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## Urticaria – Epidemiology and Investigations

**Dr David Heyworth-Smith, Consultant Clinical Immunologist, QML Pathology**

**Urticaria (“Hives”) is a common and sometimes misunderstood skin reaction. Urticarial reactions range in severity from trivial and transient to profound and chronic with substantial effects on wellbeing and quality of life.**

Urticarial reactions involve mast cell degranulation with the release of histamine and other vasoactive mediators. This, in turn, leads to local capillary dilation and increased permeability, causing localised dermal oedema and erythema (welts or hives), as well as local nerve fibre activation causing mild to intense pruritis. Urticarial reactions may or may not be accompanied by angioedema, which is a similar pathological process involving deeper elastic subcutaneous tissues with the capacity for extensive expansion. Urticarial reactions may be acute, that is, lasting for less than 6 weeks (and often just a few days), or chronic, lasting for more than 6 weeks (and as long as months or years).

The prevalence of all forms of urticaria throughout the lifetime is as high as twenty percent, however is likely understated due to under-reporting, and also due to the complexity of aetiology and classification. Available prevalence studies of urticaria are usually regional or national population studies, as exemplified by the following. During 2004 in Spain a study of 5004 adults found that the point prevalence of chronic urticaria was 0.6%. In a 2010 German study of 4093 adults, the lifetime prevalence of all types of urticaria was 8.8%; a significant increase from the preceding year, where prevalence of all forms of urticaria was 0.8%; with the lifetime prevalence of chronic urticaria being 1.8%. A Norwegian study of 4992 individuals aged 15 to 70, established the lifetime prevalence of urticaria as 9%. In a remarkable Saudi Arabian study of 854 respondents, the lifetime prevalence of all forms of urticaria was 64%, considerably higher than the average reported rate. Similar data for Australia and New Zealand is not available.

CLASSIFICATION OF URTICARIA:

The most recent comprehensive international description of the classification and diagnosis of urticaria was derived from a consensus panel discussion at the 3rd International Meeting on Urticaria 2008. These guidelines were further adopted as international guidelines by EAACI / GA(2)LEN / WAO. This classification is adapted in Table 1.

TABLE 1: CLASSIFICATION OF URTICARIA	
Spontaneous urticaria	<ul style="list-style-type: none"><li>Acute spontaneous urticaria</li><li>Chronic spontaneous urticaria</li></ul>
Induced urticaria	<ul style="list-style-type: none"><li>Type I hypersensitivity (IgE) and other hypersensitivity reactions; Bites and stings and toxins; Infections</li></ul>
Physical urticaria	<ul style="list-style-type: none"><li>Cold Urticaria</li><li>Heat Urticaria</li><li>Solar urticaria</li><li>Delayed pressure urticaria</li><li>Vibratory urticaria</li><li>Dermatographic urticaria</li></ul>
Other types of urticaria	<ul style="list-style-type: none"><li>Aquagenic</li><li>Cholinergic urticaria</li><li>Exercise induced urticaria</li><li>Contact Urticaria</li></ul>

This classification is based on duration and aetiology. Most authorities would consider type 1 hypersensitivity reactions and other hypersensitivity reactions due to strictly exogenous factors such as foods, drugs, or stings as forms of inducible urticaria and distinct from acute spontaneous urticaria. Not specifically included in these schema are other well described but rare syndromes with urticarial manifestations such as Schnitzler’s syndrome (Muckle-Wells syndrome, Gleich’s syndrome), and Well’s syndrome. In addition, whether primary mast cell disorders (mast cell activation syndrome, urticaria pigmentosa and systemic mastocytosis) should be classified with the spontaneous urticaria or in a different schema is not totally clear at this point in time.

TABLE 2: EPIDEMIOLOGY OF URTICARIA – ASSOCIATED OR CAUSAL FACTORS	
Genetic factors	<ul style="list-style-type: none"><li>HLA associated</li><li>Non- HLA associated</li></ul>
Acquired factors	<ul style="list-style-type: none"><li>Autoimmune disorders</li><li>Infections</li></ul>
Environmental factors	<ul style="list-style-type: none"><li>Climate</li><li>Diet</li><li>Medications</li><li>Cultural factors</li><li>Bites and stings</li></ul>

EPIDEMIOLOGICAL FACTORS IN URTICARIA

Possible relevant epidemiological factors in urticaria are listed in Table 2. There are many potential associations or causal factors. However it is important to note in chronic spontaneous urticaria, as few as 40% or less of patients will have an identifiable aetiological factor. This underscores the complexity of the illness and perhaps our as yet nascent understanding of its epidemiology and pathogenesis.

