Awanui Labs

THE SCOPE

Awanui Labs Northern - Pathology news

Introduction to the Scope May 2026

Kia ora and welcome to the May edition of the Scope

In this issue, we feature an article on familial hypercholesterolaemia (FH), one of the most common life-limiting metabolic disorders. FH remains under-recognised, and primary care teams are ideally placed to identify affected patients and their families, with the potential to significantly improve long-term outcomes. The article covers both adult and paediatric presentations and provides a practical reference for clinicians managing these patients. We also include a reminder that we are unable to accept handwritten additions to pre-printed PMS or electronic order forms. In addition, we regret to advise that rabies serology testing is no longer available through Awanui. As many users of this test will be aware, changes in travel patterns and vaccination practices over recent years have resulted in steadily increasing costs and declining test volumes. Despite considerable efforts throughout 2025 to establish an ongoing arrangement with a third-party provider, this was unfortunately not achievable, and the difficult decision was made to discontinue the service.

As always, thank you for your ongoing partnership and support. We welcome your feedback on both The Scope and our laboratory services, and encourage you to contact us with any comments or suggestions.

Handwritten amendments policy

From **May 29, 2026**, we will no longer accept handwritten amendments to printed PMS or printed e-order lab forms.

Rationale for the change:

- Patients have been adding tests to forms after issue
- Referrers have advised they do not wish to be responsible for tests they have not ordered
- Handwritten changes are difficult for collection staff to verify in busy centres

To support safe and appropriate practice, this change will:

- Ensure referrers are only responsible for tests they request
- Ensure testing is accurate and aligned with the request
- Reduce low-value or inappropriate testing
- Support efficient use of healthcare resources, including referrer time

Going forward:

Only electronically generated amendments will be accepted.

- Amendments must be made in PMS or e-order systems with additional and/or urgent tests digitally added.
- If the patient requires a paper copy of the form, the digitally updated forms must be printed before being given to the patient.

Please also do not include and tests in the clinical details section.

We appreciate your cooperation in supporting safe, appropriate, and efficient laboratory testing.

High LDL? Consider Familial hypercholesterolaemia (FH).

Summary

- A recent survey shows about 2.5% (one in forty) adult patients in the Northern region has an LDL ≥5.0 mmol/L - a level that conveys high CVD risk regardless of other risk factors
- Patients with such a high LDL, and others with high calculated high CVD risk (≥15% 5yr using Predict) are significantly undertreated, with only 28% of non-diabetic patients and 56% of diabetic patients taking a statin.
- Of those at high risk on statins only about 25% achieve an LDL <1.8 mmol/L and less than 10% achieve the internationally recommended target of <1.4 mmol/L.
- New comments by Awanui will highlight the importance of aggressive statin management in such patients. They will also highlight the importance of identifying those with a strong genetic cause, ***familial hypercholesterolaemia (FH).***
- FH is an autosomal dominant condition that is very common (1/250) but 90% of cases are undiagnosed. It predisposes to early and preventable cardiovascular disease.
- For the same LDL level FH patients are at much higher risk. However, widely used risk algorithms such as Predict do not adequately identify FH so high clinical suspicion is needed.
- Genetic confirmation is important if the patient meets criteria using a validated scoring system.
- Cascade screening of identified FH families is also pivotal to identify affected relatives.
- Improving awareness, systematic screening, and lifelong management of FH is strongly encouraged in recent international guidelines [1-4]
- FH causes high LDL. Raised triglycerides and low HDL are not a feature and have different usually acquired causes (metabolic syndrome, diabetes, obesity, sedentary lifestyle, high fat/carb diet).

GPs and primary care are well placed to identify and manage FH patients, and support family cascade screening. Funded genetic testing is available to GPs without need for specialist approval if patients meet 'probable' criteria – with information accessed here.

All children who meet clinical diagnostic criteria for FH (section 7) and those with a genetic diagnosis should be referred to the National Metabolic Service.
Do not refer FH adults/families to Clinical Genetics, who are not resourced.
Adult patients can be well managed in primary care with online and specialist advice, and referred if needed and accepted, according to local Health Pathways. Statins and ezetimibe are the cornerstone of treatment

A. Large numbers of patients with very high LDL levels in the Northern region.

The table below shows the approximate numbers of adults with very high lipid levels in the Auckland-Northland region, based on a very large recent survey.

