifika infants was the only factor that partially explained the difference. Infant formula use, early introduction of complementary food, consumption of iron-fortified baby rice at six months of age, and consumption of red meat at six months of age were not able to explain the difference. This was surprising because commercial baby food pouches in NZ are not fortified with iron, containing just 0.3 mg/100g of iron on average (45). Additionally, meat-based pouches contain on average only 5–10% meat or poultry (45). It is therefore likely that non-dietary factors, possibly alongside a more complex interplay of dietary factors, such as iron being obtained from more highly bioavailable sources, or being eaten in conjunction with vitamin C, or away from dietary inhibitors such as calcium and phytic acid, may explain the differences. This idea is further supported by the high nutrient density for vitamin C in the infants' complementary diets of 6.6 – 8.0 mg/418 kJ vs the required density of 1.5-1.7 mg/418 kJ.

Perhaps surprisingly, the nutrient density for iron in the infants complementary diet was particularly low (0.8 mg / 418 kJ for Māori and 'others', 0.9 mg / 418 kJ for Pasifika, vs the recommended 4.5 mg and 3.0 mg / 418 kJ for 6-8 months and 9-11 months respectively), yet the vast majority of all ethnicities were iron sufficient (82.5% Māori, 96.4% Pasifika, 76.0% 'others'). Iron status is affected by a kaleidoscope of interrelated factors, from maternal iron stores, gestation, various confounders such as infection and inflammation, losses, genetic factors, and dietary enhancers and inhibitors

(46-58). Therefore, dietary iron intake from the complementary diet is just one of many different factors that may all have a profound influence on the infant's iron status, however the marked discrepancy between dietary iron intake from complementary foods and iron status is worthy of further investigation into the recommended dietary intake currently set.

Overall, the solution to understanding iron absorption, utilisation, and requirements is complex and affected by many factors that vary between infants. Untangling this in order to provide the best possible nutrition advice for infants on a population level is challenging and perhaps best conducted on an infant-to-infant basis.

*Conclusion*

Complementary feeding practices and foods play a critical role in the growth and development of infants. Feeding practices and complementary foods offered influence not just the health and growth of the infant, but the infant's first experience with foods which helps lay down a life-long pattern of preferences. The potential impact of feeding practices on the infant is further reinforced by the foods offered — particularly in terms of nutrient density of the complementary diet, with the infant having the highest needs per kilogram of body weight than at any other stage in life. These factors can influence the lifelong morbidity risk for infants, with both communicable and non-communicable disease disproportionately experienced by Māori and Pasifika (59). Furthermore, iron deficiency and iron deficiency anaemia has historically been prevalent particularly amongst Māori and Pasifika infants, affecting immune function, cognitive development, and gross motor skill development. Therefore, it is important to look at this through a Māori and Pasifika lens to help provide a better understanding of where future research and public health initiatives can be best focussed.

Complementary feeding practices of concern were the prevalence of pouch feeding alongside infants sucking directly from the pouch nozzle, particularly in light of the high sugar and low iron content of pouch food. Reassuringly, the majority of infants from all ethnic groups were introduced to CF at around six months of age, while 14% of both Māori and 'other' infants were baby-led weaned at six months of age, compared to just 6% of Pasifika. The majority of infants also had either red meat or iron-fortified baby rice (or both) at around six months of age, and a very low prevalence of age-inappropriate drinks. Other than pouch use, for which no formal guidelines are published in NZ, the majority of infants in all ethnic groups are fed according to the guidelines.

The nutrient density of complementary foods was inadequate for iron, zinc, and calcium but there were no significant nor trending differences between ethnic groups, indicating that solving the problem of inadequate nutrient density for these minerals in complementary food requires a broad approach suitable for all cultural backgrounds. There were significant differences in the proportion of fat, protein, total carbohydrate, and sugar intake, with 'other' infants having a significantly higher intake of fat and protein compared to Māori, and just fat compared to Pasifika. Pasifika and Māori both had a significantly higher sugar and total carbohydrate intake than 'other' infants though, which may be reflective of the relative cost of the food sources of these macronutrients given that Māori and Pasifika infants tended to live in more highly deprived neighbourhoods than 'other' infants. Despite this, the micronutrient density of the complementary diet was very similar for all groups.

Pasifika infants had the highest prevalence of iron sufficiency, followed by Māori, then 'other' infants. This was a surprising finding with previous studies indicating that Pasifika and Māori tended to have much higher rates of ID and IDA than NZE infants. Frequent pouch use was the only factor examined that could partially explain this difference. This is surprising due to lack of iron fortification in commercial baby food pouches and low proportion of meat in the meat-based pouches. However, it may be that the Pasifika ethnicity also has a partial effect, and there are other effects that were not measured for, such as the iron bioavailability of complementary foods and dietary modifiers. It is important to note that due to a high attrition rate the Māori and Pasifika groups were small, therefore further research to support these findings are needed.

Pēpi (infants) and tamariki (children) are our greatest treasure and represent hope for the future. Kai (food) is about nourishment, love, and connection, and helps tamariki to grow into strong healthy adults by giving them the best possible start in life. This study has highlighted important areas in need of greater understanding and research to help better the lives of our future generations.

*Directions for future research*

1. A high prevalence of baby food pouch consumption was identified particularly for Māori and Pasifika infants, but little is known about the potential health impacts of frequent pouch use and direct feeding from the pouch in these vulnerable groups. Research investigating both the nutritional content as well as the impact of sucking directly from the nozzle is needed.

2. Very few Māori and Pasifika reported using traditional cultural foods or practices, despite an increase in “de-colonising through kai (food)” (60). Given the health benefits of a traditional responsive feeding pattern using cultural foods, qualitative research examining barriers, prevalence, and nutrition-related health outcomes would be worthwhile.

3. Currently there appears to be loopholes in regulations relating to the fortification and food content of commercial infant food with iron, whereby food products labelled as “4+ months” are not required to be iron fortified, meaning manufacturers can avoid fortifying food with enough iron by labelling the package in this way. Additionally, there is no requirement for a minimum percentage of meat in infant foods that contain a meat ingredient in the title. Given the very low iron intake for NZ infants a review of the legislation and labelling of commercial infant foods to provide recommendations is urgently needed.

4. The nutrient density of the complementary diet of infants in NZ is lacking for iron, zinc, and calcium. Research is needed to investigate novel and practicable fortification strategies to fill this critical nutrient gap and are appropriate for a range of cultural backgrounds.

5. Non-nutritive factors appear to play a key role in the iron status of infants in NZ. Given genetic variations in hepcidin that can result in phenotypic idiosyncrasies, such as the disproportionate synthesis of hepcidin or resistance of iron transporters to its effects, further research investigating hepcidin concentration alongside iron status would greatly improve understanding of infant iron status.

