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Adverse reactions to food in New Zealand children aged 0–5 years

Christine Crooks, Rohan Ameratunga, Maia Brewerton, Michelle Torok, Stephen Buetow, Shannon Brothers, Clare Wall, Penny Jorgensen

Abstract

Aim The aim of this study was to describe parent/caregiver-reported adverse reactions to food in children aged 0–5 years in New Zealand.

Method A cross-sectional survey was undertaken in clinics conducted by the Royal New Zealand Plunket Society, which is the major healthcare provider for New Zealand's Well Child programme. Parents/caregivers of 110 (65%) children participated.

Results Of the 44 children who experienced an adverse reaction to food, only four were clinically evaluated and had undergone diagnostic testing. Two other children were hospitalised following systemic symptoms. Neither was tested for food allergy. 18 (16%) children had physician diagnosed eczema.

Conclusion Within the limitations of this small study, the data indicated adverse reactions to foods are a public health concern in New Zealand and may be under investigated even in children with severe symptoms. These children remain at increased risk of continued morbidity. Based on this preliminary study further research on food allergy in New Zealand is warranted.

Adverse reactions to food (AFR) in children are a source of increasing concern worldwide. Adverse reactions to foods are common and can be classified as either intolerance or food allergy (FA). Food intolerance is not mediated by the immune system and in general children are not at risk of severe reactions. In contrast, FA is an immunological adverse reaction to food, triggered by either an IgE- or a non-IgE-mediated immune mechanism.[1] IgE mediated FA is generally rapid in onset, occurring within minutes to two hours.[2]

The severity of IgE mediated symptoms varies from mild oral symptoms to life-threatening anaphylaxis which can include cutaneous, respiratory, ocular and gastrointestinal responses.[3] A retrospective, case-based Australian study identified adverse reactions to foods as the major cause of anaphylaxis in children presenting to an emergency department.[4] Non-IgE-mediated FA symptoms typically involve sub-acute or chronic symptoms isolated to the gastrointestinal tract.[2]

Modern diets have become increasingly diversified with international migration and the manufacture of novel foods.[5] Changes in dietary exposure have resulted in changing FA patterns with increasing sensitisation to a growing variety of foods.[5] Recent data further suggest ethnic-specific FA patterns, which may be a result of culture-bound dietary intake.[6–8]

International data indicate that the incidence of FA is highest during the first 3 years of life, when approximately 5–6% of children may be affected.[9] The prevalence of FA has not been systematically studied in New Zealand (NZ).[10] From the 2006–07 NZ Health Survey, 120,600 children (14.1%) aged from birth to 14 years were estimated to have eczema that had been diagnosed by a physician.[11] FA is frequently associated with eczema in infants.[12]

We have commenced studies to determine the burden of AFR/FA in NZ. The aim of this study was to describe parental/caregiver reported adverse reactions to food experienced by children under 5 years of age attending Plunket clinics in NZ. All children in NZ are entitled to free well child services. Most children accessing the Well Child/ Tamariki Ora programme attend clinics conducted by the Royal New Zealand Plunket Society (Plunket Society). They can consult a Plunket nurse at a clinic or home at: 4 to 6 weeks, 6 to 9 weeks, 3 months, 5 months, 9 months, 15 months, 2 years, and 3 years of age.[13]

Methods

Design—The design of the survey was cross-sectional. An interviewer assisted questionnaire was developed and administered to families attending four Plunket clinics in the Auckland and Wellington regions. Three urban Plunket clinics were selected from areas known to have diverse ethnic populations.[14] A fourth more rural clinic (Tuakau) was also selected. The questionnaire included questions about AFR symptoms, recall of foods associated with a reaction, and demographic details. It was based on previous surveys of FA symptoms in children.[15,16] Acute onset AFR symptoms are likely to be IgE mediated.[3] It is more difficult to associate delayed reactions including eczema with food consumption without diagnostic testing.[3] The AFR symptoms in the questionnaire included those occurring within two hours of consumption[15] such as urticaria, angioedema, pruritus, gastrointestinal symptoms (including, diarrhoea, emesis, flatulence, offensive stool, reflux), rhinitis, asthma, and anaphylaxis. The presence of eczema reported by the parent/caregiver was also recorded.