Gender, Age, and Socioeconomic status

Chronic urticaria (CU) is somewhat more common in women compared to men. The peak age band is 20-40, but urticarial reactions occur at any age. The prevalence does not appear to be affected by socioeconomic status.

Genetic factors

Allergic and immunological disorders tend to have complex multifactorial aetiologies and genetic factors may only be one of many contributions. Occasionally in patients with urticaria a familial history is determined, however whether this represents coincidence or true inheritance is usually impossible to ascertain. Anecdotally, genetic factors have been closely analysed in urticarial syndromes, particularly in CU. Unsurprisingly, the main associations are in relation HLA class 1 and class 2 genes, and other immune effector genes. In all instances the associations are not strong.

Acquired factors

Autoimmune disorders and other diseases

Several autoimmune disorders are associated with CU, including autoimmune thyroid disease, and Coeliac disease. The first reports of associations between thyroid autoimmunity and urticaria in Medline appeared in the 1970s and 1980s with many reports and reviews subsequently and to the present. This, and associations with other autoimmune diseases, lead to the concept of CU as a possible autoimmune disease in some patients. In 1993 Dr. M. Greaves reported the landmark finding of functional IgG autoantibodies with specificity for the high affinity Fc epsilon receptor on Mast cells . In this group of patients, who are reported as 30-60% of CU patients, it is assumed that the autoantibodies are pathological and a cause of Mast Cell activation and de-granulation. The autologous serum skin test (ASST) remains the principal test to detect these autoantibodies, however this test is generally not performed due to practical considerations. The association between thyroid autoimmunity and CU remains vexing. The prevalence of antithyroid antibodies is high in CU patients with figures from 15 to 30% with an approximate background rate of positive antithyroid antibodies 5% in non-CU normal controls. In the majority of studies statistical significance compared to control

subjects is present. However whether there is a causal or mechanistic role of antithyroid antibodies in CU is not as clear. Positive thyroid antibodies detected include anti-thyroglobulin, anti-thyroid peroxidase (most often), and anti-TSH receptor antibodies. CU patients may be euthyroid or may have Hashimoto's hypothyroidism or Graves' disease. Coeliac disease is reported in association with CU including cold urticaria. Interestingly, in the majority of reports the urticaria resolves with the introduction of a gluten free diet. Similarly to the case of antithyroid antibodies, a mechanistic or causal understanding of this association is lacking.

## Infections

Urticaria may occur as a manifestation of an acute infection, or may occur as a reactive element induced by infection or parasitosis. Certain infections seem more likely than others to manifest urticarial reactions. Infections and parasitoses which are commonly linked to urticarial reactions are listed in Table 3. The interaction between the parasitic nematode *Anisakis simplex*, immune reactivity and allergy is fascinating. *Anisakis simplex* is a parasitic nematode of marine mammals, and certain fish species including cod, herring, mackerel, and pollack, with squid being intermediate hosts. Human infection occurs after consumption of inadequately cooked or raw colonised fish or squid. Humans are a non-suitable host and whilst acute illness may result with details beyond the scope of this discussion, human infection does not persist. However primary infection may lead to sensitisation to *Anisakis* proteins. Subsequent consumption of *Anisakis*, even from adequately prepared foods, may cause significant urticaria as the allergenic protein or proteins are thermostable. *Anisakis* infection and allergy is most common in The Netherlands and Japan and relates to the cultural habit of raw fish consumption, however *Anisakis* can be found in fish from Australian waters. More broadly, urticarial reactions are described in association with a wide range of parasites, in particular parasitic helminths. Helminths have unique interactions with the immune system, including generation of elevated IgE, eosinophilia and mucosal mast cell expansion. Such canonical Th2 changes might favour an urticarial process, and yet the majority of helminth infected individuals do not urticate, suggesting more complex host-pathogen interactions with individual variability are in play.