References

1. Ministry of Health. Food and Nutrition Guidelines for Healthy Infants and Toddlers (Aged 0-2): A background paper (4th Ed) - Partially Revised December 2012). Wellington, New Zealand: Ministry of Health; 2008.
2. World Health Organization and UNICEF. Global strategy for infant and young child feeding: World Health Organization; 2003.
3. Ministry of Health. Food and nutrition guidelines for healthy pregnant and breastfeeding women: a background paper. Wellington, New Zealand: Ministry of Health 2006.
4. Hobbs MR, Morton SM, Atatoa-Carr P, Ritchie SR, Thomas MG, Saraf R, et al. Ethnic disparities in infectious disease hospitalisations in the first year of life in New Zealand. Journal of Paediatrics and Child Health. 2017;53(3):223-31.
5. Ferreira SS, Marchioni DML, Wall CR, Gerritsen S, Teixeira JA, Grant CC, et al. Prevalence and maternal determinants of early and late introduction of complementary foods: results from the Growing up in New Zealand cohort study. British Journal of Nutrition. 2022:1-12.
6. Theurich MA, Fewtrell M, Baumgartner J, Perkin MR, Breda J, Wickramansinghe K, et al. Moving Complementary Feeding Forward: Report on a Workshop of the Federation of International Societies for Pediatric Gastroenterology, Hepatology and Nutrition (FISPGHAN) and the World Health Organization Regional Office for Europe. Journal of Pediatric Gastroenterology and Nutrition. 2022;75(4):411-7.
7. Koletzko B, Bührer C, Ensenauer R, Jochum F, Kalhoff H, Lawrenz B, et al. Complementary foods in baby food pouches: position statement from the Nutrition Commission of the German Society for Pediatrics and Adolescent Medicine (DGKJ, eV). Molecular and Cellular Pediatrics. 2019;6(1):1-5.
8. Atlantis E, Joshy G, Williams M, Simmons D. Diabetes among Māori and other ethnic groups in New Zealand. Diabetes Mellitus in Developing Countries and Underserved Communities: Springer; 2017. p. 165-90.
9. Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH, et al. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. Circulation. 2009;120(11):1011-20.
10. Slavin J. Beverages and body weight: challenges in the evidence-based review process of the Carbohydrate Subcommittee from the 2010 Dietary Guidelines Advisory Committee. Nutrition Reviews. 2012;70(suppl_2):S111-S20.
11. Fidler Mis N, Braegger C, Bronsky J, Campoy C, Domellöf M, Embleton ND, et al. Sugar in infants, children and adolescents: a position paper of the European society for paediatric gastroenterology, hepatology and nutrition committee on nutrition. Journal of Pediatric Gastroenterology and Nutrition. 2017;65(6):681-96.
12. Grant CC, Wall CR, Brunt D, Crengle S, Scragg R. Population prevalence and risk factors for iron deficiency in Auckland, New Zealand. Journal of Paediatrics and Child Health. 2007;43(7-8):532-8.
13. Akel R, Frankish J, Powles C, Tyler K, Watt J, Weston H, et al. Anaemia in Maori and European infants and children on admission to hospital. A co-operative survey from six New Zealand hospitals. The New Zealand Medical Journal. 1963;62:29-33.
14. Anyon C. Normal haemoglobin values in urban Polynesian infants: the possible deleterious influence of artificial feeding. The New Zealand Medical Journal. 1976;84(578):474-6.
15. Moyes C, O'Hagan L, Armstrong C. Anaemia in Maori infants--a persisting problem. The New Zealand Medical Journal. 1990;103(883):53-.
16. Rive S, Blacklock H, Hawkins R, Mitchell E. Anaemia in children aged 6 to 23 months attending the paediatric acute assessment unit in south Auckland. The New Zealand Medical Journal. 1996;109(1016):58-.
17. Wilson C, Grant CC, Wall CR. Iron deficiency anaemia and adverse dietary habits in hospitalised children. The New Zealand Medical Journal. 1999;112(1089):203-6.

18. Cantwell RJ, Cleveland WW. The long term neurological sequelae of anemia in infancy. Pediatric Research. 1974;8(4):342-.
19. Palti H, Meijer A, Adler B. Learning achievement and behavior at school of anemic and non-anemic infants. Early Human Development. 1985;10(3-4):217-23.
20. Hurtado EK, Claussen AH, Scott KG. Early childhood anemia and mild or moderate mental retardation. The American Journal of Clinical Nutrition. 1999;69(1):115-9.
21. Lozoff B, Jimenez E, Hagen J, Mollen E, Wolf AW. Poorer behavioral and developmental outcome more than 10 years after treatment for iron deficiency in infancy. Pediatrics. 2000;105(4):e51-e.
22. Antunes H. Iron Deficiency Anaemia in Infants - A Prospective Neurodevelopment Evaluation. Faculty of Medicine, University of Portugal; 2004.
23. Dewey KG. The challenge of meeting nutrient needs of infants and young children during the period of complementary feeding: an evolutionary perspective. The Journal of Nutrition. 2013;143(12):2050-4.
24. Brooks RC, Simpson S, Raubenheimer D. The price of protein: combining evolutionary and economic analysis to understand excessive energy consumption. Obesity Reviews. 2010;11(12):887-94.
25. Hotz C, Gibson R. Complementary feeding practices and dietary intakes from complementary foods amongst weanlings in rural Malawi. European Journal of Clinical Nutrition. 2001;55(10):841-9.
26. Campos R, Hernandez L, Soto-Mendez MJ, Vossenaar M, Solomons NW. Contribution of complementary food nutrients to estimated total nutrient intakes for rural Guatemalan infants in the second semester of life. Asia Pacific Journal of Clinical Nutrition. 2010;19(4):481-90.
27. Faber M. Complementary foods consumed by 6–12-month-old rural infants in South Africa are inadequate in micronutrients. Public Health Nutrition. 2005;8(4):373-81.
28. Bruner AB, Joffe A, Duggan AK, Casella JF, Brandt J. Randomised study of cognitive effects of iron supplementation in non-anaemic iron-deficient adolescent girls. The Lancet. 1996;348(9033):992-6.
29. Lozoff B, Jimenez E, Smith JB. Double burden of iron deficiency in infancy and low socioeconomic status: a longitudinal analysis of cognitive test scores to age 19 years. Archives of Pediatrics & Adolescent Medicine. 2006;160(11):1108-13.
30. Lozoff B, Jimenez E, Wolf AW. Long-term developmental outcome of infants with iron deficiency. New England Journal of Medicine. 1991;325(10):687-94.
31. Soh P, Ferguson EL, McKenzie JE, Homs M, Gibson RS. Iron deficiency and risk factors for lower iron stores in 6–24-month-old New Zealanders. European Journal of Clinical Nutrition. 2004;58(1):71-9.
32. Daniels L, Taylor RW, Williams SM, Gibson RS, Fleming EA, Wheeler BJ, et al. Impact of a modified version of baby led weaning on iron intake and status: a randomised controlled trial. British Medical Journal Open. 2018;8(6).
33. Heath ALM, Reeves Tuttle C, Simons MS, Cleghorn CL, Parnell WR. Longitudinal study of diet and iron deficiency anaemia in infants during the first two years of life. Asia Pacific Journal of Clinical Nutrition. 2002;11(4):251-7.
34. Grant C, Wall C, Wilson C, Taua N. Risk factors for iron deficiency in a hospitalized urban New Zealand population. Journal of Paediatrics and Child Health. 2003;39(2):100-6.
35. Poppe M. Iron deficient children. The New Zealand Medical Journal. 1993;106(963):392-.
36. Wall CR, Brunt DR, Grant CC. Ethnic variance in iron status: is it related to dietary intake? Public Health Nutrition. 2009;12(9):1413-21.
37. Benyamin B, McRae AF, Zhu G, Gordon S, Henders AK, Palotie A, et al. Variants in TF and HFE explain~ 40% of genetic variation in serum-transferrin levels. The American Journal of Human Genetics. 2009;84(1):60-5.