Participant recruitment—Study materials (posters and information sheets) were made available in the clinic a month ahead of the planned interviews. The interview procedures were explained to the clinic nurses before the survey and their assistance was sought to recruit participants. All parents/caregivers attending the clinic with children aged up to 5 years were invited to participate following their consultation with the nurse. This included those scheduled for a visit as well as casual attendees. Siblings of children attending the clinics were also invited to join the study. Presentation of study material and subsequent recruitment were completed at the same Plunket visit. An explanation of the study was given by the interviewer, informed consent was obtained, and the survey was administered.

Data collection and analysis—Clinics with a separate interview room/area were selected to allow for privacy. These clinics were also known to have high attendance rates. The survey was performed from September to November 2009. The questionnaire was administered by the same interviewer who first established the child's age. The parent/caregiver was shown photographs of urticaria, eczema and angioedema and asked whether they had identified symptoms and signs listed in the questionnaire within two hours of their child consuming food (including breast milk).

Ethnicity was coded according to the subgroupings of the 2006/2007 NZ Health Survey Child Questionnaire[11] and using the total response method. This method records all ethnic groups with which the parent/caregiver identifies and so percentages exceed 100 percent.

A question about the ease of answering was included at the end of the questionnaire.

All data were entered into a Microsoft Excel spreadsheet and descriptive statistics are reported. Inferential statistics were performed to assess maternal (and child) differences between the groups of children who were reported to have reacted adversely to food or not.

Ethics approval—The study methods were reviewed by the Royal New Zealand Plunket Society and the Auckland District Health Board Research office. Ethics approval was provided by the Ministry of Health's Multi-Regional Ethics Committee (MEC09/47/EXP).

Results

Participation rate—152 children were scheduled for a visit with the nurse at the Plunket clinic on the days the interviews were performed. Parents/caregivers of 110 (65%) participated in the survey, comprising 97 (64%) of the booked appointments as well as an additional 13 (72%) casual attendees.

Table 1 compares socio-demographic characteristics of the respondent parents/caregivers (and their child) between the groups of children for whom adverse reactions to food were reported or not. No statistically significant differences at the 5% level were detected between these groups on any of the variables reported.

Table 1. Prevalence of parent/caregiver responses about adverse reactions to food by child and maternal characteristics

Variables	Total (n=110)	Adverse reactions [1] (n=44)	No adverse reactions (n=66)
	n (%)	n (%)	n (%)
Ethnicity of child[2]			
NZ European	73 (66.4)	28 (63.6)	45 (68.2)
Māori	21 (19.1)	8 (18.2)	13 (19.7)
Pacific peoples[3]	28 (25.5)	14 (31)	14 (21)
Asians[4]	25 (22.7)	12 (27.3)	16 (24.2)
Other	4 (3.6)	0	4 (6.1)
Age (months)			
Mean ± SD	16.7±15.2	18.0 ± 15.8	15.8± 14.8
Range	1.0 – 67.4	1.7 – 67.4	1.1 – 56.9
Gender			
Male	59 (53.6)	22 (50.0)	37 (56.1)
Female	51 (46.4)	22 (50.0)	29 (43.9)
Birth order of child			
1	62 (56.9)	23 (52.3)	39 (60.0)
2	26 (23.9)	14 (31.8)	12 (18.5)
3-7	19 (17.2)	5 (11.4)	13 (19.6)
Adopted	2 (1.8)	1 (2.3)	1 (1.5)
Birth weight (kg)			
Mean ± SD	3.5±0.6	3.53 ± 0.6	3.5±0.57
Range	1.4 - 5.5	1.38 – 4.50	1.9 – 5.5
Gestation (weeks)			
Mean ± SD	39.6±1.9	39.9±2.2	39.4±1.70
Range	28-42	28-42	34-42
Mother's highest educational level attained			
NCEA[5]secondary school qualification or equivalent	46 (41.8)	17 (38.6)	29 (43.9)
Post secondary school qualification	64 (58.2)	27 (61.4)	37 (56.1)

[1]Urticaria, angioedema, pruritus, gastrointestinal symptoms (including, diarrhoea, emesis, flatulence, offensive stool, reflux), rhinitis, asthma, and anaphylaxis, and worsening of eczema symptoms; [2]All ethnic groups with which the child identifies; [3]Pacific peoples: (Samoan, Cook Island Māori, Tongan, Niuean, Fijian, Tokelauen, Fijian/Indian/Zimbabwean); [4]Asians (Chinese, Indian, Japanese, Philippine, Iraqi); [5]Other (includes other European); [6]National Certificate of Educational Achievement.

