

QML Pathology Newsletter

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An Update on Diagnosis and Monitoring of Diabetes: The Laboratory Perspectives

Dr Julia Chang, Consultant Chemical Pathologist, QML Pathology

Traditionally the only method recommended for the diagnosis of diabetes was the direct measurement of glucose levels in plasma for the demonstration of hyperglycaemia.

However, there are practical issues with the fasting blood glucose and the oral glucose tolerance tests (OGTT). For example, the patients need to have a satisfactory period of overnight fasting and with OGTT, WHO states that patients should have had an appropriate diet for three days prior to the test. OGTT is not only time consuming and laborious to perform, but is also poorly tolerated by a significant number of patients.

The use of glycated haemoglobin (HbA1c) measurement as an alternative diagnostic test overcomes many of these issues. HbA1c can be performed at any time of the day, does not require any special pre-test preparation by the patient, and the blood sample is very stable. HbA1c test measures chronic glycaemia, rather than instantaneous blood glucose levels.

The blood glucose concentration cut-offs in table 1 were chosen based on their association with the presence of microvascular complications. Several studies have shown that HbA1c concentrations above 6.5% were at least as strongly correlated with the development of diabetic retinopathy as blood glucose concentrations.

TABLE 1: DIAGNOSTIC CRITERIA FOR TYPE 2 DIABETES

FBG	≥ 7.0 mmol/L (on two separate occasions)
2 hour postprandial	≥ 11.0 mmol/L on OGTT (on two separate occasions)
HbA1c	$\geq 6.5\%$ (48 mmol/mol) (on two separate occasions)

HbA1c has been endorsed as a diagnostic test for diabetes by the WHO, ADA and the Australian Diabetes Society in 2012. It has also been recognised by Medicare as a diagnostic test since 1 November 2014.

DIAGNOSIS OF GESTATIONAL DIABETES MELLITUS (GDM)

The screening and diagnostic criteria for GDM have recently been modified extensively. The current recommendations are based on the findings from The Hyperglycaemia and Adverse Pregnancy Outcome (HAPO) study. This study examines approximately 25,000 pregnant women and reports a strong correlation between increasing maternal glucose levels at 24-32 weeks gestation and a range of adverse maternal and foetal outcomes.

The latest key recommendations for the diagnosis of GDM are:

Testing Method

Diagnosis of GDM is based upon an oral glucose tolerance test. HbA1c currently plays no diagnostic role in testing for gestational diabetes.

All pregnant women should undergo a 75-gram two-hour oral glucose tolerance test (OGTT). The one-hour Glucose Challenge Test is no longer recommended. The three day high carbohydrate diet is no longer required and women should maintain a normal diet until 10 hours before the OGTT, and then fast.

Timing of Testing

Women not known to have risk factors for hyperglycaemia in pregnancy should undergo a 75g OGTT at 24-28 weeks gestation.

Women with risk factors for hyperglycaemia in pregnancy should be tested with first antenatal bloods or at the first antenatal visit. If the glucose tolerance is normal, a repeat OGTT should be performed at 24-28 weeks.

Revised New Diagnostic Criteria for Gestational Diabetes

The WHO has recommended that hyperglycaemia first detected at any time during pregnancy be classified as either diabetes mellitus in pregnancy or gestational diabetes mellitus. Women with diabetes mellitus in pregnancy are at higher risk of major pregnancy complications. They require urgent medical attention, including evaluation for other complications of undiagnosed diabetes.

The diagnosis of GDM should be based on any one of the following values:

- Fasting plasma glucose 5.1 – 6.9 mmol/L
- 1-h post 75 g oral glucose load > 9.9 mmol/L
- 2-h post 75 g oral glucose load 8.5 – 11.0mmol/L

The diagnosis of DM in pregnancy should be based on any one of the following values (2006 WHO criteria for DM):

- Fasting plasma glucose > 6.9 mmol/L
- 2-h plasma glucose > 11.0 mmol/L following a 75 g oral glucose load

DETERMINING THE CAUSE OF HYPERGLYCAEMIA

Once the diagnosis of diabetes has been established, it is important to determine the underlying cause of hyperglycaemia as it may alter the treatment and prognosis significantly.

The two major clinical classifications of diabetes are type 1 diabetes and type 2 diabetes. Type 1 diabetes is characterised by a state of pancreas beta-cell destruction by an autoimmune process and type 2 diabetes is characterised by a combination of resistance to insulin action and an inadequate compensatory response in insulin secretion. Type 2 diabetes is the most common form, affecting 90-95% of all people with diabetes.

Most patients with hyperglycaemia can be easily classified as having type 1 or type 2 diabetes by their clinical presentations. However, a small proportion of patients do not fit into these two classifications and other aetiologies, such as drug-or chemical-induced cases and LADA and MODY, should be considered.

TABLE 2: CAUSES OF HYPERGLYCAEMIA	
Type 1 diabetes	5 – 10%
Including latent autoimmune diabetes in adults (LADA)	
Type 2 diabetes	90 – 95%
Genetic defects	1 – 2%
Including maturity-onset diabetes of the young (MODY) and mitochondrial diabetes	
Other specific types of diabetes	
Diseases of the exocrine pancreas, drug or chemical-induced causes	
Gestational Diabetes	

LATENT AUTOIMMUNE DIABETES IN ADULTS (LADA)

LADA also known as late-onset autoimmune diabetes is a rather common and often under-recognised form of diabetes whose clinical presentation falls somewhere between that of type 1 diabetes and type 2 diabetes. From a pathophysiological perspective, LADA is more closely related to type 1 DM. It is a slowly progressive form of autoimmune diabetes and the majority of patients with LADA have at least 2 of 5 following distinguishing clinical features at diagnosis of diabetes:

- Age <50 years
- Acute symptoms of hyperglycaemia
- BMI <25 kg/m2
- Family history of autoimmune disease
- Personal history autoimmune disease

The presence of less than 2 distinguishing clinical features is a highly reliable method for excluding LADA (NPP 99%).

If there is clinical uncertainty, the diagnosis of LADA can be confirmed by the presence of one or more autoantibodies. The presence of multiple autoantibodies has the highest positive predictive value for LADA. Insulin autoantibodies are less useful because antibodies to the injected insulin develop as early as 7-10 days after initiation of insulin therapy. C-peptide and insulin assays are probably not useful because these patients tend to present with more preserved beta cell function than those with classic type 1 diabetes.