38. Benyamin B, Ferreira MA, Willemsen G, Gordon S, Middelberg RP, McEvoy BP, et al. Common variants in TMPRSS6 are associated with iron status and erythrocyte volume. Nature Genetics. 2009;41(11):1173-5.
39. Shackleton N, Broadbent JM, Thornley S, Milne BJ, Crengle S, Exeter DJ. Inequalities in dental caries experience among 4-year-old New Zealand children. Community Dentistry and Oral Epidemiology. 2018;46(3):288-96.
40. Schluter PJ, Lee M. Water fluoridation and ethnic inequities in dental caries profiles of New Zealand children aged 5 and 12–13 years: analysis of national cross-sectional registry databases for the decade 2004–2013. BMC Oral Health. 2016;16(1):1-10.
41. Trent R, Mickleson K, Yakas J, Hertzberg M. Population genetics of the globin genes in Polynesians. Hemoglobin. 1988;12(5-6):533-7.
42. Trent R, Mickleson K, Wilkinson T, Yakas J, Bluck R, Dixon M, et al. α Globin gene rearrangements in polynesians are not associated with malaria. American Journal of Hematology. 1985;18(4):431-3.
43. Yenchitsomanus P, Summers K, Board P, Bhatia K, Jones G, Johnston K, et al. Alpha-thalassemia in Papua New Guinea. Human Genetics. 1986;74(4):432-7.
44. Kirch PV. Peopling of the Pacific: A holistic anthropological perspective. Annual Review of Anthropology. 2010;39(1):131-48.
45. Katiforis I, Fleming EA, Haszard JJ, Hape-Cramond T, Taylor RW, Heath A-LM. Energy, Sugars, Iron, and Vitamin B12 Content of Commercial Infant Food Pouches and Other Commercial Infant Foods on the New Zealand Market. Nutrients. 2021;13(2):657.
46. Leung A, Chan KW. Iron deficiency anemia. Advances in Pediatrics. 2001;48:385-408.
47. Kleinman RE. Pediatric nutrition handbook. Actividad Dietética. 2009;13(1):46.
48. Choi J, Kim C, Pai S. Erythropoietic activity and soluble transferrin receptor level in neonates and maternal blood. Acta Paediatrica. 2000;89(6):675-9.
49. Preziosi P, Prual A, Galan P, Daouda H, Boureima H, Hercberg S. Effect of iron supplementation on the iron status of pregnant women: consequences for newborns. The American Journal of Clinical Nutrition. 1997;66(5):1178-82.
50. Oski FA. Iron deficiency in infancy and childhood. New England Journal of Medicine. 1993;329(3):190-3.
51. Rao R, Georgieff M. Perinatal aspects of iron metabolism. Acta Paediatrica. 2002;91:124-9.
52. Van Rheenen P, Brabin BJ. Late umbilical cord-clamping as an intervention for reducing iron deficiency anaemia in term infants in developing and industrialised countries: a systematic review. Annals of Tropical Paediatrics. 2004;24(1):3-16.
53. Bothwell TH. Overview and mechanisms of iron regulation. Nutrition Reviews. 1995;53(9):237-45.
54. Boccio JR, Iyengar V. Iron deficiency. Biological Trace Element Research. 2003;94(1):1-31.
55. Jiang T, Jeter JM, Nelson SE, Ziegler EE. Intestinal blood loss during cow milk feeding in older infants: quantitative measurements. Archives of Pediatrics & Adolescent Medicine. 2000;154(7):673-8.
56. Thorsdottir I, Gunnarsson BS, Atladottir H, Michaelsen K, Palsson G. Iron status at 12 months of age—effects of body size, growth and diet in a population with high birth weight. European Journal of Clinical Nutrition. 2003;57(4):505-13.
57. Perry GS, Byers T, Yip R, Margen S. Iron nutrition does not account for the hemoglobin differences between blacks and whites. The Journal of Nutrition. 1992;122(7):1417-24.
58. Domellöf M, Lönnerdal B, Dewey KG, Cohen RJ, Rivera LL, Hernell O. Sex differences in iron status during infancy. Pediatrics. 2002;110(3):545-52.
59. Tapera R, Harwood M, Anderson A. A qualitative Kaupapa Māori approach to understanding infant and young child feeding practices of Māori and Pacific grandparents in Auckland, New Zealand. Public Health Nutrition. 2017;20(6):1090-8.

60. Rapata H, Heath A-LM, Wall C, Taylor R, Te Morenga L. Māori first foods: a Māori centred approach to understanding infant complementary feeding practices within Māori whānau. Kōtuitui: New Zealand Journal of Social Sciences Online. 2021:1-16.

# Āpitihanga

**Appendix A**: Information Sheet

# Feeding our babies – are we getting it right?

FIRST FOODS
— New Zealand —

## We would like to invite you to take part in the First Foods New Zealand (FFNZ) study – an exciting new project about how and what New Zealand babies are being fed

July, 2020


**Investigators**

Assoc Prof Anne-Louise Heath, University of Otago (Dunedin)
Prof Rachael Taylor, University of Otago (Dunedin)
Dr Cathryn Conlon, Massey University (Albany)
Dr Kathryn Beck, Massey University (Albany)
Dr Lisa Te Morenga, Victoria University (Wellington)


1

## Why?

How babies are introduced to solid foods might be important for baby's health, including how they grow, how healthy their teeth are, and whether they are getting enough nutrients like iron. We want to look at how and what babies are being fed, and what effect that has on their nutrition and health. This research will provide important information so that health professionals and policy makers can advise whānau on how to introduce solids safely to their babies. This study has ethical approval from the Health and Disability Ethics Committee: 19/STH/151.

## What does this study involve?

We are looking for parents or guardians from Dunedin, Auckland or Wellington with infants who are 7, 8, or 9 months of age to take part in this study. First Foods NZ is an 'observational' study, looking at what and how babies are fed. You will **not** have to change the way you feed your baby in any way or do anything different. We need to recruit a large number of families – a total of 625 – so expect that the study will finish in 2022.

## What would I be asked to do?

Attend three visits when your baby is between 7 and 10 months old. If you are breastfeeding, you may have two extra visits. These visits will take place over two weeks and will take 4-7 hours in total.

## First visit

This visit will take place at our Albany Massey University research center, your home, or your preferred location. Our research center is a comfortable and safe place with a breastfeeding area and plenty of toys for your babies and/or other children. There is also free parking available. During visit one we will measure baby's weight and length and ask you to fill out a couple of questionnaires about baby's feeding and health. We will ask you to tell us what, and how much, baby ate the day before. Because this can be difficult to remember, we will ask you to take photos of baby's food at the start of their meals and snacks the day before we visit. These are just taken on a phone or camera – we can lend you one if needed. This visit will take about one and a half hours.

## Second visit

This visit will take place at our Albany Massey University research center, your home, or preferred location. One of our staff will take some photographs of baby's mouth and teeth. Then we will ask you to tell us what, and how much, baby ate the previous day (like you did for the first visit). This visit will take less than one hour.

There will be one last online questionnaire after this visit.

## Third visit

This visit will be at your local Lab test collection center. A blood test will be taken, so that we can measure your baby's iron levels. The blood test will be done by someone who is very experienced at collecting blood samples from babies, and we will provide you with a numbing gel to make sure it doesn't hurt.

We will ask your permission to let us access information from your baby's "B4 School Check" when they are four years old so that we can see how their growth and dental health are tracking. We will need to access your baby's National Health Index (NHI) number to do this.

### If you are breastfeeding

If you are breastfeeding, we may invite you to be in another part of the study. We would ask you to drink a small amount of "special" water that lets us track how much breast milk your baby drinks. This method is very safe and is used all over the world. If you did this part of the study, then you would do the same study as everyone else but complete some extra tasks at the first and second visits and have two extra visits. This part of the study would take about 20-30 minutes at each of the four visits. At the first visit you would drink the water. At all four visits we would collect some saliva (spit) from you and your baby, so that we can measure how much of the "special" water your baby has got from your breast milk. At a couple of the visits, we would collect your height and weight.

This is an important part of the study because, for the first time, parents and health professionals will know how much breast milk New Zealand babies are getting.

## Who pays for the study?

This study is funded by the Health Research Council of New Zealand. There is no cost for you to take part. However, as a recognition for taking part in the study we will give you a $150 voucher as a thank you.