Foods reported by the parent/caregiver to be associated with adverse reactions— 29 different foods were associated with adverse reactions reported by the

parent/caregiver. These have been grouped according to the main allergen they contain in common (Table 2). For example the 'Dairy' group includes cow's milk, cows' milk formula, cheese, ice cream, yoghurt (contained fruit), and goats' milk formula.

Table 2. Parent/Caregiver-reported adverse reaction to a food by their young child, symptoms, diagnosis and ease of answering questionnaire

Variables	n=44	%
Food groups associated with reactions		
Dairy	27	61.4
Breast milk	18	40.9
Foods containing multiple allergens	15	34.1
Fruit/Vegetables	8	18.2
Egg	3	6.8
Crustacean	2	4.5
Tree nut	2	4.5
Soy	1	2.3
Could not identify food	5	11.4
Symptoms		
Angioedema	5	11.4
Eczema	33	75.0
Hay fever	2	4.5
Life-threatening reaction (vomiting and aspiration)	1	2.3
Pruritus	17	38.6
Gastrointestinal symptoms (including, diarrhoea, emesis, flatulence, offensive stool, reflux)	13	29.5
Urticaria	12	27.3
Who diagnosed food allergy		
Allergy specialist	3	6.8
Paediatrician	1	2.3
Not investigated	40	90.9
Diagnostic test (n=4)		
Specific IgE (RAST[1])	2	50.0
SPT[2]	2	50.0
Diagnosed allergies (n=4)		
Cat	2	50.0
Cow's milk	3	75.0
Dust mite	2	50.0
Egg	3	75.0
Peanut	4	100.0
Soy	3	75.0
How easy to answer were the questions asked you today? (n=89)		
Very Easy	72	80.9
Easy	17	19.1

[1]Radioallergosorbent testing; [2]Skin prick testing.

It was not possible to identify the nature of the allergenic constituent in the foods containing a mixture of potential allergens. For example, commercially produced Spaghetti Bolognese contains tomato, wheat, cheese and traces of egg, while home-made scrambled egg may contain egg, cows' milk or soy milk. In addition some parents were not able to recall the brand of food consumed; one child who reacted to a 'baby cereal' was subsequently diagnosed with allergies to soy, cows' milk and

peanut. Another parent noticed their child reacted to strawberry yoghurt but tolerated other dairy. It was also not possible to determine the nature of the allergen causing reactions to breast milk.

Symptoms—Forty percent (44/110) of the children were reported to have experienced an adverse reaction to food and 64% (28/44) of them experienced the reaction within 2 hours of the consumption. Two children were hospitalised after experiencing AFR symptoms.

Thirty three children were reported to have eczema. Eczema was diagnosed by a doctor (family physician/ general practitioner) in 18/110 (16%) children. In this small sample of children the percentage with doctor diagnosed eczema (14%) is higher than reported in the 2006/2007 New Zealand National Health survey.[11]

Eczema symptoms were reported to worsen within two hours of food consumption in 10 children. Two of these 10 children had physician diagnosed eczema.

Eczema symptoms were reported to improve in 6/6 children after their diet was altered. Parents/caregivers modified their child's diet by attempting to eliminate suspected food allergens. Diet changes were recommended by a physician for one of these six children. No allergy testing was recommended for this child.

Eczema symptoms improved in 3/3 children after their mothers altered their diet while breastfeeding. Diet changes were recommended by a physician for two of these mothers. Again, no allergy testing was undertaken. No parent/caregiver in this study received advice from a dietitian; the reason for this was not investigated.

Diagnosis—Food allergy was investigated and diagnosed in only four (9%) of the 44 children whose parent/caregivers reported AFR symptoms and eczema. FA was not investigated in 40 (91%) of the 44 children whose parent caregiver reported AFR symptoms, including the two who were hospitalised.

Discussion

Self-reported FA has become more frequent in developed countries in recent years.[17] [18] The reasons for the increase are still poorly understood.[19] There have been no previous community based studies of AFR/FA in New Zealand, yet anecdotal evidence suggests that AFR/FA is a problem in the community, which is not being addressed. Patients calling the Allergy New Zealand helpline and those on internet forums indicate they are uncertain where to seek further assistance.[20]

The health status of children attending Plunket clinics has been investigated previously,[21] however there are some limitations to this approach. Attendance at Plunket clinics wane after second and subsequent pregnancies, therefore it will not be possible to measure the occurrence of AFR in younger siblings by this method. Plunket nurses report transportation and language barriers are problems which impede clinic attendance and will further affect selection bias. Plunket data of attendance at the clinics in this study indicated 30-80% of contacts are visited at home. Future surveys of AFR in children seen by Plunket nurses require procedures to include those visited at home.