Testing for islet autoantibodies in suspected LADA

- GAD autoantibodies
- IA-2 autoantibodies
- Insulin autoantibodies (less useful)

MATURITY-ONSET DIABETES OF THE YOUNG (MODY)

Another important subtype of diabetes is MODY. MODY affects 1-2% of people with diabetes, although it often goes unrecognised. These patients are often misclassified as having type 1 diabetes, because they tend to be young and slim. In contrast to type 1 diabetes, MODY is a form of monogenic diabetes mellitus and is characterised by a lack of autoantibodies. It is important to identify these patients because some subtypes of MODY may be treated by diet or tablets, and do not always need insulin treatment.

The main clinical features of MODY are:

- Diabetes often develops before the age of 25
- No autoantibodies or features of insulin resistance
- Diabetes runs in families from one generation to the next (Autosomal Dominant)
- Diabetes may be treated by diet or tablets and does not always need insulin treatment

The diagnosis of MODY is made by performing diagnostic genetic testing. To date over 800 different mutations have been shown to be associated with MODY and 6 genes have been identified that account for the majority of MODY.

MODY 1	HNF-4A
MODY 2	Glucokinase (13% of cases)
MODY 3	HNF-1A (70% of cases)
MODY 4	IPF-1
MODY 5	HNF-1B
MODY 6	NeuroD1

The genetic tests for MODY are available in Brisbane however they are not covered by Medicare. Due to the cost of genetic testing, it is not cost-effective to screen every patient with diabetes. A more practical approach would be to calculate the probability of MODY using the diabetesgenes.org website calculator, and refer the patients with suspected MODY to a specialist.

MONITORING PATIENTS WITH DIABETES: HbA1c, FRUCTOSAMINE AND 1,5-AG

HbA1c

HbA1c measures the proportion of adult haemoglobin that has undergone glycation.

The higher the glucose level and the longer an elevated level in the blood, the more haemoglobin will be glycosylated. Once the haemoglobin is glycosylated, it stays that way for the rest of the life of the red blood cells. Therefore HbA1c reflects long term average blood glucose levels over the previous two to three months, and it is relatively unaffected by recent acute fluctuations in glucose levels.

What should the HbA1c numbers be?

FOR DIAGNOSIS OF DIABETES

<6.1 %	(<43 mmol/mol)	Current diabetes is excluded
6.1 – 6.4 %	(43 – 47 mmol/mol)	High risk of future diabetes
>6.4%	(>47 mmol/mol)	Diabetes is likely

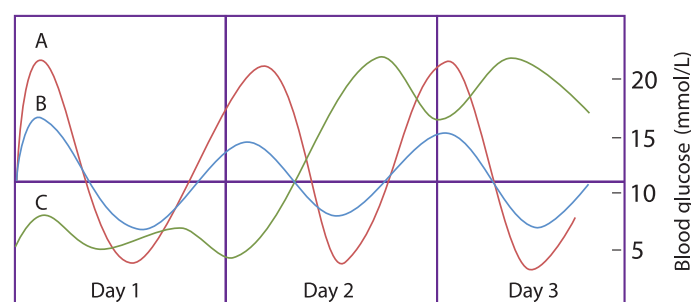
MONITORING PATIENTS WITH KNOWN DIABETES

- A general target HbA1c level of $\leq 7.0\%$ for most patients
- Individualisation of glycaemic targets based on patient-specific factors

What doesn't HbA1c result tell you?

Interpretation of HbA1c results may not always be straightforward. Although HbA1c is a marker for long term average blood glucose levels, it does not provide information on glucose fluctuations over the previous two to three months.

As demonstrated in Graph 1, although all of three hypothetical patients have a HbA1c level of 8.2%, they have different overall blood glucose control. Patient B has relatively small variations during the day and on different days; whereas patient A has marked blood glucose variations on the same day and patient C has marked blood glucose variations on different days. HbA1c level doesn't identify patients with marked glucose fluctuations and these patients are at an increased risk of having significant physical symptoms and complications.



Graph 1.

Factors affecting HbA1c results

If there is a discrepancy in HbA1c result and clinical findings, factors (Table 3) other than chronic glycaemic control need to be considered.

What if the HbA1c is unreliable?

When HbA1c concentration measurement is confounded in patients with increased red cell turnover, anaemia, haemoglobinopathy, liver disease or renal impairment, alternative markers can be used.

The patient can either perform self blood glucose monitoring before and after meals, or other glycated proteins such as fructosamines or the level of 1,5-Anhydroglucitol can be measured.

Medicare Benefits Schedule Pathology Items for HbA1c

- Monitoring of glycaemic control (ITEM 66551)
- The requirement of “established diabetes” must be satisfied
 - Subsidised up to four times in a 12 month period
- Monitoring of glycaemic control in pregnant patient with pre-existing diabetes (ITEM 66554)
- Subsidised by Medicare up to 6 tests in a 12 month period
- Diagnosis of diabetes in asymptomatic patient at high risk (ITEM 66841)
- One test in a 12 month period

TABLE 3: SOME OF THE FACTORS THAT INFLUENCE HBA1C AND ITS MEASUREMENT

Factor	Falsely increased HbA1c result	Falsely decreased HbA1c result	Variable changes in HbA1c result
Red Cell Survival Erythropoiesis	Iron deficiency Vitamin B12 deficiency Renal impairment	Administration of iron, vitamin B12 and erythropoietin Reticulocytosis Chronic liver disease	
Red Cell Survival Erythrocyte destruction	Splenectomy (increased RBC life span)	Haemoglobinopathies Splenomegaly Medications e.g., antiretrovirals	
Altered haemoglobin			Foetal haemoglobin Haemoglobinopathies Methaemoglobin

TABLE 4: COMPARING DIABETES BLOOD TESTS

	Uses	Limitations	MBS for diabetes blood test item numbers
HbA1c	Diagnosis and monitoring of diabetes Reflects long-term blood (2-3 months) glucose concentrations	Interference resulting in falsely increased or lowered results due to haemoglobinopathies, shortened red blood cell life span, liver disease, renal disease etc	Monitoring of glycaemic control (ITEM 66551) Subsidised up to four times in a 12 month period and the requirement of “established diabetes” must be satisfied Monitoring of glycaemic control in pregnant patient with pre-existing diabetes (ITEM 66554) Subsidised by Medicare up to 6 tests in a 12 month period Diagnosis of diabetes in asymptomatic patient at high risk (ITEM 66841) One test in a 12 month period
Fructosamine	Reflects short- to medium-term (2-4 weeks) blood glucose concentrations Allows the effectiveness of therapy adjustments to be evaluated after a few weeks Prognostic value is comparable to HbA1c	Falsely low results occur with rapid albumin turnover e.g., cirrhosis, nephrotic syndrome	Monitoring of glycaemic control (ITEM 66557) Subsidised up to four times in a 12 month period in the management of established diabetes
1,5-AG	Reflects short-term (1-2 weeks) blood glucose concentrations Complements HbA1c testing, and most useful in detecting post prandial hyperglycaemia in well or moderately controlled state (HbA1c 6.5 – 8 %)	Falsely low results can occur in Stage 4 or 5 kidney disease, advanced liver diseases and during pregnancy Acarbose and SGLT2 inhibitors may cause low values	No Medicare Rebate