Other parasites and chronic infections have been linked to chronic urticaria. Recent interest has focused on the common organisms *Blastocystis hominis* and *Helicobacter pylori*. The protozoan *Blastocystis hominis* has been identified in stool samples at a much higher rate in CU patients compared to controls in developed nations. There are case reports of successful eradication of *Blastocystis* and cure of urticaria. Nonetheless, *Blastocystis* is very common

with a prevalence of 5% in developed nations and higher in developing nations and the risk for ascertainment bias is high. Similarly there are reports of the role of *H. pylori* in a variety of dermatological disorders including chronic urticaria since 1995. There are numerous smaller studies indicating relief from chronic urticaria after eradication of *Helicobacter* with antibiotic therapy. However critical appraisals and other case control studies have suggested only weak evidence of causality. Therefore, for both *Blastocystis* and *Helicobacter* infection, until further well-designed studies provide definitive evidence, caution should temper enthusiasm regarding the role of these organisms in acute or chronic urticaria.

Numerous acute or seroconversion infections are causative of acute urticaria, including Human Immunodeficiency Virus. These are summarised in Table 3.

**TABLE 3: INFECTIONS AND PARASITOSEs THAT HAVE BEEN LINKED TO URTICARIAL REACTIONS**

Viral and other infections	Parasites
<ul style="list-style-type: none"> <li>• Herpes group viruses</li> <li>• Human immunodeficiency virus</li> <li>• Hepatitis B and C viruses</li> <li>• <i>Mycoplasma pneumoniae</i></li> </ul>	<ul style="list-style-type: none"> <li>• <i>Giardia lamblia</i></li> <li>• <i>Blastocystis hominis</i></li> <li>• <i>Strongyloides stercoralis</i></li> <li>• <i>Toxocara</i></li> <li>• <i>Anisakis simplex</i><sup>†</sup></li> <li>• <i>Toxocara canis</i></li> <li>• Ticks<sup>#</sup></li> </ul>

<sup>†</sup>See text for further information

<sup>#</sup> In relation to sensitisation to beef galactose alpha - 1,3 – galactose sensitisation

## Environmental factors

### Climate, region, diet and culture

Urticarial reactions and various forms of chronic urticaria are a global and ubiquitous disorder. Urticaria was recognised by Hippocrates in the 4th century BC and therefore cannot be a disease of "modern lifestyle" alone. Well characterised cohorts with urticaria or guidelines for urticaria have been published from authors in expert groups in all regions or continents. Review of the available literature does not suggest any obvious regional variation in overall prevalence, nor any population which is unaffected. Furthermore, the spectrum of the physical urticarias (which might be affected by climate or temperature) appears to be widespread with, for example, cold urticaria reported in the tropics. Certain causes of urticarial reactions are regionally or culturally determined, and good examples are *Anisakis simplex* related urticaria, where the distribution is linked to consumption of undercooked or raw affected fish; and the Tick-Beef related urticaria syndrome described in the Australian eastern seaboard and central and eastern USA.

Stress and anxiety are frequently suggested causative or exacerbating factors for spontaneous urticaria, however not all studies have found associations and the risk of ascertainment bias in retrospective studies is likely to be significant. Nonetheless, many patients will anecdotally describe life stressors that precede or exacerbate their urticaria, and this should be considered in patient management. Type I hypersensitivity due to food allergy is a well described cause of acute urticarial reactions, with dietary “pseudoallergens” (e.g. dietary amines, salicylates, and glutamate) thought by some experts to have a role in the prolongation of chronic urticaria.

Medications

Many medications may cause an acute inducible urticarial reaction as part of a hypersensitivity reaction. Mechanisms are variable including type 1 hypersensitivity, immune complex formation, and direct histamine release. Examples of notable drugs or agents are listed in Table 4.

TABLE 4: EXAMPLES OF DRUGS IMPLICATED IN URTICARIAL REACTIONS	
Medication	Examples
Aspirin	• Aspirin
Non-steroidal anti-inflammatory drugs	• Diclofenac • Ibuprofen
Beta-lactam antibiotics	• Penicillins
Neuromuscular blocking agents	• Atracurium
Opioids	• Morphine
Immunisations	• Influenza A vaccination
Antiseptics	• Chlorhexidine
Dyes	• Patent blue dye • iodinated radiocontrast agents

Bites and Stings

Insect bites and stings are a common cause of acute inducible urticaria and other reactions. Commonly implicated insects include the hymenoptera (bees, wasps and ants) and regionally the Lepidoptera (caterpillars and moths). In maritime regions Medusozoa (jellyfish) stings are relevant.