This study had a 65% participation rate. It is uncertain whether the children who did not participate in the study had a similar rate of food related symptoms. If

parent/caregivers of the children who experienced FA symptoms were more likely to participate in the study, surveys of this nature may overestimate the prevalence of AFR in Plunket attendees.

Parents/caregivers may inaccurately recall the food related to an adverse reaction, since FA affects multiple organ systems including the gut, skin, respiratory tract, and causes anaphylaxis, while some IgE mediated reactions are delayed, particularly eczema. Therefore parental reporting of FA can be higher compared to that confirmed by food challenges (11.8% compared to 2.5 %).[22] This study was based on self-report (no clinical or lab confirmation). For this reason we limited the probable FA case definition to try to capture reactions most likely to be FA and we asked about reactions within 2 hours of food consumption.

Personal interview is a common method of collecting original data for epidemiological studies.[23] Interviewers may reduce errors by encouraging a better response rate. Alternatively errors may be introduced by their presence, manner, method of administration, or method of recording responses.[23] The interviewer for this study was an experienced dietary interviewer. The questionnaire achieved its purpose of collecting data about reported adverse reactions to food experienced within two hours of consumption and about the presence of eczema. Most (80.9%) parents/ caregivers found the questionnaire very easy to complete and the remaining (19.1%) found it easy to complete. However, the ability of the questionnaire to capture the presence of food allergy has not yet been assessed. Clinical and laboratory validation is planned for future research. .

Despite these limitations, our preliminary data suggest that AFR symptoms are a public health concern in NZ. Given that 28/110 (25.5%) children experienced AFR within two hours of food consumption and another 10/110 (9%) had worsening eczema symptoms, FA may be at least as common as reported overseas.[24] We cannot however validate this inference because no allergy testing or food challenges were undertaken in this study.

Interestingly 18/44 (40%) children experienced an adverse reaction after consuming breast milk. This study was not designed to investigate the association of maternal dietary intake during lactation with FA symptoms. The benefits of dietary restriction during breastfeeding are still unknown as studies to date have been limited by their small size and methodological considerations.[25-27] However the data suggests the effect of maternal diet in lactation on the development of food allergy requires further investigation.

Parents may have difficulty identifying the cause of an adverse reaction as 5/44 (11.4%) could not associate reactions with a food. Alternatively it is possible that food was not the cause. Clinical assessment and diagnostic testing are required to prevent ongoing morbidity.

Our most important finding is that only 4/44 (11%) of children with reported AFR symptoms appear to have been clinically evaluated and undergone diagnostic testing. All four children were found to be sensitised to food allergens. Clinical evaluation and diagnostic testing are crucial in identifying food allergen(s). Children may be allergic to multiple foods.[7] For example, one child reacted to a commercial baby cereal and upon clinical evaluation had a positive skin prick test to egg and a positive specific

IgE results to egg and peanut. Without testing, the first manifestation of peanut allergy may have been anaphylaxis.

It is also of concern that two children were admitted to hospital with probable systemic allergic reactions to food and yet no testing was undertaken to identify food allergy. If these children have food allergies, they remain at risk for continued and possibly severe reactions.

Lack of clinical consultation and laboratory confirmation of FA can also result in unnecessary elimination diets, which can pose nutritional sequelae for the children.[28] [29] Unsupervised avoidance diets, when followed by breastfeeding mothers is also a risk for nutrient deficiency for both mother and infant.[30] Failure to thrive is commonly seen in children experiencing FA as a result of multiple foods being removed from their diet.[31] It is very important for children with a defined food allergy to consume a nutritionally restricted but balanced diet and for their growth to be monitored. [28 30]

In this study 6% (6/89) parent/caregivers reported modifying their children's diets without advice from a physician or dietitian. Furthermore, three breastfeeding mothers in this study eliminated foods from their own diet to improve their infants' symptoms. Moreover, none of the families including the four children diagnosed with FA had received advice from a dietitian. The study did not investigate the reason for this. The inclusion of a paediatric dietitian in the health professional team managing their FA should be an integral part of their treatment.[31]

This study also confirms previous international data that AFR symptoms are not confined to children of European origin.[6] Our study has shown that AFR symptoms occur in ethnic minorities as well as European children. In this study, the frequency of AFR was 8/21 (38%) in Māori children. None of the Māori children with reported AFR symptoms was clinically investigated. Māori have on average the worst health status of any ethnic group in New Zealand.[32] For example Māori experience greater morbidity associated with asthma.[32] Our study suggests AFR could be a significant source of morbidity in Māori children and further investigation is required to reduce health disparities.