Fructosamine

Fructosamine testing measures the proportion of blood and tissue proteins that have undergone glycation. A high fructosamine means that the patient's average glucose level over the previous 2 to 3 weeks has been elevated. Since albumin is the most abundant protein in the blood, fructosamine levels typically reflect albumin glycation. A falsely low fructosamine result will be seen in patients with a rapid albumin turnover, for example in patients with cirrhosis or nephrotic syndrome.

Fructosamine has not been as widely accepted as HbA1c because the association between a chronically elevated HbA1c level and an increased risk for certain diabetic complications is well established. A recent study shows that chronically elevated fructosamine is strongly associated with prevalent retinopathy and has an elevated risk of developing CKD among persons with diagnosed diabetes. Its prognostic value is comparable to HbA1c.

Medicare Benefits Schedule Pathology Item for fructosamine

Monitoring of glycaemic control (ITEM 66557)

- Subsidised up to four times in a 12 month period in the management of established diabetes

1,5-Anhydroglucitol (1,5-AG)

GlycoMark is a relatively new blood test that measures a glucose-like sugar called 1,5-anhydroglucitol. As blood glucose level increases, the 1,5-AG value decreases. It indicates blood glucose control over a one- to two-week period in patients with diabetes. It offers complementary information to HbA1c, and is most useful in looking at post prandial hyperglycaemia in diabetic patients with well or moderately controlled state (HbA1c 6.5 – 8 %). As discussed previously, some of the patients in "good control" may have significant glucose variability. Currently there is no Medicare rebate for 1,5-AG.

PATHOLOGIST PROFILE

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Dr Julia Chang graduated with a Bachelor of Medical Science (Honours) from the University of Sydney in 1997. She continued her studies, graduating in 2000 from the University of Sydney with a Bachelor of Medicine, Bachelor of Surgery (Honours). Dr Chang undertook an internship with the Concord Repatriation General Hospital in 2001. Following this, she was employed as a Chemical Pathology Registrar at the Princess Alexandra Hospital from 2003 to 2004, and with Queensland Health Pathology Services (QHPS) Central Laboratory, Royal Brisbane Hospital between 2004 and 2007.

Dr Chang joined QML Pathology as a Chemical Pathology Registrar, moving into the role of Consultant Chemical Pathologist in 2008. This same year she obtained her fellowship with the Royal College of Pathologists Australasia (FRCPA). Her special interests include iron disorders, clinical chemistry of pregnancy and drug testing.

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High-Sensitivity Troponin Testing in Primary Care

Dr Julia Chang, Consultant Chemical Pathologist, QML Pathology

In general, the indications to use hs-TnT in the management of acute coronary syndrome in a primary care setting are limited. Current Australian guidelines recommend that patients with suspected AMI should activate emergency medical services to enable transport to the nearest appropriate health care facility for urgent assessment.

TROPONIN TESTING PROTOCOL

All Troponin requests are automatically treated as URGENT. To ensure that these results can be phoned, faxed or downloaded to you as soon as they are available, please provide your contact details for business hours and out-of-hours, on the request form.

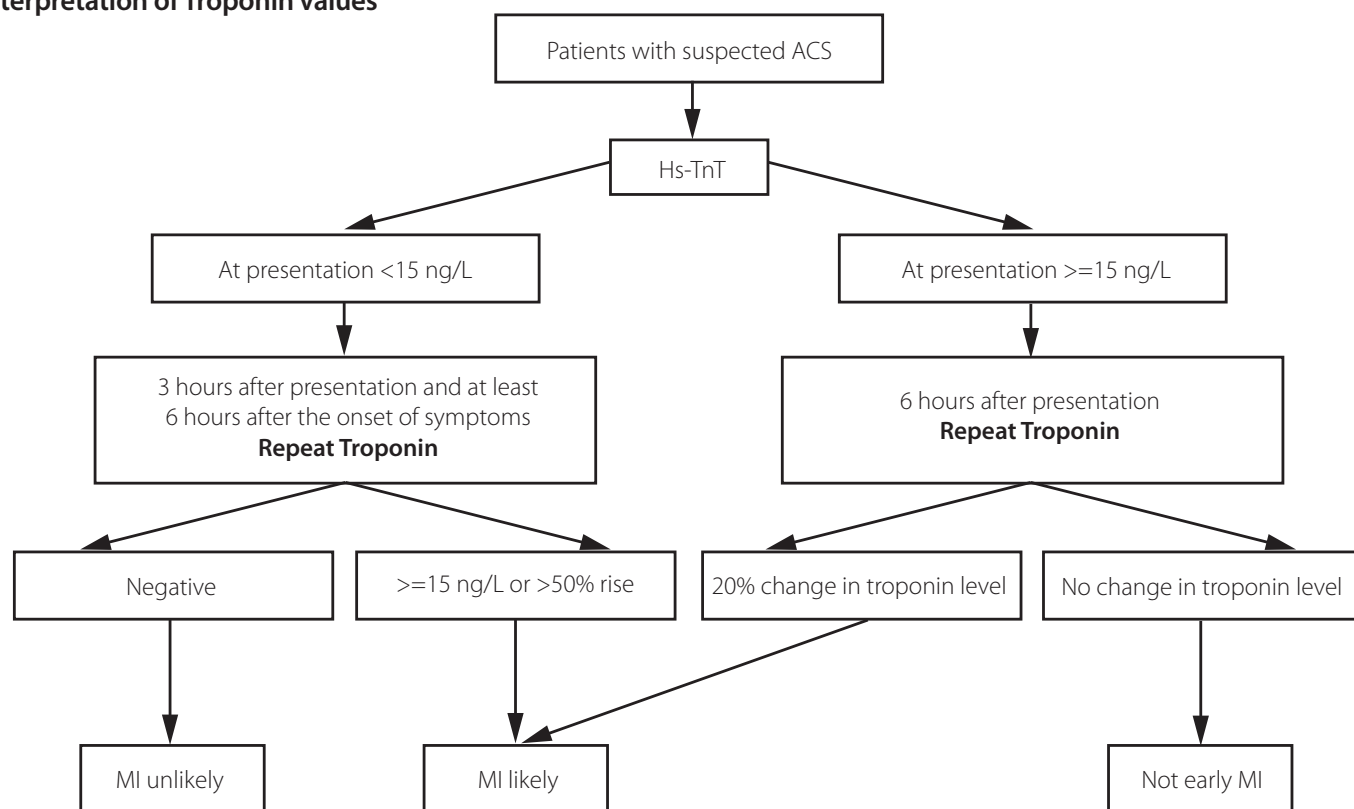
It has come to our attention that some clinicians request troponin testing as part of "routine cardiac assessment" in patients with low risk of coronary heart disease. If Troponin requests are for NON-URGENT cases, please indicate "Routine Request" on the request form.