	Age 18-39 yr	Age 40-79 yr
LDL ≥6.5 mmol/L	≈400	≈2,000
LDL 5.0 – 6.4 mmol/L	≈3,500	≈29,000
=est. FH cases	≈250	≈1250

Only about 5% of adults with LDL of 5.0-6.4 mmol/L and 10% with LDL ≥6.5 mmol/L were found to have a secondary cause.

International evidence shows about one in eight adult patients with LDL ≥6.5 mmol/L and 2-3% of patients with LDL 5.0-6.4 mmol/L have a strong underlying 'monogenic' cause - 'familial hypercholesterolaemia' ('FH'). The frequency is even higher in younger patients, especially with a family history. These patients are at especially high risk and most likely to benefit from intervention.

From this data there are estimated to be at least 1500 adult cases of FH in the Northern region with LDL ≥5.0 mmol/L, most currently undiagnosed and undertreated. There will also be others with LDL in the 4.1-4.9 mmol/L range (population frequency about 1-1.5%), including many children. However, these can only be identified from the large number with polygenic or acquired causes by a family history and other suspicious features.

A practice of 3000-4000 patients is likely to have 12-16 patients with FH, most undiagnosed.

B. What is familial hypercholesterolaemia (FH)?

1. Population Frequency, Diagnosis, and Treatment Rates

Familial hypercholesterolaemia (FH) is the most common life-limiting inherited metabolic disorder, with a very high population carrier frequency of one in 250, similar to type 1 diabetes. There are estimated to be over 20,000 FH carriers in New Zealand. It spans all ethnicities, but there is a higher prevalence in some ethnic groups such as South Africans, French Canadians and some migrant groups.

FH is a silent killer, with lifelong elevation of LDL causing a markedly increased risk of premature atherosclerotic cardiovascular disease. CVD risk in affected patients is 2-4 fold higher, and in young patients up to 17-fold higher, than unaffected siblings [1].

Over 90% of cases in most Western countries, including New Zealand, are undiagnosed or diagnosed too late, after a preventable cardiac event. Over 50% of untreated men have a coronary event by age 50 and 30% of women by age 60. Prevalence in patients with established ASCVD overall is around 7% (about 20-fold increased) but is likely even higher in early onset cases.

For any level of LDL the CVD risk of an FH patient is much higher than someone without the disorder, due to the increased lifetime LDL exposure. This is underestimated by traditional risk algorithms such as Predict, so a high level of clinical suspicion and aggressive treatment of diagnosed cases is important.

2. Causes and Genetic Basis

FH is an autosomal dominant disorder so carriers manifest the high LDL and heightened CVD risk. Half the family members are also affected, so one almost always diagnoses a family not just a patient.

The homozygous form (HoFH) is much rarer, occurring in about 1 in 160,000 to 300,000 individuals, and characterised by extremely high cholesterol and childhood presentation of vascular events.

The underlying basis of FH is one or more pathogenic variants in key genes involved in LDL uptake into cells, especially the liver. The main genetic defects occur in:

- ***LDLR*** – the LDL receptor (90% of cases)
- ***APOB*** – apolipoprotein B (apoB), the protein which binds LDL to its receptor (10%)
- ***PSCK9*** – a protease that regulates LDL receptor recycling (<1%)
- ***LDLRAP1*** – mutations can cause a recessive form (rare)

These mutations impair LDL clearance, leading to lifelong elevation of plasma LDL compared to age-matched controls, with accelerated atherosclerosis, and increased cardiovascular mortality if untreated.

3. Clinical Suspicion and Differentiation from Secondary or Polygenic Causes

FH is very different to the more common 'polygenic' hypercholesterolaemia, due to the cumulative small effects of variants in many genes and lifestyle. While there is overlap in polygenic causes, the degree of LDL elevation is typically less marked, the family history of early CVD is less prominent, and characteristic features such as xanthomas are lacking. *Note that other lipid abnormalities such as raised triglycerides or low HDL are not features of FH but may occur in FH patients for other reasons.*

The key to recognising FH is a high level of suspicion, particularly for:

- ***Chronically high LDL*** not explained by secondary/acquired causes.