**Appendix B:** Consent form

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA
UNIVERSITY OF NEW ZEALAND

# Consent form for the First Foods NZ study

| **Please tick to show you consent to the following** | | |
|---|---|---|
| I have read and understand the information pamphlet for volunteers in the First Foods NZ study. | | |
| I have had enough time to decide if I want to take part in the study. | | |
| I have had the opportunity to get friend, family, or whānau support to help me ask questions and understand the study. | | |
| I am happy with the answers I have been given about this study and I have a copy of this consent form and the information pamphlet. | | |
| I understand that taking part in this study is my choice and that I may withdraw from the study at any time without it affecting my health care or that of my baby. | | |
| I consent to a blood sample being from my baby from an appropriate Labtest facility. | Yes ☐ | No ☐ |
| **If I consent to a blood sample being taken from my baby:**<br>I understand that any blood that is left over after analysis will be disposed of with:<br>a) Standard biohazard laboratory disposal ☐<br>OR<br>b) An appropriate karakia ☐ | | |
| I consent to allowing the researchers to access information about my child's growth and dental health from their "B4 School Check" when they are about four years old. I understand that this means they will need to access my baby's National Health Index (NHI) number. | Yes ☐ | No ☐ |
| If I decide to withdraw from the study, I agree that the information collected about me up to the point when I withdraw may continue to be processed. | Yes ☐ | No ☐ |

**[PTO]**

| | | |
|---|---|---|
| I consent to my baby's GP being informed about any significant abnormal results from their iron blood test.<br>Name of GP<br>.....<br>Address or Name of GP's practice<br>..... | | |
| I consent to a dentist or dental therapist being informed about any significant abnormal results from my baby's teeth check.<br>Name of my preferred dentist or dental therapist<br>.....<br>Address or Name of practice<br>..... | | |
| I don't have a preferred dentist or dental therapist so if there are any significant abnormal results from my baby's teeth check please refer my baby to my local Community Oral Health Service (a free service). | Yes ☐ | No ☐ |
| I understand that taking part in this study is confidential and that no material which could identify me, or my baby, will be used in any reports on this study. | | |
| I understand the ACC provisions in case of injury during the study. | | |
| I know who to contact if I have questions about the study. | | |
| I understand my responsibilities as a volunteer taking part in the study. | | |
| I wish to receive a summary of the results from the study when the study is finished and will keep the researchers informed if my address changes. | Yes ☐ | No ☐ |
| I am happy for the data collected in this study to be used in future research on topics related to this study. | Yes ☐ | No ☐ |
| I am happy to be contacted in the future to see if I might be interested in taking part in other related studies. | Yes ☐ | No ☐ |

**[PTO]**

**Declaration by participant:**

I hereby consent to take part in this study.

Participant's name: _____

Signature: _____ Date: _____

**Declaration by member of research team:**

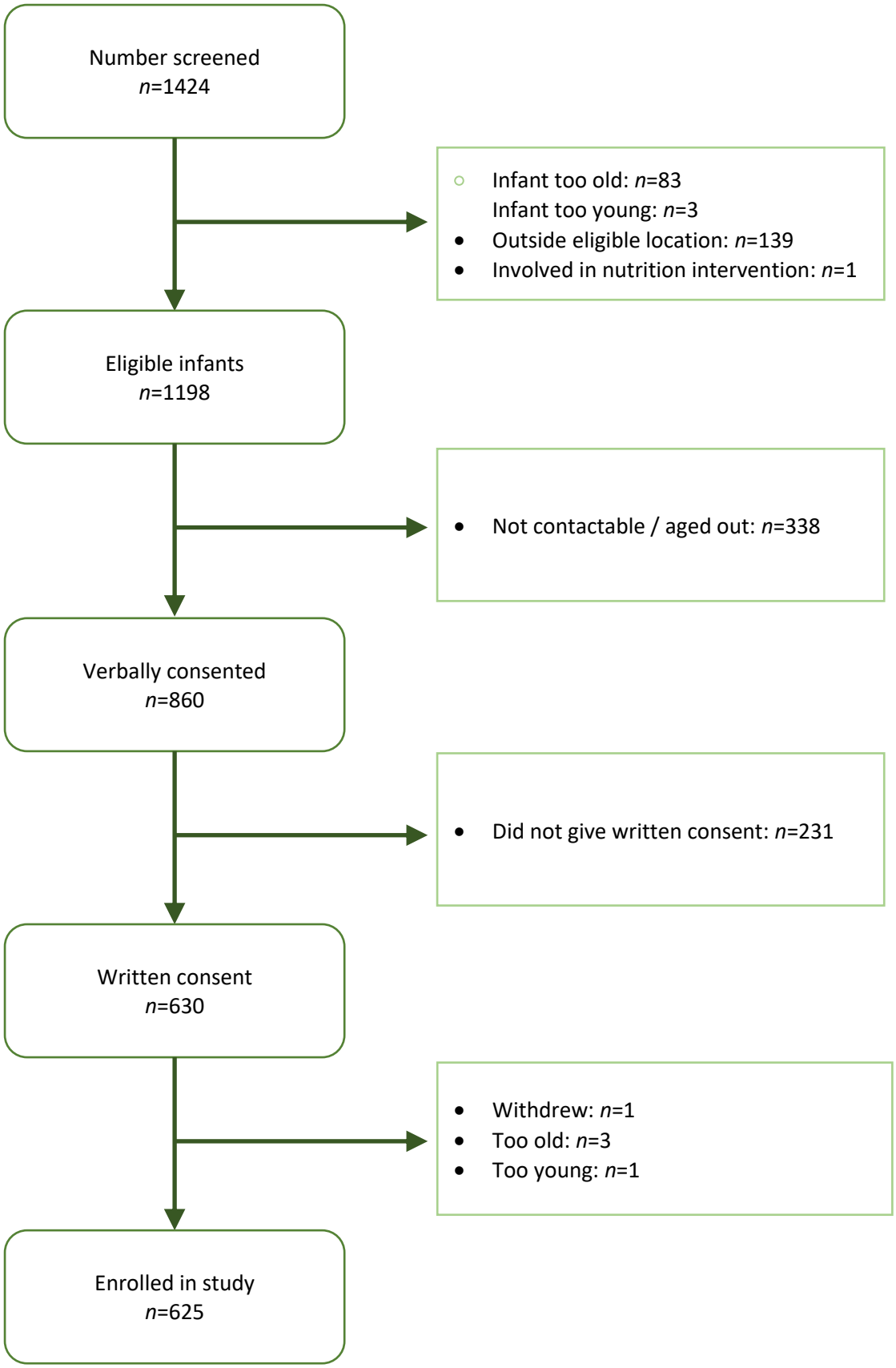
I have given a verbal explanation of the research project to the participant, and have answered the participant's questions about it.

I believe that the participant understands the study and has given informed consent to participate.