INVESTIGATION OF URTICARIA

Mild and transient instances of spontaneous urticaria may not require any specific investigation unless the episodes are recurrent. If there is a possible inducing actor, for example a preceding bite or sting, or medication exposure, or possible food allergen, then investigation to confirm the causation is desirable.

Chronic urticaria lasting for more than 6 weeks should be investigated as a substantial minority of patients will have an important association, e.g. thyroid autoimmunity. Unfortunately the probability of discovery of a reversible or reliable cause in chronic urticaria is low. Conversely the majority of patients with chronic urticaria will not have any discernible underlying association or pathology, and these findings are often very reassuring. A reasonable approach to the investigation of urticaria is proposed in Table 5.

TABLE 5: AN APPROACH TO THE INVESTIGATION OF URTICARIA.		
Acute Induced Urticaria	Acute Spontaneous Urticaria (< 6 weeks duration)	Chronic Spontaneous Urticaria (> 6 weeks duration)
<ul style="list-style-type: none"><li>• Confirmation of suspected causation when possible.</li><li>• e.g. serum specific IgE for food allergens such as nuts, milk, egg, wheat, seafoods</li><li>• e.g. confirmation of drug allergy by laboratory or clinical testing when appropriate</li></ul>	<ul style="list-style-type: none"><li>• It may be appropriate to perform no investigations in mild to moderate cases that are not recurrent. Otherwise, consider investigations as per Chronic Urticaria.</li></ul>	<ul style="list-style-type: none"><li>• FBE</li><li>• ELFT</li><li>• CRP</li><li>• Serum EPP</li><li>• Antithyroid antibodies</li><li>• TSH</li><li>• Anti-TTG</li><li>• Basal serum tryptase</li><li>• ANA</li><li>• Serology for HIV, Hepatitis B, Hepatitis C if clinically indicated</li><li>• Stool OCP and PCR</li><li>• Specific IgE for food components including wheat*, omega-5-gliadin*, nuts*, alpha-gal**</li></ul>

\* If food dependent, exercise induced anaphylaxis (FDEIA) is suspected  
\*\* If tick –meat allergy syndrome (mammalian meat allergy) is suspected

TREATMENT OF URTICARIA

An in depth review of treatment of urticaria is beyond the scope of this article. However the first line treatment which provides substantial relief in most patients is with 2nd generation non-sedating antihistamine, such as loratidine, desloratidine, fexofenadine, or cetirizine. First generation sedating antihistamines, such as promethazine, may be useful acutely but are usually not recommended for intermediate to long term therapy due to their sedative and other side-effects.

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## PATHOLOGIST PROFILE

**Dr David Heyworth-Smith FRCPA, FRACP**

Consultant Clinical Immunologist

**P: (07) 3121 4325****E: DrDavid.HeyworthSmith@qml.com.au**

Dr David Heyworth-Smith graduated with a Bachelor of Medicine and Bachelor of Surgery (Hons) from The University of Queensland in 1993. From 1994 through to 2002 he undertook Clinical training at Princess Alexandra Hospital, Royal Brisbane Hospital, and Royal Prince Alfred (Sydney) Hospital. In 2002 Dr Heyworth-Smith was awarded his Fellowship at both the Royal Australasian College of Physicians and the Royal College of Pathologists of Australasia.

During 2003 he practiced as a Clinical Immunologist and Immunopathologist at the Princess Alexandra and Royal Brisbane Hospitals before joining QML Pathology in 2004. His special interests include Vasculitis syndromes, Hereditary angioedema, the Antiphospholipid syndrome, immunological disorders in pregnancy, and food allergy. Currently Dr Heyworth-Smith is a Pathologist with QML Pathology, a Clinical Immunologist VMO principally at Greenslopes Private Hospital, as well as visiting a number of Brisbane hospitals.

## Update on Zika Virus

Zika virus outbreak has now been declared as an international public health emergency by the World Health Organisation due to a large number of cases of congenital microcephaly and other neurological disorders in the Americas.<sup>11</sup>

A number of countries in South and Central America have reported cases of Zika virus since the Pan American Health Organization (PAHO) confirmed the first case of Zika virus infection in Brazil in May 2015.<sup>1</sup>

The Zika affected countries as per Centers for Disease Control and Prevention (CDC) are shown below.