-Secondary causes include ketogenic diet, hypothyroidism, cholestatic liver disease, nephrotic proteinuria, drugs (e.g. steroids, atypical antipsychotics, immunosuppressants, antiretrovirals). An LDL rise of 0.7-1 mmol/L is also common in women through the menopause.

-The higher the LDL the more likely FH is an explanation. With levels ≥8.5mmol/L it is very high, at 6.5-8.4 mmol/L the risk is at least 10-15%, at 5.0-6.4 it is 2-3%. Even in the range of 4.1-4.9 mmol/L it is 1-1.5% so other clues should be sought to identify FH patients from the many others with similar LDL levels.

- ***Early vascular events,*** especially if not explained by traditional risk factors

- ***Strong family history*** of early CVD events (especially MI), xanthomas or clearly high LDL

- ***Clinical features***

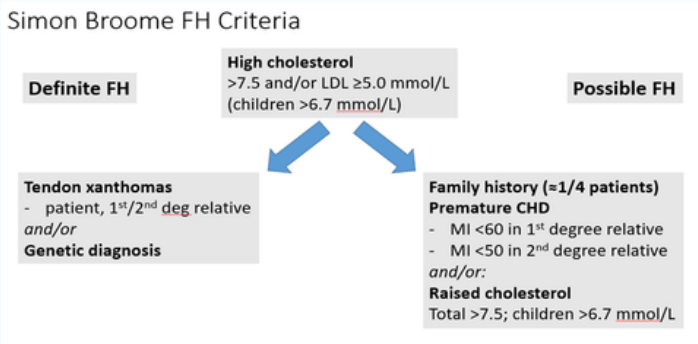
-Cholesterol deposits (xanthomas) causing thickened tendons (e.g. Achilles) or under skin.
-Arcus senilis and xanthelasma at a young age (before 45yr).
-Such features are highly specific but their absence does not exclude the disorder
-These features take time to develop so are often not present in children unless severe
-Note that coronary calcium scoring is unreliable as a guide to treatment in FH and patients should be aggressively treated with statins and ezetimibe regardless

Reasons to suspect possible FH: unexplained surprisingly high LDL, early vascular events in patient or close family, clinical features (tendons, eyes).

FH causes high LDL. High triglycerides and low HDL are not a feature and usually have a different set of acquired causes (commonly obesity, high fat/carb diet, poorly controlled diabetes, sedentary lifestyle, high alcohol intake)

4. Clinical Diagnostic Criteria for FH: Validated Scoring Systems

Clinical diagnosis is guided by a validated scoring system that combines cholesterol levels, personal and family history, and physical signs.
There are two main validated scoring systems. The more simple one, called the Simon-Broome criteria, was developed in the UK[6]. It can be used in both adults and children, but with different cholesterol and LDL thresholds. It can be a helpful screen, but is not formally used here. Suspected cases should be formally assessed using the Dutch Lipid Clinic Score (DLCS).



Dutch Lipid Clinic Network Score (DLCS)

The *scoring system used in adults in New Zealand* and Australia was developed in the Netherlands, and is called the ***DLCS score (Dutch Lipid Clinic Score).*** Formal assessment using the DLCS score is needed for genetic testing to be performed (see section 5, below). A score of 6 or more indicates 'probable' FH and is sufficient to proceed to publicly funded genetic testing. An online tool for quick assessment is available at www.athero.org.au/fh/calculator.
Note that the DLCS score cannot be used in children, and an alternative diagnostic method is used based on European criteria [7,8] (see section 7, FH in children).

Dutch Lipid Clinic Network Criteria Score (DLCNS score)	Score
Family History	
First degree relative with known premature coronary and/or vascular disease (men <55, women <60yr), **OR** First degree relative with LDL cholesterol >95th %ile for age and sex	1
First degree relative with tendon xanthomata and/or arcus senilis **OR** Children <18yr with LDL >95th percentile for age & sex	2
Patient clinical History	
Premature coronary artery disease (age cutoffs as above)	2
Premature cerebral or peripheral vascular disease (age cutoffs as above)	1
Physical Examination	
Tendon xanthomas	6
Arcus senilis at age <45 yrs (xanthelasma is not specific or counted)	4
LDL Cholesterol (mmol/L)	
≥ 8.5	8
6.5-8.4	5
5.0-6.4	3
4.0-4.9	1
DNA Analysis	
Pathogenic variant in the LDLR, APOB, PCSK9 or LDLRAP1 gene	8
Definite FH	8
Probable FH	6-7
Possible FH	3-5
Unlikely FH	<3

5. Genetic Testing is important

Genetic testing provides definitive confirmation in 60–80% of clinically diagnosed FH cases. Identifying a pathogenic variant is highly specific but not all FH phenotypic cases have an identifiable mutation. Importantly, a negative test does not always exclude FH, and aggressive treatment on a clinical basis should still proceed if LDL and family history clearly support the diagnosis [1,2].