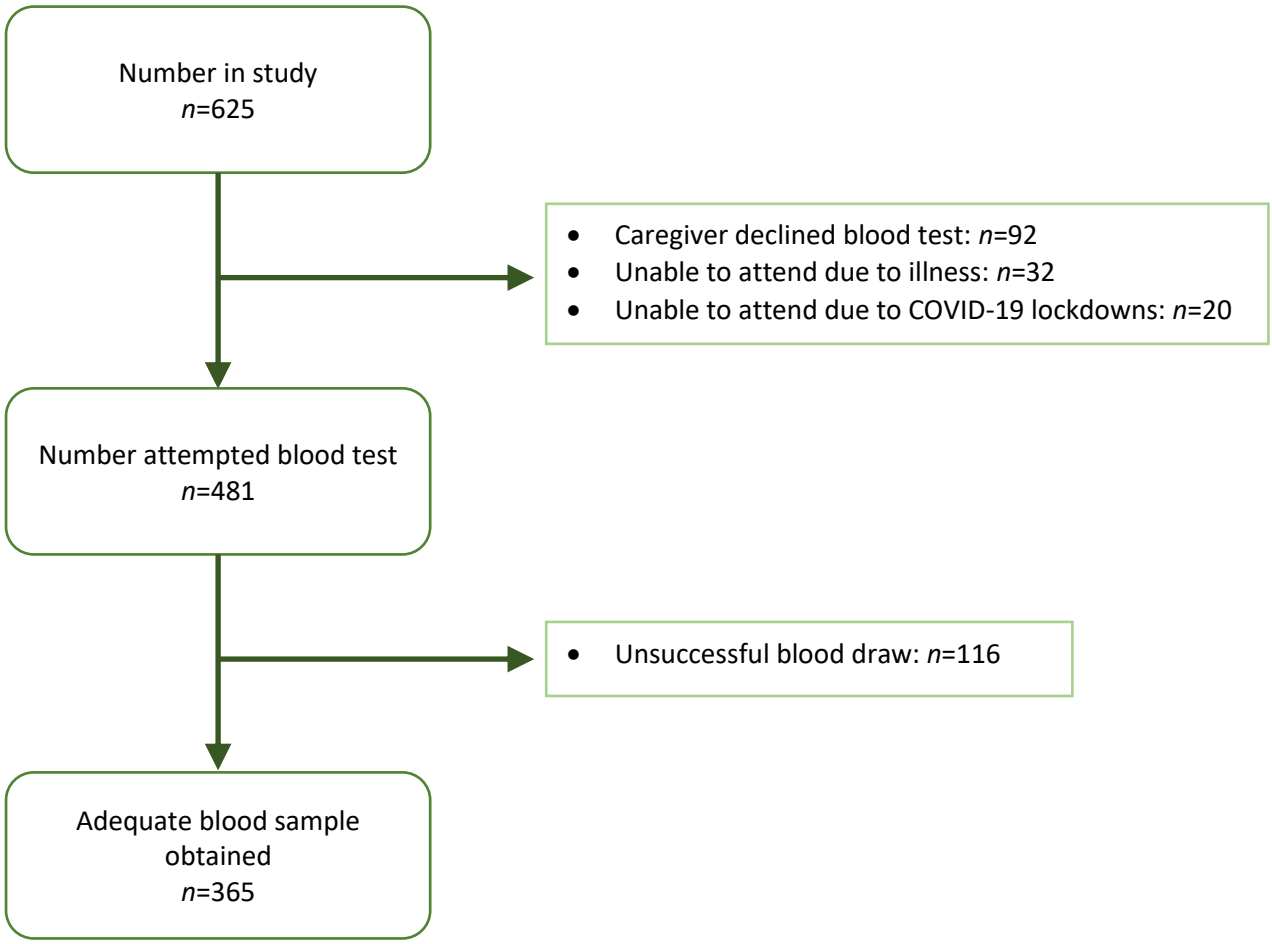
Researcher's name: _____

Signature: _____ Date: _____

**Appendix C:** Flowchart of Participants for First Foods New Zealand



**Appendix D:** Flowchart of Participants and Blood Tests



## **Appendix E:** Infant and Caregiver Ethnicity

**Table E1.** Classification of ethnicity in infants and caregivers (*n*=625)

| **Ethnicity groups** | **Reported ethnicities** | **Infant** | **Caregiver** |
|---|---|---|---|
| *Ethnicity Level 1: total response* | | **n (%)** | **n (%)** |
| NZ European | | 494 (79.0) | 448 (71.7) |
| Māori | | 131 (21.0) | 85 (13.6) |
| Pacific | Samoan, Cook Island Māori, Tongan, Niuean, Tuvaluan, Tokelauan, Rotuman, Papua New Guinea, Kiribati, Hawaiian, Fijian | 82 (13.1) | 48 (7.7) |
| Southeast Asian | Vietnamese, Thai, Malaysian, Indonesian, Filipino, Burmese | 28 (4.5) | 27 (4.3) |
| East Asian | Chinese, Taiwanese, Korean, Japanese, Asian | 44 (7.0) | 39 (6.2) |
| South Asian | Indian, Sri Lankan, South African Indian, Pakistani, Fijian Indian | 30 (4.8) | 25 (4.0) |
| West Asian | Syrian, Middle Eastern, Lebanese | 4 (0.6) | 4 (0.6) |
| European | Swiss, Scottish, Russian, Romanian, Lithuanian, Irish, Hungarian, German, French, English, Dutch, Danish, British, European, Croatian, Slovenian, Polish | 48 (7.7) | 40 (6.4) |
| Other European | South African, Canadian, Australian, American | 14 (2.2) | 17 (2.7) |
| African | African, Ethiopian, Zimbabwean | 3 (0.5) | 2 (0.3) |
| South American | Peruvian, Nepalese, Latin American, Brazil, Argentinian, South American | 12 (1.9) | 11 (1.8) |
| Other | Native American, Aboriginal | 2 (0.3) | 0 |
| *Ethnicity Level 2: total response* | | | |
| NZ European | | 494 (79.0) | 448 (71.7) |
| Māori | | 131 (21.0) | 85 (13.6) |
| Pacific | | 82 (13.1) | 48 (7.7) |
| Southeast Asian | | 28 (4.5) | 27 (4.3) |
| East Asian | | 44 (7.0) | 39 (6.2) |
| South Asian | | 30 (4.8) | 25 (4.0) |
| Other European | European & Other European | 61 (9.8) | 57 (9.1) |
| Others | Other, West Asian, African, South American | 21 (3.4) | 17 (2.7) |
| *Ethnicity Level 2: prioritised (as listed)* | | | |
| Māori | | 131 (21.0) | 85 (13.6) |
| Pacific | | 44 (7.0) | 32 (5.1) |
| East Asian | | 40 (6.4) | 35 (5.6) |
| South Asian | | 24 (3.8) | 23 (3.7) |
| Southeast Asian | | 26 (4.2) | 26 (4.2) |
| Others | | 16 (2.6) | 15 (2.4) |
| Other European | | 34 (5.4) | 50 (8.0) |
| NZ European | | 310 (49.6) | 359 (57.4) |
| *Ethnicity Level 3: prioritised (as listed)* | | | |
| Māori | | 131 (21.0) | 85 (13.6) |
| Pacific | | 44 (7.0) | 32 (5.1) |
| Asian | Southeast Asian, South Asian, East Asian | 90 (14.4) | 84 (13.4) |
| Others | | 16 (2.6) | 15 (2.4) |
| European | NZ European, European, Other European | 344 (55.0) | 409 (65.4) |

**Table E2.** Self-identified infant ethnicity (*n*=625)

| **Self-identified ethnicity** | ***n*** | **Level 1 ethnicity group** |
|---|---|---|
| New Zealand European | 494 | NZE |
| Māori | 131 | Māori |
| Samoan | 44 | Pacific |
| Cook Island Māori | 24 | Pacific |
| Tongan | 22 | Pacific |
| Niuean | 9 | Pacific |
| Chinese | 31 | East Asian |
| Indian | 22 | South Asian |
| Vietnamese | 2 | Southeast Asian |
| Tuvaluan | 1 | Pacific |
| Tokelauan | 1 | Pacific |
| Thai | 4 | Southeast Asian |
| Taiwanese | 1 | East Asian |
| Syrian | 1 | West Asian |
| Swiss | 2 | European |
| Sri Lankan | 4 | South Asian |
| South American | 1 | South American |
| South African Indian | 1 | South Asian |
| South African | 5 | Other European |
| Scottish | 1 | European |
| Russian | 2 | European |
| Rotuman | 1 | Pacific |
| Romanian | 2 | European |
| Peruvian | 1 | South American |
| Papua New Guinea | 1 | Pacific |
| Pakistani | 2 | South Asian |
| Nepalese | 1 | South American |
| Native American | 1 | Other |
| Middle Eastern | 2 | West Asian |
| Malaysian | 1 | Southeast Asian |
| Lithuanian | 1 | European |
| Lebanese | 1 | West Asian |
| Latin American | 6 | South American |
| Korean | 3 | East Asian |
| Kiribati | 1 | Pacific |
| Japanese | 2 | East Asian |
| Irish | 5 | European |
| Indonesian | 1 | Southeast Asian |
| Hungarian | 1 | European |
| Hawaiian | 1 | Pacific |
| German | 5 | European |
| French | 1 | European |
| Filipino | 20 | Southeast Asian |
| Fijian Indian | 1 | South Asian |
| Fijian | 4 | Pacific |
| European | 7 | European |
| Ethiopian | 2 | African |
| English | 2 | European |

| **Self-identified ethnicity** | ***n*** | **Level 1 ethnicity group** |
|---|---|---|
| Dutch | 6 | European |
| Danish | 1 | European |
| Canada | 1 | Other European |
| Burmese | 1 | Southeast Asian |
| British | 13 | European |
| Brazil | 2 | South American |
| Australian | 4 | Other European |
| Asian | 8 | East Asian |
| Argentinian | 1 | South American |
| American | 4 | Other European |
| African | 1 | African |
| Aboriginal | 1 | Other |