WHAT IS TROPONIN?

Cardiac Troponin T (cTnT) and Troponin I (cTnI) are specific cardiac structural proteins. The cTn assays are highly specific for cardiac tissues but they do not indicate the mechanism for the injury. cTn elevations are not specific for acute coronary syndrome and can be seen in non-cardiac conditions such as pulmonary embolism, chronic renal failure etc. (See appendix 1).

cTnT will start to rise 3-4 hours after injury and can stay elevated for 7 to 10 days. Within the normal healthy population, 99% of people will have a Troponin T level less than 14 ng/L when a high-sensitivity assay for TnT (hs-TnT) is used.

Interpretation of Troponin values¹



Modified from the "2011 Addendum to the National Heart Foundation of Australia/Cardiac Society of Australia and NZ Guidelines for the management of acute coronary Syndromes (ACS), 2006".

TROPONIN AS A MARKER FOR MYOCARDIAL INFARCT

cTnT and cTnI are the preferred biomarkers for assessing myocardial injury and the dynamics of troponin levels (rise and/or fall over time) are central to the universal definition of acute myocardial infarction (AMI):

Typical rise and gradual fall in the level of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit (with imprecision <10% at this level) and with at least one of the following:

- Ischaemic symptoms;
- ECG changes indicative of ischaemia (ST-segment elevation or depression); or
- Coronary artery intervention (e.g., coronary angioplasty or coronary bypass surgery).

WHEN SHOULD A TROPONIN TEST BE REQUESTED IN THE COMMUNITY?

In general, the indications to use hs-TnT in the management of acute coronary syndrome in a primary care setting are limited. Although GPs have sufficient understanding of hs-TnT test for use in primary care, proper use of hs-TnT in primary care requires interpreting results in a clinical context, interpreting ECG findings, chasing hs-TnT results in a timely manner, and repeating hs-TnT assay as required.

Current Australian guidelines recommend that patients with suspected AMI should be assessed urgently in the nearest appropriate health care facility. Patients with high probability of an AMI should be admitted to hospital without delay.

All urgent requests for hs-TnT should include a contact number and clinical history, so that abnormal results can be phoned to the referring doctor or passed to the out-of-hours deputation service with the appropriate history.

It has come to our attention that some clinicians request hs-TnT as part of the "routine cardiac assessment" in patients with low risk of coronary heart disease. If the request is for non-urgent cases, please indicate on the request form, "Routine Request", "Non-Urgent Request", "Routine Risk Assessment" or similar.

Note: As more experience is gained with the use of the high-sensitivity Troponin test, we are seeing moves to shorten the intervals between repeat tests. This is an exciting area of developing diagnosis.

APPENDIX 1. ELEVATIONS OF CARDIAC TROPONINS WITHOUT OVERT ISCHEMIC HEART DISEASE

- Trauma (including contusion, ablation, pacing, implantable cardioverter defibrillator firings including atrial defibrillators, cardioversion, endomyocardial biopsy, cardiac surgery, after interventional closure of atrial septal defects)
- Congestive heart failure – acute and chronic
- Aortic valve disease and hypertrophic obstructive cardiomyopathy with significant left ventricular hypertrophy
- Hypertension
- Hypotension, often with arrhythmias
- Postoperative noncardiac surgery patients who seem to do well
- Renal failure
- Critically ill patients, especially with diabetes, respiratory failure, gastrointestinal bleeding, sepsis
- Drug toxicity e.g., adriamycin, 5-fluorouracil, herceptin, snake venoms, carbon monoxide poisoning
- Hypothyroidism
- Abnormalities in coronary vasomotion, including coronary vasospasm
- Apical ballooning syndrome
- Inflammatory diseases e.g., myocarditis, parvovirus B19, Kawasaki disease, sarcoid, smallpox vaccination, or myocardial extension of bacterial endocarditis
- Post-PCI patients who appear not to have complications
- Pulmonary embolism, severe pulmonary hypertension
- Sepsis
- Burns, especially if total surface burn area is >30%
- Infiltrative diseases, including amyloidosis, hemochromatosis, sarcoidosis, scleroderma
- Acute neurological disease, including cerebrovascular accident, subarachnoid bleeds
- Rhabdomyolysis with cardiac injury
- Transplant vasculopathy
- Vital exhaustion

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Hepatitis C Management Update

Dr R Vohra, Dr S Appleton

2016 estimates show chronic Hepatitis C virus (HCV) affects approximately 230,000 Australians with the projected number of Australians living with HCV set to triple by 2030.^{1,2}

There are multiple clinical benefits to curing HCV infection, notably HCV-associated disease is the most common cause of liver transplantation in Australia.²

Recent changes in the Pharmaceutical Benefit Scheme (PBS) listing of direct-acting antiviral (DAA) therapies for HCV treatment allow patients to be treated by general practitioners under certain criteria. This PBS listing of DAA medicines enables GPs to prescribe HCV medicines in consultation with an experienced gastroenterologist, hepatologist or infectious diseases physician.

A consultation with one of the above specialists removes the need for GPs to gain formal accreditation when treating HCV and has the increased benefit of improving access to HCV treatment within our communities.²

A positive HCV antibody test confirms a patient has been exposed to the virus at some point in time but does not reveal whether or not the patient has an active infection. To determine this, a molecular test is performed on blood.