The frequency of children with reported AFR was also high for Pacific peoples (Samoan, Cook Island Maori, Tongan, Niuean) (12/24, 50%), and Asian ethnicities (Chinese, Indian) (10/20, 50%). These data suggest FA impacts on the health status of all ethnic groups in NZ.

Among the children in this study 16% (18/110) had physician diagnosed eczema compared to a previous New Zealand study whose authors reported a physician diagnosed prevalence of 14% among children aged 0 to 14 years.[11] Our finding may be of concern because up to 40% of children with eczema can have a food allergic trigger.[12] However our observation is limited by the small sample size and the possibility of selection bias since only 65% of possible children participated.

More than half of the children (18/33, 54.5%) with eczema were being treated by a physician. Nearly a third (5/18, 27.8%) of these children continued to have problems with their skin despite being prescribed topical therapy. One possible explanation for this observation is undiagnosed FA. Without testing, allergic triggers for eczema could not be identified in these participants. The results from this study suggest that further investigation of FA as a cause of eczema is warranted.

In conclusion, results from this preliminary study suggest that AFR may be an important public health concern for diverse ethnic groups in NZ. Lack of medical assessment and diagnostic testing may place a substantial proportion of children with AFR symptoms at continued risk for reactions. Further development and validation of the tools to research AFR in New Zealand is required. There is an urgent need to investigate the epidemiology, diagnosis, and prevention of FA in New Zealand to reduce morbidity, improve child health, and reduce the burden to health costs.

Competing interests: None.

Author information: Christine Crooks, Post-Doctoral Research Fellow, Department Virology and Immunology, LabPlus, Auckland Hospital, Auckland District Health Board, Auckland; Rohan Ameratunga, Adult and Paediatric Allergy Specialist/Clinical Immunologist, Department Virology and Immunology, LabPlus, Auckland Hospital, Auckland District Health Board, Auckland; Maia Brewerton, Immunology Registrar, Wellington Hospital, Capital and Coast District Health Board, Wellington; Michelle Torok, USA based Consultant Epidemiologist Auckland; Stephen Buetow, General Practice and Primary Health Care, School of Population Health, University of Auckland, Auckland; Shannon Brothers, Paediatrician, Starship Hospital, Auckland District Health Board, Auckland; Clare Wall, Senior Lecturer, Faculty of Medical and Health and Sciences, University of Auckland, Auckland; Penny Jorgensen, Chief Executive Officer, Allergy New Zealand, Auckland

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Correspondence: Associate Professor Rohan Ameratunga, Adult and Paediatric Allergy Specialist/Clinical Immunologist, Department of Virology and Immunology, LabPlus, Auckland Hospital, Park Rd, Grafton, Auckland 1023, New Zealand. Fax: +64 (0)9 3072826; email: rohana@adhb.govt.nz

References:

1. Johansson S, Bieber T, Dahl R, et al. Revised nomenclature for allergy for global use: Report of the Nomenclature Review Committee of the World Allergy Organization, October 2003. J Allergy Clin Immunol 2004;113:832-836.
2. Jones S, Burks W. The Spectrum of Allergic Reactions to Foods. In: Metcalfe D, Sampson H, Simon R, editors. Food Allergy: Adverse Reactions to Foods and Food Additives. 4th ed: Blackwell Publishing, 2008:101-109.
3. Sicherer SH, Sampson HA. Food allergy. J Allergy Clin Immunol 2010;125(2 Suppl 2):S116-25.
4. Braganza S, Acworth J, McKinnon D, et al. Paediatric emergency department anaphylaxis: different patterns from adults. Arch Dis Child 2005;91:159-163.
5. Warner JO. The changing face of paediatric allergy. Pediatr Allergy Immunol 2009;20(1):1-2.
6. Chiang WC, Kidon MI, Liew WK, et al. The changing face of hypersensitivity in an Asian community. Clin Exp Allergy 2007;37:1055-1061.
7. Kanagawa Y, Matsumoto S, Koike S, Imamura T. Association analysis of food allergens. Pediatr Allergy Immunol 2009;20(4):347-52.