**Table E3.** Self-identified caregiver ethnicity (*n*=625)

| Self-identified ethnicity | n | Level 1 ethnicity group |
|---|---|---|
| New Zealand European | 448 | NZE |
| Māori | 85 | Māori |
| Samoan | 20 | Pacific |
| Cook Island Māori | 18 | Pacific |
| Tongan | 11 | Pacific |
| Niuean | 4 | Pacific |
| Chinese | 29 | East Asian |
| Indian | 20 | South Asian |
| Zimbabwean | 1 | African |
| Vietnamese | 3 | Southeast Asian |
| Tuvaluan | 1 | Pacific |
| Tokelauan | 1 | Pacific |
| Thai | 3 | Southeast Asian |
| Taiwanese | 1 | East Asian |
| Syrian | 1 | West Asian |
| Swiss | 3 | European |
| Sri Lankan | 2 | South Asian |
| South American | 1 | South American |
| South African | 6 | Other European |
| Slovenian | 1 | European |
| Scottish | 1 | European |
| Russian | 2 | European |
| Romanian | 1 | European |
| Polish | 1 | European |
| Peruvian | 1 | South American |
| Papua New Guinea | 1 | Pacific |
| Pakistani | 1 | South Asian |
| Middle Eastern | 2 | West Asian |
| Lithuanian | 1 | European |
| Lebanese | 1 | West Asian |
| Latin American | 6 | South American |
| Korean | 2 | East Asian |
| Kiribati | 1 | Pacific |
| Japanese | 1 | East Asian |
| Irish | 1 | European |
| Indonesian | 1 | Southeast Asian |
| Hungarian | 1 | European |
| Hawaiian | 1 | Pacific |
| German | 3 | European |
| French | 1 | European |
| Filipino | 20 | Southeast Asian |
| Fijian Indian | 2 | South Asian |
| Fijian | 2 | Pacific |
| European | 5 | European |
| English | 2 | European |
| Dutch | 5 | European |
| Croatian | 1 | European |
| Canada | 3 | Other European |

| Self-identified ethnicity | n | Level 1 ethnicity group |
|---|---|---|
| Burmese | 1 | Southeast Asian |
| British | 11 | European |
| Brazil | 3 | South American |
| Australian | 4 | Other European |
| Asian | 6 | East Asian |
| American | 5 | Other European |
| African | 1 | African |

**Appendix F:** Study Questionnaire

| **Question** | **Answer options** |
|---|---|
| How are you related to this baby? | Radio, required<br>1 Mother<br>2 Father<br>3 Grandparent<br>4 Guardian<br>5 Other (please state) |
| What is your date of birth? | Text (dd/mm/yyyy) |
| What is today's date? | Text (dd/mm/yyyy) |
| Was your baby born pre-term or term? | Radio<br>1 Preterm (less than 37 weeks gestation)<br>2 Term (37 weeks gestation or older)<br>3 I don't know |
| Is your baby a boy or a girl? | Radio<br>1 Girl<br>2 Boy<br>3 I would rather not say |
| Which ethnic group does your child belong to? Select all that apply to your child. | Checkbox<br>1 NZ European<br>2 Māori<br>3 Samoan<br>4 Cook Island Māori<br>5 Tongan<br>6 Niuean<br>7 Chinese<br>8 Indian<br>9 Other, e.g., Dutch, Japanese, Tokelauan<br><br>If other, please enter ethnicity(s) |
| Is your child descended from a Māori (that is, did they have a Māori birth parent, grandparent, or great-grandparent, etc) | Radio<br>1 Yes<br>2 Don't know<br>3 No |
| How old was your baby when they first had anything to drink that wasn't breast milk? (e.g., infant formula, water, other liquids) | Radio<br>1 They didn't have any breast milk<br>2 Breast milk is the only drink they've had so far<br>3 1 month old<br>4 2 months old<br>5 3 months old<br>6 4 months old |

| | |
|---|---|
| | 7 5 months old<br>8 6 months old<br>9 7 months old<br>10 8 months old<br>11 9 months old<br>12 10 months old |
| Is baby still being breastfed? | Yes / No |
| If no, how old was your baby when they stopped being breastfed? | Radio<br>1 Less than 1 month old<br>2 1 month old<br>3 2 months old<br>4 3 months old<br>5 4 months old<br>6 5 months old<br>7 6 months old<br>8 7 months old<br>9 8 months old<br>10 9 months old<br>11 10 months old |
| Has your baby ever had infant formula? | Yes / No |
| If yes, how old was your baby when they first had infant formula? | Radio<br>1 Less than 1 month old<br>2 1 month old<br>3 2 months old<br>4 3 months old<br>5 4 months old<br>6 5 months old<br>7 6 months old<br>8 7 months old<br>9 8 months old<br>10 9 months old<br>11 10 months old |
| Does your baby still drink infant formula? | Yes / No |
| If no, how old was your baby when they stopped drinking infant formula? | Radio<br>1 Less than 1 month old<br>2 1 month old<br>3 2 months old<br>4 3 months old<br>5 4 months old<br>6 5 months old<br>7 6 months old<br>8 7 months old |

| | |
|---|---|
| | 9 – 8 months old<br>10 – 9 months old<br>11 – 10 months old |
| Are you currently offering your baby any drinks other than water, breast milk, or infant formula? | Yes / No |
| What other drinks are you currently offering your baby? Please select all that apply. | Checkbox<br>1 – Cow's milk – undiluted<br>2 – Cow's milk – diluted<br>3 – Soy or other milk alternative<br>4 – Tea<br>5 – Juice or fruit drink<br>6 – Other (please specify) |
| How old was your baby when they first had solid foods? (solid foods are anything that isn't breast milk, infant formula, or other drinks. They don't have to be thick – some solids are runny, and some have drinks like breast milk or infant formula mixed into them) | Radio<br>1 – They haven't had solids yet<br>2 – Less than 1 month old<br>3 – 1 month old<br>4 – 2 months old<br>5 – 3 months old<br>6 – 4 months old<br>7 – 5 months old<br>8 – 6 months old<br>9 – 7 months old<br>10 – 8 months old<br>11 – 9 months old<br>12 – 10 months old |
| What was the first food you gave your baby? | Free text |
| What texture was the first food you gave your baby? | Radio<br>1 – Pureed<br>2 – Mashed<br>3 – Chopped<br>4 – Finger food<br>5 – Other |
| How was your baby fed when they first started eating solids? | Radio<br>1 – Spoon fed by an adult<br>2 – Mostly spoon fed by an adult, some baby feeding themselves<br>3 – About half spoon fed by an adult and half baby feeding themselves<br>4 – Mostly baby feeding themselves, some spoon feeding by an adult<br>5 – Baby feeding themselves |
| How was your baby fed at around 6 months of age? | Radio<br>1 – Spoon fed by an adult |

| | |
|---|---|
| | 2 Mostly spoon fed by an adult, some baby feeding themselves<br>3 About half spoon fed by an adult and half baby feeding themselves<br>4 Mostly baby feeding themselves, some spoon feeding by an adult<br>5 Baby feeding themselves |
| How is your baby being fed solids now? | Radio<br>1 Spoon fed by an adult<br>2 Mostly spoon fed by an adult, some baby feeding themselves<br>3 About half spoon fed by an adult and half baby feeding themselves<br>4 Mostly baby feeding themselves, some spoon feeding by an adult<br>5 Baby feeding themselves |
| Have you ever used baby-led weaning with your baby? | Radio<br>1 I don't know what baby-led weaning is<br>2 Yes, we have followed baby-led weaning most or all of the time<br>3 Yes, we have followed baby-led weaning some of the time<br>4 Yes, we tried baby-led weaning but we stopped<br>5 No, we did not try baby-led weaning |
| If yes, how old was your baby when you first tried baby-led weaning? | Radio<br>1 Less than 1 month old<br>2 1 month old<br>3 2 months old<br>4 3 months old<br>5 4 months old<br>6 5 months old<br>7 6 months old<br>8 7 months old<br>9 8 months old<br>10 9 months old<br>11 10 months old |
| Have you used any traditional foods or practices when starting baby on solids? You might have chosen them because they are traditional to your culture, or your family or whānau. | Yes / No |
| If yes, please describe: | Free text |