Genetic testing is extremely helpful because it:

- clearly distinguishes monogenic from polygenic causes of high LDL
- provides additional prognostic risk beyond just LDL measurement, to focus treatment on whose who will benefit most
- provides patients a known cause of their high cholesterol and motivates them to pursue treatment
- enables more reliable cascade screening of close relatives, especially in children where the lipid phenotype may not be sufficiently distinctive [9]
- will likely facilitate approval for expensive lipid modifying treatments when these eventually become funded e.g. PCSK9 inhibitors

Studies in general practice show genetic testing is received positively by patients[10]. However, it is important to discuss the implications before obtaining informed consent from the patient or legal guardian.

Funded genetic testing for FH is performed in the Northern region if the DLCS score is 6 or more ('probable' FH). The LabPlus Test Guide (http://testguide.adhb.govt.nz/EGuide/) has a form under keyword 'hypercholesterolaemia'. The page can be accessed here. The form needs to be completed, imaged as a pdf, and emailed to chemicalpathologist@adhb.govt.nz. The form should be completed at the time the genetic test is made through usual lab request, as samples are not processed for testing until the DLCS form is received.

Funded genetic testing to confirm FH is available to primary care without need for specialist approval and is encouraged if the patient meets a DLCS score of ≥6. Requests are vetted and the approval form must be emailed to LabPlus chemicalpathologist@adhb.govt.nz. The form can be accessed here.

Evidence from international programs (e.g., the Dutch FH Register) shows cascade screening is highly cost-effective, increases detection at least several-fold and especially benefits young family members by preventing many years of high LDL exposure with potentially irreversible arterial damage.

The cascade screening process includes [11]:

1. Identifying an index case (proband) through clinical or genetic diagnosis.
2. Testing first-degree relatives (siblings, children, parents) with lipid panels ± genetic testing.
3. Extending screening to second-degree relatives where positive results are found.

Screening relatives should begin from age 5-10 years, or earlier if there is a family history of early MI.

Cascade screening—testing relatives of confirmed FH patients—is an efficient way to detect undiagnosed cases. Each first-degree relative has a 50% chance of inheriting the disorder.

Further resources and links to online help for cascade screening are available here.

7. FH in children

International guidelines recommend early detection of FH and higher levels of suspicion are needed. Early diagnosis reduces lifelong accelerated atherosclerosis and delays CVD events [1,8].Affected children already show evidence of increased carotid intima-media thickness by around age 10. Early treatment significantly slows rate of progression and clearly reduces long-term cardiovascular risk compared with untreated parents [12].

In children, lipid levels may not adequately identify FH carriers in a known family and up to 50% may be missed, hence while a high LDL is strongly suggestive, genetic testing is also important [1,9]

The US National Heart, Lung, and Blood Institute and American Academy of Pediatrics recommend universal lipid screening between ages 9–11, or earlier for those with a strong family history. The recent 2026 Guideline from the US AHA/ACC also recommends early detection of affected children[1]. Once identified their parents and other relatives can be tested (so-called 'reverse cascade screening').

There are initiatives worldwide to improve early detection, including national screening programs in some countries and advanced plans in others [1,8,13,14]. New Zealand currently has no national screening program for FH, but it is likely that screening will shortly become available in some states in Australia.

New EAS 2026 guidelines[17] state an LDL >4.5 mmol/L in a child is effectively diagnostic if not otherwise explained, while a lower level >3.5 mmol/L is also considered diagnostic in the context of a clear family history. Genetic testing may be appropriate in other cases above the 95 percentile of 3.5 mmol/L, but such cases should be discussed with the National Metabolic Service or Paediatric lipidologist.