The Qualitative HCV RNA test is used to detect the presence of HCV in the blood. Results of HCV RNA are reported as 'positive' or 'detected' if any HCV viral RNA is identified, or 'negative' or 'not detected' if otherwise. A positive HCV RNA result indicates active or current infection. The HCV RNA test may take up to three months to appear positive after viral exposure; however it is usually positive when tested at six weeks.

The HCV Viral Load test is a quantitative test that detects and measures the amount of viral RNA particles in a patient's blood. This test is often performed prior to treatment.

The Qualitative HCV RNA test or the HCV Viral Load may be used during or after treatment to help determine the patient's response to the therapy. Both tests have Medicare restrictions placed on their ordering. Additional tests within a 12 month period may incur a charge to the patient.

HCV genotype is the tool to determine the most appropriate Direct-Acting Antiviral therapy (DAA) for your patient.

Medicines listed on the PBS for treatment of HCV¹

- Daklinza® (daclatasvir)
- Harvoni® (sofosbuvir + ledipasvir)
- Ibavyr® (ribavirin)
- Sovaldi® (sofosbuvir)
- Viekira Pak® (paritaprevir + ritonavir + ombitasvir + dasabuvir)
- Viekira Pak RBV® (paritaprevir + ritonavir + ombitasvir + dasabuvir + ribavirin)

These HCV medicines may be used in various combinations depending on the clinical circumstances and will be available through both the PBS General Schedule (Section 85) and the Section 100 (S100) Highly Specialised Drugs (HSD) Program. PBS patient and prescriber eligibility will be the same whether the medicines are being prescribed under the PBS General Schedule or HSD Program.

Clinical guidance for diagnosing and treating Hepatitis 2 is summarised in the tables below.

Diagnosis

Hepatitis C antibody (serology)	Serology indicates exposure
Qualitative HCV RNA test	Presence indicates active infection

Post-diagnosis (Pre-treatment)

HCV Viral Load test (quantitative)	Confirms infection if detected
HCV genotype	Genotype determines treatment regimen

At each on-treatment visit assess for medication adherence, treatment adverse effects and drug-drug interactions.

Undergoing Therapy	
Week 0	FBE, U&E, LFTs, INR, HCV RNA level
Week 4	FBE, LFTs
Week 12 +/- 24	FBE, LFTs, Qualitative HCV RNA test
Week 12 after EOT (SVR)	FBE, LFTs, HCV PCR (qualitative)

Medicare billing criteria apply to a number of these tests as below

Qualitative HCV RNA Test

A patient is eligible for a Medicare rebate if at least one of the following criteria is satisfied:

1. The patient is Hepatitis C seropositive;
2. The patient's serological status is uncertain after testing;
3. The test is performed for the purpose of:
 - Determining the Hepatitis C status of an immuno-suppressed or immunocompromised patient; or
 - The detection of acute Hepatitis C prior to sero-conversion where considered necessary for the clinical management of the patient.

The patient may have a maximum of one test within a 12 month period.

Qualitative HCV RNA test in patients undergoing therapy.

A patient is eligible for a Medicare rebate if:

1. They are undertaking antiviral therapy for chronic HCV infection and the test is performed for the detection of Hepatitis C viral RNA.

The patient may have a maximum of four tests within a 12 month period

HCV Viral Load test (quantitative)

A patient is eligible for a Medicare rebate if:

1. The Quantitative HCV RNA is used in their pre- treatment evaluation or;
2. The Quantitative HCV RNA is used in the assessment of efficacy of a patient's antiviral therapy.

The test must be made by or on the advice of the specialist or consultant physician who manages the treatment of the patient with chronic HCV Hepatitis.

The patient may have a maximum of two tests within a 12 month period.

Please be aware if the patient does not fulfil the Medicare Criteria guidelines above a fee may apply

For further information please contact Dr R Vohra, Dr S Appleton, or Dr P Bartley on **(07) 3121 4444**

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Dr Renu Vohra obtained a Doctor of Medicine in Microbiology from the University of Delhi, India in 1994 and commenced training as a Pathologist in Australia in 1997. In 2000 she gained her fellowship with the Royal College of Pathologists Australasia (FRCPA). Dr Vohra worked in private pathology in Queensland, progressing from a Registrar position to a Consultant in Microbiology between 1999 and 2002.

From 2002 to 2004, Dr Vohra worked as a Clinical Microbiologist with Pathology Queensland, before joining QML Pathology as a Clinical Microbiologist. In 2009 she was appointed as Pathologist in Charge of Microbiology and Immunology at QML Pathology. Dr Vohra's special interests include bacteriology, molecular microbiology and antimicrobial stewardship.

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Dr Sally Appleton graduated with a Bachelor of Medicine and Bachelor of Surgery in 2004 from the University of Queensland and then continued her studies, completing her Fellowship in Microbiology in 2012.

Dr Appleton held the position of Senior Fellow in Microbiology with Queensland Health at their Central laboratory, Princess Alexandra Hospital Laboratory and the Public Health laboratory.

In addition to her full time duties in the Microbiology and Infectious Immunology areas since 2013, Dr Appleton has held a casual tenure since 2006 with the QML Pathology Warfarin Department continuing a long family association.

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From the Education Desk of QML Pathology

Cat 1 Quality Improvement - Important information for all Doctors

- There will be no new registrations for the QML Pathology Surgical Skin Audit and Cytology Pap Smear Audit after the 31st August 2016.
- Completion of both Audits and final upload of points will be December 2016.

We have had a great deal of queries and questions regarding QI components for the QML Pathology Surgical Skin and Cytology Pap Smear audits. Both audits are RACGP QI approved. We are working very hard to ensure the completion of all your QI requirements as we are fast approaching the end of the triennium.

IMPORTANT INFORMATION FOR AUDITS

- If you have not been receiving your monthly reports for either audit please contact QML Pathology Education department as we may need to check your registration particulars.
- Both Surgical Skin and Cytology Pap Smear Audits have specialised request forms. Please ensure these requests are utilised for specimen inclusion, and to order please use your stores request forms.
- For the Surgical Skin Audit please complete the reverse of the request form.
- For the Cytology Pap Smear Audit ensure you tick all relevant clinical boxes applicable.
- The aforementioned aspects must be adhered to as this data takes a different computer pathway through our system to give you all of your statistical information.
- Please note: 2017 changes to cytology screening are rapidly approaching. As the QML Pathology Cytology Pap smear audit was designed from the outset to report your patients +HPV, Chlamydia, Gonorrhoea and Trichomonas, please include all relevant clinical pathology and complete the clinical tick box area.
- If you relocate surgeries or commence in a new practice please let the QML Pathology Education team know so we adjust/add the audits to your QML Pathology doctor code.
- Please make sure we have your RACGP & ACRRM numbers and email address available to us at registration. Having your numbers at registration will guarantee a smooth transition for processing your achievements.