<table>
<tr><td>Are there any traditional or cultural (family or whānau) foods you like your baby to have now?</td><td>Yes / No</td></tr>
<tr><td>If yes, please describe:</td><td>Free text</td></tr>
<tr><td>When your baby was around 6 months of age, did they eat bought baby rice cereal?</td><td>Radio<table><tr><td>1</td><td>Yes</td></tr><tr><td>2</td><td>No – I offered it but they wouldn’t eat it</td></tr><tr><td>3</td><td>No – I didn’t offer it because I don’t agree with my baby eating it</td></tr><tr><td>4</td><td>No – I didn’t offer it because I didn’t think it would be safe for my baby</td></tr><tr><td>5</td><td>No – I didn’t offer it because my baby hadn’t started eating solids yet</td></tr><tr><td>6</td><td>No – I didn’t offer it for another reason (free text to state answer)</td></tr></table></td></tr>
<tr><td>When your baby was around 6 months of age, did they eat red meat (like beef or lamb)?</td><td>Radio<table><tr><td>1</td><td>Yes</td></tr><tr><td>2</td><td>No – I offered it but they wouldn’t eat it</td></tr><tr><td>3</td><td>No – I didn’t offer it because I don’t agree with my baby eating it</td></tr><tr><td>4</td><td>No – I didn’t offer it because I didn’t think it would be safe for my baby</td></tr><tr><td>5</td><td>No – I didn’t offer it because my baby hadn’t started eating solids yet</td></tr><tr><td>6</td><td>No – I didn’t offer it for another reason (free text to state answer)</td></tr></table></td></tr>
<tr><td>Has your baby ever eaten food from a baby food pouch?</td><td>Yes / No</td></tr>
<tr><td>How often has your baby eaten from a ‘ready-to-eat’ baby food pouch in the past month? (i.e., pouches that are filled when you buy them)</td><td>Radio<table><tr><td>1</td><td>Never</td></tr><tr><td>2</td><td>More than once a day</td></tr><tr><td>3</td><td>Once a day</td></tr><tr><td>4</td><td>5–6 times a week</td></tr><tr><td>5</td><td>2–4 times a week</td></tr><tr><td>6</td><td>Once a week</td></tr><tr><td>7</td><td>2–3 times a month</td></tr><tr><td>8</td><td>Once a month</td></tr><tr><td>9</td><td>Less than once a month</td></tr></table></td></tr>
<tr><td>How often has your baby eaten from a ‘home-filled’ baby food pouch in the past month? (i.e., pouches that you have to put the food in at home)</td><td>Radio<table><tr><td>1</td><td>Never</td></tr><tr><td>2</td><td>More than once a day</td></tr><tr><td>3</td><td>Once a day</td></tr><tr><td>4</td><td>5–6 times a week</td></tr><tr><td>5</td><td>2–4 times a week</td></tr></table></td></tr>
</table>

| | |
|---|---|
| | 6 Once a week<br>7 2–3 times a month<br>8 Once a month<br>9 Less than once a month |
| When baby has food from a baby food pouch, how does baby get the food? | Radio<br>1 Always suck it straight from the pouch nozzle<br>2 Mostly suck it straight from the pouch nozzle, sometimes on a spoon<br>3 About half the time suck it straight from the pouch nozzle and half the time on a spoon<br>4 Mostly from a spoon, sometimes suck it straight from the pouch nozzle<br>5 Always on a spoon |
| When your baby first started eating solids, were they having food from a baby food pouch? | Yes / No |
| When your baby first started eating solids, how often would they have food from a 'ready-to-eat' baby food pouch? (i.e., pouches that are filled when you buy them) | Radio<br>1 Never<br>2 More than once a day<br>3 Once a day<br>4 5–6 times a week<br>5 2–4 times a week<br>6 Once a week<br>7 2–3 times a month<br>8 Once a month<br>9 Less than once a month |
| When your baby first started eating solids, how often would they have food from a 'home-filled baby food pouch? (i.e., pouches that you have to put the food in at home) | Radio<br>1 Never<br>2 More than once a day<br>3 Once a day<br>4 5–6 times a week<br>5 2–4 times a week<br>6 Once a week<br>7 2–3 times a month<br>8 Once a month<br>9 Less than once a month |
| When baby had food from a baby food pouch when they first started solids, how did baby get the food? | Radio<br>1 Always suck it straight from the pouch nozzle<br>2 Mostly suck it straight from the pouch nozzle, sometimes on a spoon<br>3 About half the time suck it straight from the pouch nozzle and half the time on a spoon |

<table>
<tr><td></td><td><table>
<tr><td>4</td><td>Mostly from a spoon, sometimes suck it straight from the pouch nozzle</td></tr>
<tr><td>5</td><td>Always on a spoon</td></tr>
</table></td></tr>
<tr><td>Was your baby eating food from a baby food pouch when they were around 6 months of age?</td><td>Yes/No</td></tr>
<tr><td>How often would they have food from a ‘ready-to-eat’ baby food pouch when they were around six months? (i.e., pouches that you have to put the food in at home)</td><td>Radio<table>
<tr><td>1</td><td>Never</td></tr>
<tr><td>2</td><td>More than once a day</td></tr>
<tr><td>3</td><td>Once a day</td></tr>
<tr><td>4</td><td>5–6 times a week</td></tr>
<tr><td>5</td><td>2–4 times a week</td></tr>
<tr><td>6</td><td>Once a week</td></tr>
<tr><td>7</td><td>2–3 times a month</td></tr>
<tr><td>8</td><td>Once a month</td></tr>
<tr><td>9</td><td>Less than once a month</td></tr>
</table></td></tr>
<tr><td>How often would they have food from a ‘home-filled’ baby food pouch when they were around six months? (i.e., pouches that you have to put the food in at home)</td><td>Radio<table>
<tr><td>1</td><td>Never</td></tr>
<tr><td>2</td><td>More than once a day</td></tr>
<tr><td>3</td><td>Once a day</td></tr>
<tr><td>4</td><td>5–6 times a week</td></tr>
<tr><td>5</td><td>2–4 times a week</td></tr>
<tr><td>6</td><td>Once a week</td></tr>
<tr><td>7</td><td>2–3 times a month</td></tr>
<tr><td>8</td><td>Once a month</td></tr>
<tr><td>9</td><td>Less than once a month</td></tr>
</table></td></tr>
<tr><td>When baby had food from a baby food pouch at around 6 months of age, how did baby get the food?</td><td>Radio<table>
<tr><td>1</td><td>Always suck it straight from the pouch nozzle</td></tr>
<tr><td>2</td><td>Mostly suck it straight from the pouch nozzle, sometimes on a spoon</td></tr>
<tr><td>3</td><td>About half the time suck it straight from the pouch nozzle and half the time on a spoon</td></tr>
<tr><td>4</td><td>Mostly from a spoon, sometimes suck it straight from the pouch nozzle</td></tr>
<tr><td>5</td><td>Always on a spoon</td></tr>
</table></td></tr>
<tr><td>What ethnic group do you belong to? Select all that apply to you.</td><td>Checkbox<table>
<tr><td>1</td><td>NZ European</td></tr>
<tr><td>2</td><td>Māori</td></tr>
<tr><td>3</td><td>Samoan</td></tr>
<tr><td>4</td><td>Cook Island Māori</td></tr>
<tr><td>5</td><td>Tongan</td></tr>
<tr><td>6</td><td>Niuean</td></tr>
<tr><td>7</td><td>Chinese</td></tr>
<tr><td>8</td><td>Indian</td></tr>
</table></td></tr>
</table>

| | |
|---|---|
| | 9 Other, e.g., Dutch, Japanese, Tokelauan<br>If other, please enter the ethnicity: (free text) |
| Are you descended from a Māori (that is, did you have a Māori birth parent, grandparent, or great-grandparent, etc)? | Radio<br>1 Yes<br>2 No<br>3 Don't know |
| Do you currently have paid employment? | Radio<br>1 No<br>2 Yes – part-time<br>3 Yes – full-time<br>4 Paid parental leave<br>5 Unpaid parental leave |
| What is the highest level of education you have completed? | Radio<br>1 School<br>2 Polytechnic or similar<br>3 University<br>4 Other (please state – free text) |
| How many children have you / the baby's mother given birth to (including this baby)? | Radio<br>1 One<br>2 Two<br>3 Three<br>4 Four or more |
| How many children usually (at least half the time) live in this household (including this baby)? | Radio<br>1 One<br>2 Two<br>3 Three<br>4 Four or more |
| How many adults usually live in your household (including yourself)? | Radio<br>1 One<br>2 Two<br>3 Three<br>4 Four or more |