Recommended formal diagnostic criteria (2026) for FH in children

(European Atherosclerosis Society approach, followed in Australia and New Zealand)

1. LDL >4.5 mmol/L (done twice) regardless of family history without secondary cause
2. LDL >3.5 mmol/L with
   a. family history of premature ASCVD, and/or
   b. high baseline cholesterol in a parent
3. LDL >3.0 mmol/L with confirmed pathogenic mutation in a parent

All children who meet diagnostic criteria above, and families with affected children under age 20yr, should be referred to the New Zealand National Metabolic Service. Those with affected parents but LDL ≤3.5 mmol/L should be discussed with the service; usually lipids should be repeated in 5 years.

High LDL? Consider Familial hypercholesterolaemia (FH)...*cont*

In addition to lifestyle changes, affected patients should usually be treated from age 8-10, and even earlier in more severe cases. Statin treatment is safe and well tolerated with no detectable effects on growth and development [1].

8. Managing FH

An FH diagnosis justifies lifelong lipid-lowering therapy starting as early as possible. The cornerstone of treatment in adults is high-intensity statin therapy. Children can start at lower doses. Statins have been shown to significantly lower ASCVD mortality when started early [12]. Ezetimibe provides additive LDL reduction of up to 25%.
For compliant patients on stable long-term treatment annual lipid measurement is usually sufficient.
GPs and practice staff are in an ideal position initiate and support cascade screening of close family members [15,16]. However, testing of wider family (e.g. cousins on an affected side when one parent is diagnosed) is limited by privacy, distance and limited resources. Enlisting family champion(s) is very helpful. Additional information and resources on cascade screening are available here.

*It is **not** necessary to refer new adult cases of FH. The majority can be effectively managed in primary care, with support from online resources and specialist advice if needed. While primary care can assist, families should be encouraged to take initiative in cascade screening.*

Consider specialist discussion, and referral if agreed in:

- Children with an established or strongly suspected diagnosis of FH. They should be referred for specialist assessment to the New Zealand National Metabolic service.
- Patients not achieving LDL goals despite maximum tolerated lipid lowering treatment
- Patients with clinical atherosclerotic cardiovascular disease for assessment (current regional referral guidelines)