GENERAL EDUCATION SESSIONS

Thus far, 2016 has been very busy for the QML Pathology Education team with numerous Category 2 events held in regional areas of Queensland, Gladstone, Cairns and Townsville to name a few. These events were attended by GPs, Specialists and Nursing staff with positive and incredibly appreciative evaluations. We have also been very pleased with the response received from several small group interactive Clinicopath meetings that we have recently trialled. We hope to bring more of these types of events to the medical community in the future.

As you may be aware we are making the exciting move towards digital correspondence for all QML Pathology Education. QML Pathology will still send your reports, certificates and evaluations to you, so there is no need to remember to access these via the website. Please let us know your best email address for your Audit reports, certificates, evaluations and associated clinical documentation. Your documents must be retained in case of any issues that may arise with your college. Please be aware the option for hard copy correspondence is still available through QML Pathology.

If you have any queries regarding education please do not hesitate to contact either Tina or myself at the email and phone numbers noted below.

Jo Wilson-Farr

Manager - Dr Education and Private Practice Development

Education department contact details:

Phone: **07 3121 4453** or **07 3121 4565**

Fax: 07 3121 4478

Email: education@qml.com.au

A Quality Improvement Cat 1 is mandatory for 2014-2016 triennium. Do you have yours?



Cytology Pap Smear Audit Registration

Please complete registration details & return via courier,
fax (07) 3121 4478 or email education@qml.com.au



DOCTOR INFORMATION

Last Name: _____ First Name: _____

QML Dr. Code (if known): _____ RACGP QI&CPD/ACRRM No.: _____

HAVE YOU INCLUDED YOUR
RACGP QI&CPD/ACRRM
NUMBER?

PRACTICE INFORMATION

Practice Name / Address: _____

Practice Email Address: _____



Surgical Skin Audit Registration

Please complete registration details & return via courier,
fax (07) 3121 4478 or email education@qml.com.au



DOCTOR INFORMATION

Last Name: _____ First Name: _____

QML Dr. Code (if known): _____ RACGP QI&CPD/ACRRM No.: _____

HAVE YOU INCLUDED YOUR
RACGP QI&CPD/ACRRM
NUMBER?

PRACTICE INFORMATION

Practice Name / Address: _____

Practice Email Address: _____

Dear Doctor,

On Friday the 13th of May, Pathology Australia announced it had struck a deal with the Coalition Government whereby the members of Pathology Australia¹ (in particular Sullivan Nicolaides Pathology) would agree to cutting the funding of the bulk billing incentive for pathology MBS items, on the condition that rents paid by those pathologists to medical practitioners for collection centres were reregulated.

To clarify QML Pathology's position:

1. QML Pathology or its parent entity are NOT members of Pathology Australia.
2. QML Pathology is NOT supportive of the purported agreement struck by the members of Pathology Australia (including Sullivan Nicolaides Pathology) to regulate rents paid for collocated collection centres.
3. QML Pathology does NOT support any health care policy that withdraws funding from critical community-based frontline health care.
4. QML Pathology recognises that health care is an integrated network of care, with reduced funding in any segment impacting accessibility, quality and affordability throughout the health care "ecosystem".
5. QML Pathology is supportive of the RACGP's opposition to the proposed cuts in rent. RACGP president, Dr. Frank Jones, stated: "The Coalition's announcement of a backroom deal to cap rent will create further strain on the ability of general practice to keep their doors open and provide patient services."

We are aware that many pathology leases entered into by our peers include clauses that allow pathologists to reset rents to lower levels at any time due to changes in government regulation.

We believe that any reduction in investment in frontline care as envisaged under this is a false economy and will lead to greater healthcare costs in the future. This position, of course, extends to campaigning against the freeze on indexation of the Medicare Benefits Schedule fees until 2020, which we see as an attack on the hard-working clinicians who provide accessible health care to Australia.

Please do not hesitate to contact us if you wish to discuss this matter further.



John McKechnie
CEO - QML
0413 481 704



Dr Debra Norris
Medical Director - QML
07 3121 4444

¹ Pathology Australia members include, among others, Sonic Healthcare and its pathology divisions, Australian Clinical Laboratories, and other private pathology operators.



a new *era* in prenatal testing



Generation™ Non-Invasive Prenatal Testing (NIPT) represents a major advance in screening and risk assessment for chromosomal abnormalities.

Generation™ is a highly efficient, accurate, non-invasive prenatal screening test, based on Whole Genome Sequencing ("WGS") with proprietary algorithms, that analyses circulating cell-free fetal DNA from a maternal blood sample from as early as 10 weeks gestation.

The clinical utility and benefit of the **Generation™** test has been demonstrated in all pregnant women - regardless of age or risk category - in numerous publications, including studies in the New England Journal of Medicine, as well as reports with cohorts of over 34,000 patients^{1, 2, 3, 4}.

What does the **Generation™** NIPT test for?

Generation™ NIPT screens for the most commonly seen and tested chromosomal anomalies, including:

- ✓ trisomy 21 (Down syndrome)
- ✓ trisomy 18 (Edwards syndrome)
- ✓ trisomy 13 (Patau syndrome)

If specifically requested, the following more rarely occurring genetic abnormalities can also be tested for:

- ✓ Sex chromosome abnormalities
- ✓ Trisomy 9*
- ✓ Trisomy 16*
- ✓ Common microdeletions*
 - DiGeorge syndrome (22q11.2 deletion syndrome)
 - Angelman syndrome
 - Prader-Willi syndrome
 - Wolf-Hirschhorn syndrome
 - Cri-du-chat syndrome

References

1. Bhatt S, Parsa S, Snyder H, et al. Clinical Laboratory Experience with Noninvasive Prenatal Testing: Update on Clinically Relevant Metrics. ISPD 2014 poster.
2. Bianchi DW, Platt LD, Goldberg JD, et al. Genome-wide fetal aneuploidy detection by maternal plasma DNA sequencing. *Obstet Gynecol.* 2012; 119:890-901.
3. Futch T, Spinoso J, Bhatt S, et al. Initial clinical laboratory experience in non-invasive prenatal testing for fetal aneuploidy from maternal plasma DNA samples. *Prenat Diagn.* 2013;33:569-574.
4. Bianchi DW, Parker RL, Wentworth J et al. DNA Sequencing versus Standard Prenatal Aneuploidy Screening. *N Engl J Med* 2014; 370:799-808
5. Taneja et al. *Prenatal diagnosis*, Dec 2015
6. McCullough RM et al. *PLoS One.* 2014
7. Norton ME, et al. *New Engl J Med* 2015
8. Dar, et al. *Am J Obstet Gynecol.* 2014

Why use **Generation™** NIPT?