8. Oh JW, Pyun BY, Choung JT, et al. Epidemiological change of atopic dermatitis and food allergy in school-aged children in Korea between 1995 and 2000. J Korean Med Sci 2004;19(5):716-23.
9. Venter C, Pereira B, Voigt K, et al. Prevalence and cumulative incidence of food hypersensitivity in the first 3 years of life. Allergy 2008;63(3):354-359.
10. Crooks C, Ameratunga R, Simmons G, et al. The changing epidemiology of food allergy--implications for New Zealand. N Z Med J 2008;121(1271):74-82.
11. McKernan S. A Portrait of Health. Key Results of the 2006/07 New Zealand Health Survey. Wellington: Ministry of Health, New Zealand, 2008:119-180
12. Hill DJ, Hosking CS, de Benedictis FM, et al. Confirmation of the association between high levels of immunoglobulin E food sensitization and eczema in infancy: an international study. Clin Exp Allergy 2008;38(1):161-8.
13. Plunket Visits, Providing Support. The Royal New Zealand Plunket Society Inc. Wellington, New Zealand, 2010. http://www.plunket.org.nz/plunket-you/what-we-offer/plunket-visits/providing-support/
14. Bascand G. Profile of New Zealander Responses, Ethnicity Question: 2006 Census: Statistics New Zealand, Tatauranga Aotearoa, Wellington, New Zealand, 2007.
15. Du Toit G, Katz Y, Sasieni P, et al. Early consumption of peanuts in infancy is associated with a low prevalence of peanut allergy. J Allergy Clin Immunol 2008;122(5):984-991.
16. Venter C, Pereira B, Voigt K, et al. Factors associated with maternal dietary intake, feeding and weaning practices, and the development of food hypersensitivity in the infant. Pediatr Allergy Immunol 2009.
17. Branum AM, Lukacs SL. Food allergy among children in the United States. Pediatrics 2009;124(6):1549-55.
18. Gerez IF, Shek LP, Chng HH, Lee BW. Diagnostic tests for food allergy. Singapore Med J;51(1):4-9.
19. Lack G. Epidemiologic risks for food allergy. J Allergy Clin Immunol 2008;121(6):1331-6.
20. Jorgensen P. Patients calling Allergy NZ helpline. In: Ameratunga R, editor, 2010.
21. Mitchell EA, Subramaniam K, Blackburn J, Shooter D. Plastic wrapping of cot mattresses: results from a pilot study. N Z Med J 2000;113(1115):326-7.
22. Venter C, Pereira B, Grundy J, et al. Prevalence of sensitization reported and objectively assessed food hypersensitivity amongst six-year-old children: A population-based study. Pediatr Allergy Immunol 2006;17(5):356-363.
23. White E, Armstrong B, Rodolfo S. Principles of Exposure Measurement in Epidemiology. 2nd ed. Oxford: Oxford University Press, 2008.
24. Rona R, Keil T, Summers C, et al. The prevalence of food allergy: A meta-analysis. J Allergy Clin Immunol 2007;120(3):638-646.
25. Cant AJ, Bailes JA, Marsden RA, Hewitt D. Effect of maternal dietary exclusion on breast fed infants with eczema: two controlled studies. Br Med J (Clin Res Ed) 1986;293(6541):231-3.
26. Lovegrove JA, Osman DL, Morgan JB, Hampton SM. Transfer of cow's milk beta-lactoglobulin to human serum after a milk load: a pilot study. Gut 1993;34(2):203-7.
27. Terracciano L, Bouygue GR, Sarratud T, et al. Impact of dietary regimen on the duration of cow's milk allergy: a random allocation study. Clin Exp Allergy 2010;40(4):637-42.
28. Mofidi S. Nutritional Management of Pediatric Food Hypersensitivity. Pediatrics 2007;111:1645-1653.
29. Sicherer SH, Sampson HA. Food allergy. J Allergy Clin Immunol 2006;117(2):S470-S475.
30. Kirby M, Danner E. Nutritional deficiencies in children on restricted diets. Pediatr Clin North Am 2009;56(5):1085-103.
31. Noimark L, Cox HE. Nutritional problems related to food allergy in childhood. Pediatr Allergy Immunol 2008;19(2):188-195.

32. Blakey K, H W, Rippon R, Yeh L, et al. Tatau Kahukura: Maori Health Chart Book: Public Health Intelligence Monitoring Report No.5: Ministry of Health, Wellington, New Zealand, 2006.