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References:
1.Blumenthal RS, Morris PB, Gaudino M, et al. 2026ACC/AHA/AACVPR/ABC/ACPM/ ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the management of dyslipidemia. Available (free) at 2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Dyslipidemia
2.Watts GF, Gidding SS, Hegele RA, et al. International Atherosclerosis Society guidance for implementing best practice in the care of familial hypercholesterolaemia. Nat. Revs. Cardiol. 2023; 20:845-69. Available (free) at International Atherosclerosis Society guidance for implementing best practice in the care of familial hypercholesterolaemia | Nature Reviews Cardiology
3.UK National Institute for Heath and Care Excellence (NICE). Familial hypercholesterolaemia: identification and management. Clinical Guideline. (Pub 2008, updated 2019) Available (free) at Evidence | Familial hypercholesterolaemia: identification and management | Guidance | NICE
4.Kemsley S, Davis A, Blair J, et al. Lifespan Pathway for Lipid Management 2024. Guidance for all those caring for people with raised CVD risk. Available (free) at Lifespan Pathway for Lipids UK 2024_08.04.24.pdf
5.Hu P, Dharmayat KI, Stevens CAT, et al. Prevalence of familial hypercholesterolemia among the general population and patients with atherosclerotic cardiovascular disease: a systematic review and meta-analysis. Circulation. 2020;141(22):1742-1759.
6.Simon Broome diagnostic criteria for index individuals and relatives. Available in UK NICE Guideline on FH at Evidence | Familial hypercholesterolaemia: identification and management | Guidance | NICE (see Appendix F)
7.Wiegman A, Gidding SS, Watts GF, et al. Familial hypercholesterolaemia in children and adolescents: gaining decades of life by optimizing detection and treatment. Eur. Heart J. 2015; 36: 2425-37
8.Gidding SS, Wiegman A, Groselj U, et al. Paediatric familial hypercholesterolaemia screening in Europe: public policy background and recommendations. Eur. J. Prev. Cardiol. 2022; 29:2301-11. Available (free) at Paed FH screening in Europe public policy background and recommendations ESC 2022.pdf
9.Starr B, Hadfield, Hutten BA, et al. Development of sensitive and specific age- and gender-specific low-density lipoprotein cholesterol cutoffs for diagnosis of first-degree relatives with familial hypercholesterolaemia in cascade testing. Clin. Chem. Lab. Med 2008; 46(6): 791803
10. Silva L, Condon L, Qureshi N, et al. Introducing genetic testing with case finding for familial hypercholesterolaemia in primary care: qualitative study of patient and health professional experience.Br. J. Gen. Pract. 2022; Available (free) at DOI: https://doi.org/10.3399/BJGP.2021.0558
11.Louter L, Defesche J, Roeters van Lennep J. Cascade screening for familial hypercholesterolaemia: Practical consequences. Atherosclerosis Supps 2017; 30: 77-85. http://dx.doi.org/10.1016/j.atherosclerosissup.2017.05.019
12. Luirink IK, Wiegman A, Kusters DM, et al. 20-year follow-up of statins in children with familial hypercholesterolaemia. N. Engl. J. Med. 2019: 381:1547-56. Available (free) at 20-Year Follow-up of Statins in Children with Familial Hypercholesterolemia
13. The time is now: achieving FH paediatric screening across Europe. Prague Declaration. Available (free) at Prague Declaration in EU for FH screening. 2022.pdf
14.Martin AC, Gidding SS, Wiegman A, Watts GF. Knowns and unknowns in the care of pediatric familial hypercholesterolemia. J. Lipid Res. 2017; 58(9): 1765-76. Available (free) at DOI: 10.1194/jlr.S074039
15. Sarkies M, Sullivan D, Black A, et al.Developing a national implementation strategy for enhancing the detection of familial hypercholesterolemia: An Australian experience. J. Clin. Lipidol. Available (free) at Developing a national implementation strategy for enhancing the detection of familial hypercholesterolemia: An Australian experience - Journal of Clinical Lipidology
16.Watts GF, Sullivan DR, Hare DL, et al. Integrated guidance for enhancing the care of familial hypercholesterolaemia in Australia. Heart Lung Circ 2020: 30(3): 324-49. Available (free) at Integrated Guidance for Enhancing the Care of Familial Hypercholesterolaemia in Australia* - Heart, Lung and Circulation
17. Wiegman A, Bourbon M, Freiberger T, et al. Familial hypercholesterolaemia in children and adolescents: a European Atherosclerosis Society consensus statement. 2026 Available (free) at https://doi.org/10.1093/eurheartj/ehag382

Northland Key Contacts (09) 438 4243

Results		Press '1'	24 hours/7 days per week
Test Bookings	Book online through www.awanuilabs.co.nz	Press '2'	
Home Visits	Email nth.homevisits@awanuilabs.co.nz If the home visit cannot be booked for the date requested Home Visits staff will contact the referrer to arrange an alternative date. Phone enquiries to (09)438 4243	Press '3'	Mon-Fri: 8:00am to 3:00pm
Stores	nth.stores@awanuilabs.co.nz	Press '4'	Mon-Fri 8:00am to 5:00pm
Other Enquiries	nth.admin@awanuilabs.co.nz	Press '5' or Hold the line	Mon-Fri 8:00am to 6:00pm
E-orders Helpline	Email: helpdesk@eorder.co.nz	0508 37 37 83	

Auckland Key Contacts (09) 574 7399

Results		Press '1'	24 hours/7 days per week
Courier		Press '2'	24 hours/7 days per week
Home Visits	Email to auk.home.visits@awanuilabs.co.nz (preferred) If the home visit cannot be booked for the date requested Home Visits staff will contact the referrer to arrange an alternative date.	Press '3'	Mon-Fri: 8:00am to 6:00pm Sat: 8:00am to 12:00pm
Special test bookings	Book online anytime through www.awanuilabs.co.nz	Press '4'	Mon-Fri 8:00am to 6:00pm
Other Enquiries		Hold the line	Mon-Fri 7:00am to 11pm Sat-Sun 8:00am to 7:00pm
Add on tests	Requests for add on tests can be emailed to: call.centre@awanuilabs.co.nz		Note: some add on tests may require pathologists' approval.
Consumables orders	To enquire about consumables orders	Press '2'	Mon–Fri 07:00am to 3:30pm
Dedicated line for practitioners to access all results (24/7)		(09) 574 7398	

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