Generation™ uses whole genome / genome-wide sequencing which investigates more abnormalities, requires less foetal DNA, and has lower recollection and failure rates, compared to other methods^{5, 6, 7, 8}.

Who should be offered the **Generation™** NIPT test?

Numerous studies have conclusively demonstrated the benefits for NIPT in women with a high risk pregnancy, including:

- ✓ Women aged over 35
- ✓ Women with abnormal first trimester combined biochemical and ultrasound findings
- ✓ Women with a family history of chromosomal abnormalities
- ✓ Women with a high risk for invasive testing (e.g. IVF)

In addition, there is significant evidence to suggest that women in a normal risk population could also benefit from NIPT, particularly for peace of mind.

How much does **Generation™** NIPT cost?

The cost of this test is \$395* (or \$450* if the Microdeletion Panel is also requested by the Doctor) and is NOT Medicare rebatable.

*Prices are correct at time of printing and are subject to change without notice. This testing will incur additional costs. It is highly recommended that testing for microdeletion syndromes be accompanied by specialised genetic counselling.

For more information on **Generation™** NIPT

Please visit genomicdiagnostics.com.au
or call us on **1800 822 999**



Genomic Diagnostics
LEADING THE WAY TO IMPROVE HEALTH



ML Pathology

Specialists in Private Pathology since the 1920s

ImmunoCAP® ISAC - Allergy Testing

INTRODUCTION

Many allergic patients have positive test results to numerous allergens and the true cause of symptoms can be difficult to identify due to an inconclusive medical history regarding the role of different allergens and reactions.

QML Pathology offers ImmunoCAP® ISAC which provides a large amount of allergen specific IgE antibody information in a single step.

HOW CAN IMMUNOCAP® ISAC HELP?

- Shed light on the real sensitisation profile of multi-sensitised patients.
- Reveal potential risks for severe food-related reactions.
- Identify the IgE antibody profile in patients with unsatisfactory response to treatment.
- Assess patients with idiopathic anaphylaxis.
- Define sensitisation to component allergens from 51 sources the proteins are derived from.
- Reveal unexpected sensitisations or help you rule out allergy by delivering IgE results for a broad spectrum of allergens.

Allergen components by source



ImmunoCAP® ISAC contains a wide array of proteins from various allergen sources.

WHO SHOULD BE TESTED?

Testing may be recommended for patients with:

- Multiple food allergies
- Atopic dermatitis
- Oral allergy syndrome
- Food allergies where component resolved diagnosis would help risk stratification, e.g., peanut allergy
- 'Idiopathic' anaphylaxis for diagnosis of hidden food triggers
- Reassessment of sensitisation in patients failing to improve on specific immunotherapy (desensitisation)

FEATURES AND BENEFITS OF IMMUNOCAP® ISAC

Overall picture of the patient's IgE antibody profile

The results give you a highly detailed overview of primary and cross-reactive sensitisers, helping you assess the clinical risk for reactions.

Broad molecular allergen panel

It delivers IgE antibody results for 112 allergen components from 51 allergen sources in a single patient test.

Low sample volume

Only 30 µl of serum or plasma is needed.

Semi-quantitative determination

Semi-quantitative results correlate with the specific IgE level by CAP determination.

Low risk for false positive results

Low background gives blank results for non-atopic healthy controls as well as very good specificity in patients with high total IgE.

HOW TO ORDER IMMUNOCAP® ISAC

Request 'ISAC' in the tests requested section of your request form.

TURNAROUND TIME

The turnaround time for results is up to 4 weeks from the time of payment.

COST

ImmunoCAP® ISAC testing at QML Pathology \$350.00*.

FURTHER INFORMATION

For any further enquiries regarding this test, please contact Dr David Heyworth-Smith, Clinical Immunologist on **(07) 3121 4444** or david.heyworthsmith@qml.com.au

*Prices are correct at time of printing and are subject to change.

Doctors' Noticeboard

Dr David Sharp MBBS, FRACS

Dr Sharp is a Queensland trained Plastic and Reconstructive Surgeon. He obtained Fellowship of the Royal Australasian College of Surgeons and is a certified member of the Australian Society of Plastic Surgeons.

Dr Sharp is a visiting specialist at Greenslopes Private Hospital, Brisbane Private Hospital, Ipswich Day Hospital and South Brisbane Day Hospital, whilst maintaining a public commitment in plastic surgery at the Royal Brisbane and Women's Hospital.

P: (07) 3202 4744

E: info@drdavidsharp.com.au

Dr Gunasiri Mallikarachchi MBBS, MD, FRACP

Dr Mallikarachchi is a Consultant Physician and Specialist in General Medicine. Dr Mallikarachchi is a member of the Royal Australasian College of Physicians, the Internal Medicine Society of Australia and New Zealand, the Australian Association of Consultant Physicians and of the College of Physicians in Sri Lanka.

Sunnybank Private Hospital

Suite 10, Sunnybank Private Hospital, 245 McCulloch St
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W: www.brisbanephysicians.com.au

Dr Anthony J Kiosoglous

BSc(Hons) BMedSc MBBS(Hons) FRACS(Urol)

Dr Kiosoglous is a Urological and Reconstructive Surgeon at the Wesley Private Hospital. He completed a 2-year fellowship in London (Guy's and University College Hospitals). He manages all General Urology cases, with an interest in Urinary Incontinence, Prolapse, and Neuro-Urology. He is an International Presenter and Publisher, Journal Reviewer, Qld Committee Representative of RACS, member of Urological Society of Australia and NZ, and a Senior Lecturer at the University of Qld, School of Medicine.

Wesley Medical Centre

Suite 42, Level 4, 40 Chasely St, Auchenflower 4066

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E: urology@dranthonykiosoglous.com.au

The Doctors' Noticeboard is a free service for medical practitioners. If you wish to place a notice, please email no more than 75 words to info@qml.com.au

Collection Centre Search

QML Pathology's new Collection Centre Search console has launched, providing enhanced search functionality for both doctors and patients. Visit the qml.com.au webpage to experience our search revolution.