**Appendix G:** Example blood test results letters

1. Example of a letter when there has been an abnormal result



ID = *participant id*

Caregiver name
Address

Date

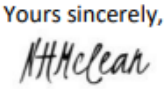
Dear *caregiver name* and *infant name*,

Thank you very much for taking part in the First Foods New Zealand (FFNZ) study and for completing the blood test component of the study. We have now received the results from this, and although most of the results from the test were as expected, one or more of the values were outside the expected reference range. This is a common occurrence in this age group so we have checked the results with our study paediatrician to see whether they are likely to be of any concern. He sees no reason for concern based on the information he has available. We have sent the results to *caregiver General Practitioner*, as provided on your Consent form for the study, so that the results are with your child's health records. Please do call your General Practitioner though if you have any concerns about *infant name's* wellness or growth to see whether you need to take them in for a visit.

Because we have referred the blood test results to your General Practitioner, we ask that you talk to them directly about gaining access to these results.

If you would like to discuss anything to do with the FFNZ blood test or the FFNZ study as a whole, please feel free to call us (contact details below). Thank you very much again for taking part in the FFNZ study.

Yours sincerely,



**Neve McLean**
PhD Candidate
FFNZ Study
Department of Human Nutrition
University of Otago
Phone 021 279 0553

**Associate Professor Anne-Louise Heath**
Co-Principal Investigator
FFNZ Study
Department of Human Nutrition
University of Otago
Phone (03) 479 8379

1. Example of a letter when there has been an expected result



*Caregiver Name*
*Caregiver Address*

*ID* = participant ID

Date

Dear *caregiver name and infant name*,

Thank you very much for your participation in the First Foods New Zealand study and for taking part in the blood test component of the study. We have reported *infant name's* blood test results on the back of this page. We are happy to say that they are all within the expected reference ranges.

If you would like to discuss anything to do with the blood test, or the study as a whole, please feel free to call us (contact details below). Thank you again for taking part in the First Foods New Zealand study.

Yours sincerely,

NHMcLean

**Neve McLean**
PhD student
First Foods New Zealand Study
Department of Human Nutrition
University of Otago
Phone 021 279 0553

**Associate Professor Anne-Louise Heath**
Co-Principal Investigator
First Foods New Zealand Study
Department of Human Nutrition
University of Otago
Phone (03) 479 8379

Tel 64 3 479 7959 • Fax 64 3 479 7958
Email human-nutrition@otago.ac.nz • Web http://www.otago.ac.nz/humannutrition/

**Blood test results**

| Measure of iron status | Your child's blood test result | Southern Community Laboratories reference range for children aged 6-11 months |
|---|---|---|
| Haemoglobin (g/L) | *value* | 105 - 140 |
| Plasma Ferritin (µg/L) | *value* | 15 - 150 |
| Haematocrit | *value* | 0.31 - 0.40 |

**Haemoglobin**
Haemoglobin is an iron-containing protein found in red blood cells. It is important for carrying oxygen throughout the body. The haemoglobin level is lower than the reference range when there isn't enough iron in the body.

**Plasma ferritin**
Plasma ferritin is a measure of how much iron the body has in its stores.

**Haematocrit**
Haematocrit is a measure of how many red blood cells there are and their size. The haematocrit is lower than the reference range when there isn't enough iron in the body.

*Note 1: As long as the haematocrit and haemoglobin results are inside the reference range it doesn't matter if they are at the higher or lower end of the range.*

*Note 2: If your child was sick at the time of the blood test it is possible that their true plasma ferritin concentration is lower than is shown above.*

**Appendix H:** Statement of contribution – Chapter 3

DRC 16

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA
UNIVERSITY OF NEW ZEALAND

GRADUATE RESEARCH SCHOOL

## STATEMENT OF CONTRIBUTION
## DOCTORATE WITH PUBLICATIONS/MANUSCRIPTS

We, the candidate and the candidate's Primary Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

| | |
|---|---|
| Name of candidate: | Maria Casale |
| Name/title of Primary Supervisor: | Professor Pamela von Hurst |
| In which chapter is the manuscript /published work: | 3 |

Please select one of the following three options:

○ The manuscript/published work is published or in press

- Please provide the full reference of the Research Output:

○ The manuscript is currently under review for publication – please indicate:

- The name of the journal:
- The percentage of the manuscript/published work that was contributed by the candidate:
- Describe the contribution that the candidate has made to the manuscript/published work:

◉ It is intended that the manuscript will be published, but it has not yet been submitted to a journal

| | |
|---|---|
| Candidate's Signature: | MCasale |
| Date: | 05-Apr-2023 |
| Primary Supervisor's Signature: | Pamelavh |
| Date: | 5/4/2023 |

This form should appear at the end of each thesis chapter/section/appendix submitted as a manuscript/ publication or collected as an appendix at the end of the thesis.

*GRS Version 5 – 13 December 2019*
*DRC 19/09/10*

**Appendix I:** Statement of contribution – Chapter 4

DRC 16

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA
UNIVERSITY OF NEW ZEALAND

GRADUATE RESEARCH SCHOOL

## STATEMENT OF CONTRIBUTION
## DOCTORATE WITH PUBLICATIONS/MANUSCRIPTS

We, the candidate and the candidate's Primary Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

| | |
|---|---|
| Name of candidate: | Maria Casale |
| Name/title of Primary Supervisor: | Professor Pamela von Hurst |
| In which chapter is the manuscript /published work: | 4 |

Please select one of the following three options:

○ The manuscript/published work is published or in press

- Please provide the full reference of the Research Output:

○ The manuscript is currently under review for publication – please indicate:

- The name of the journal:
- The percentage of the manuscript/published work that was contributed by the candidate:
- Describe the contribution that the candidate has made to the manuscript/published work:

◉ It is intended that the manuscript will be published, but it has not yet been submitted to a journal

| | |
|---|---|
| Candidate's Signature: | MCasale |
| Date: | 05-Apr-2023 |
| Primary Supervisor's Signature: | Pamlaw |
| Date: | 5/4/2023 |

This form should appear at the end of each thesis chapter/section/appendix submitted as a manuscript/ publication or collected as an appendix at the end of the thesis.

*GRS Version 5 – 13 December 2019*
*DRC 19/09/10*

**Appendix J:** Statement of contribution – Chapter 5

DRC 16

MASSEY UNIVERSITY
UNIVERSITY OF NEW ZEALAND
GRADUATE RESEARCH SCHOOL

## STATEMENT OF CONTRIBUTION
## DOCTORATE WITH PUBLICATIONS/MANUSCRIPTS

We, the candidate and the candidate's Primary Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

| | |
|---|---|
| Name of candidate: | Maria Casale |
| Name/title of Primary Supervisor: | Professor Pamela von Hurst |
| In which chapter is the manuscript /published work: | 5 |

Please select one of the following three options:

○ The manuscript/published work is published or in press

- Please provide the full reference of the Research Output:

○ The manuscript is currently under review for publication – please indicate:

- The name of the journal:
- The percentage of the manuscript/published work that was contributed by the candidate:
- Describe the contribution that the candidate has made to the manuscript/published work:

◉ It is intended that the manuscript will be published, but it has not yet been submitted to a journal

| | |
|---|---|
| Candidate's Signature: | MCasale |
| Date: | 05-Apr-2023 |
| Primary Supervisor's Signature: | Pamela H |
| Date: | 5/4/23 |

This form should appear at the end of each thesis chapter/section/appendix submitted as a manuscript/ publication or collected as an appendix at the end of the thesis.

*GRS Version 5 – 13 December 2019*
*DRC 19/09/10*