The functions for the Collection Centre Search console include:

- ✓ Locate collection centres within a desired region from suburb or postcode information.
- ✓ Obtain collection centre operational hours and contact information.
- ✓ Receive up-to-date public holiday or temporary closure times.
- ✓ Search for collection centres who perform specific tests.
- ✓ Find licence details and general centre features (e.g. on-site parking, on-site bathroom facilities, and test payment options).

The screenshot displays the QML Pathology website. On the left, there is a banner for 'Allergy Testing' with the text 'Know the Allergy, know the symptoms.' and a 'Click here for more' button. Below the banner are several service tiles: 'COLLECTION CENTRES' (We have over 600 collection centres across QLD and northern NSW), 'PAY YOUR ACCOUNT' (Easy, secure online payment, powered by Commonwealth Bank BPay), 'QML'S GASTROLAB' (Laboratory testing for functional gut disorders and carbohydrate malabsorption), 'VACCINE SUPPLIES' (Doctors can purchase Flu and Travel vaccines from one of Australia's leading vaccine suppliers), 'ONLINE RESULTS' (On the Go? - MedWay provides access to real-time results), and 'QML'S GENOM' (Our national ordinated gen testing service). On the right, the 'Collection Centre Search' console is shown. It features a search bar with the value '4007', filters for 'Any day', 'Any date', 'Any time', 'Standard test', and a checkbox for 'with wheelchair access'. Below the search bar, a list of collection centres is displayed: 'Acacia Ridge', 'Acacia Ridge', 'Agnes Water', 'Airlie Beach', and 'Aitkenvale'. A map on the right shows the location of these centres in Queensland, Australia.

Infectious Diseases Report

GEOGRAPHIC DISTRIBUTION - MAR 2016

ORGANISM	Regions (as per key below)															TOTAL			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	MAR	FEB	JAN	DEC
Adenovirus (not typed)	1	12	6	2			13		1	1	28	7	3	5		79	72	77	85
Adenovirus (typing pending)		1					1		1	2						5	10	15	16
Barmah Forest virus	5								2	1		2		6	2	18	27	5	3
Bordetella pertussis	2	73	30	9			38		33	12	91	40	16	3	10	357	464	442	406
Brucella species		2	1						1	1			3	1		9	6	7	7
Campylobacter jejuni		1									5					6	0	0	0
Chlamydia pneumoniae																0	2	1	0
Chlamydia trachomatis, not typed	73	153	48	27	2		118		65	24	213	103	34	46	25	931	1065	933	821
Coxiella burnetii		1	5							4			5	1		16	8	14	13
Cryptococcus species										2			1			3	1	2	2
Cytomegalovirus (CMV)		4	3	4			7		6	4	5	4	1	4	3	45	61	48	50
Entamoeba histolytica																0	0	0	0
Enterovirus - not typed																0	0	1	0
Epstein-Barr virus (EBV)	5	33	7	10			22		16	3	37	17	5	7	4	166	158	179	167
Flavivirus unspecified	8	4		1			4		3		5	2		3	2	32	23	20	11
Hepatitis A virus	1		1				1				4					7	13	3	3
Hepatitis B virus	5	15	18	1			18		8	2	69	5	4	2	4	151	132	147	105
Hepatitis C virus	27	96	46	13	1		69		49	20	157	65	28	22	37	630	453	357	354
Hepatitis D virus																0	1	1	0
Hepatitis E virus											1					1	0	0	1
Herpes simplex Type 1	23	69	26	13			56		34	12	84	47	10	15	9	398	363	407	379
Herpes simplex Type 2	12	67	14	6			32		25	6	47	28	2	11	9	259	239	211	206
Herpes simplex virus - not typed																0	0	0	0
HIV-1		6					8				4		1			19	25	13	19
HTLV-1																0	0	0	0
Human Metapneumovirus	1	17	2				12		3	3	14	8	2	3		65	53	60	128
Influenza A virus	14	44	6	5			65		25	30	72	47	8	33	6	355	253	92	45
Influenza B virus	5	13	3				9		1	4	9	4		3	4	55	53	16	19
Legionella pneumophila (all serogroups)							1									1	0	2	4
Legionella species																0	6	9	1
Leptospira species	3	1					1				1			3		9	5	5	5
Measles virus																0	0	0	
Mumps virus									1					1		2	1	2	2
Mycoplasma pneumoniae	1	42	25	13			52	1	29	20	91	44	9	6	11	344	380	312	275
Neisseria gonorrhoeae	5	14	1				17	1	5	1	14	3		1		62	69	66	59
Parainfluenza virus	4	35	16	2	1		44		24	28	67	14	8	21	4	268	170	69	112
Parvovirus		1	3				4			1	6	1		1	1	18	7	11	8
Pneumocystis carinii																0	3	2	0
Respiratory Syncytial virus	8	55	26	2			52		26	4	95	53	4	14	7	346	156	67	82
Rhinovirus (all types)	13	64	23	5			46		22	21	98	30	18	7	9	356	359	136	296
Rickettsia - Spotted Fever Group																0	0	1	0
Ross River virus	21	27	13	22			40		17	35	24	49	16	48	39	351	205	107	87
Rubella virus																0	1	0	0
Salmonella paratyphi A																0	0	0	0
Salmonella paratyphi B																0	0	0	0
Salmonella typhi							1				1					2	2	1	2
Streptococcus Group A	9	7	2	3	2		17	62	7	4	25	6	3	1	7	155	142	138	144
Toxoplasma gondii	1			3			2				1	3	1	1		12	19	11	13
Treponema pallidum	26	15	11	2	13		57	3	8	6	55	10	9	34	1	250	246	226	206
Trichomonas vaginalis	13	3	4		8				2	1	9	1	2	6		49	36	35	26
Varicella Zoster virus	15	46	22	8			39		41	10	83	47	5	5	5	326	336	297	284
Yersinia enterocolitica																0	0	0	0
TOTAL	301	921	362	151	27	0	846	67	454	260	1418	640	198	314	199	6223	5678	4548	4446

REGIONS:

1 Cairns

2 Gold Coast/Tweed

3 Ipswich

4 Mackay

5 Mount Isa

6 New England

7 North Brisbane

8 Northern Territory

9 Redcliffe

10 Rockhampton

11 South Brisbane

12 Sunshine Coast

13 Toowoomba

14 Townsville

15 Wide Bay/Burnett

FURTHER HISTORICAL CLINICAL DATA CAN BE OBTAINED BY CONTACTING MARKETING ON INFO@QML.COM.AU.