### \*LIST OF COUNTRIES AND TERRITORIES WITH ACTIVE ZIKA VIRUS TRANSMISSION<sup>6</sup>

<b>Americas</b>	Barbados, Bolivia, Brazil, Colombia, Commonwealth of Puerto Rico, Costa Rica, Curacao, Dominican Republic, Ecuador, El Salvador, French Guiana, Guadeloupe, Guatemala, Guyana, Haiti, Honduras, Jamaica, Martinique, Mexico, Nicaragua, Panama, Paraguay, Saint Martin, Suriname, U.S. Virgin Islands, Venezuela
<b>Oceania/Pacific Islands</b>	American Samoa, Samoa, Tonga
<b>Africa</b>	Cape Verde

\*This list is accurate as of February 3, 2016. As the outbreak is rapidly evolving, updates on areas with ongoing Zika virus outbreaks can be obtained from the CDC website.<sup>6</sup>

### WHAT IS ZIKA VIRUS AND HOW IS IT TRANSMITTED?

Zika virus is a flavivirus that was first isolated in 1947 in Uganda.<sup>5,9</sup> Zika virus is related to other flaviviruses like dengue, yellow fever and Japanese encephalitis. Prior to 2015, outbreaks of Zika virus have been reported in Africa, Southeast Asia and Pacific Islands<sup>9</sup>. This virus is transmitted through the bite of an infected *Aedes aegypti* mosquito<sup>1,7</sup>.

*Aedes aegypti* are the predominant vectors for transmission, however other *Aedes* species like *Aedes albopictus* are also potential transmitters of the virus<sup>7</sup>. *Aedes aegypti* mosquitoes which also transmit dengue are found in north Queensland and some areas in central Queensland; therefore, it is possible for local transmission of Zika virus to be established if a case of Zika virus infection is diagnosed in a returned traveller in north Queensland. Hence it is important for clinicians to urgently notify their local public health unit to report clinically suspected cases of Zika virus in north Queensland and parts of central Queensland where mosquito vectors are present.<sup>3</sup>

The transmission of Zika virus from mother to fetus has been reported.<sup>1</sup> Transmission of Zika virus through sexual contact has been reported and CDC have issued guidelines for prevention of sexual transmission of Zika virus<sup>10</sup>. Transmission through blood transfusion is also possible.<sup>1</sup>

### WHAT ARE THE SYMPTOMS OF ZIKA VIRUS?

In most cases Zika virus causes a mild and self-limiting infection, about 1 in 5 people infected with Zika virus become symptomatic after an incubation period of 3-12 days.<sup>3</sup> Common symptoms include fever, maculopapular rash, headache, red eyes, myalgia and arthralgia.<sup>3</sup> Symptoms of abdominal pain, diarrhoea, constipation, mucous membrane ulcerations and pruritus are rare.<sup>3</sup> Severe illness leading to death is rare. Guillain-Barré syndrome has been reported in patients following suspected Zika virus infection.<sup>1</sup>

### HOW TO DIAGNOSE ZIKA VIRUS?

Travellers presenting with symptoms consistent with Zika virus infection within two weeks of return from areas with recent outbreaks should be tested for Zika virus infection<sup>3</sup>.

As symptoms of Zika virus closely resemble dengue and chikungunya virus infection and the fact that they are all transmitted by the same *Aedes* mosquito species it is important to rule out these infections in a returned traveller. Rash in a returned traveller could also be due to measles, rubella, parvovirus and other alphaviruses, therefore these diseases should be considered in the differential when working up the patient.

### TESTS AVAILABLE TO DIAGNOSE ZIKA VIRUS

1. PCR: PCR for Zika virus on serum is positive in the first week after onset of symptoms.<sup>2</sup>
2. Serology: Virus-specific IgM and neutralizing antibodies can be detected towards the end of first week of illness.<sup>2</sup> It is important to collect an acute sample within 5 days of onset and a convalescent sample 14-21 days after onset of symptoms. Cross-reaction with related flaviviruses (e.g. dengue and yellow fever viruses) is common and therefore collection of acute and convalescent samples will help in resolving cross reactions.<sup>3</sup> Cross reactions are more common in patients with a history of previous flavivirus infection or vaccination against a related flavivirus.<sup>8</sup>

Specimens sent to QML Pathology for testing will be referred to the Public Health Virology Laboratory which performs these tests. Zika virus infection is notifiable in Australia.

## ZIKA VIRUS AND PREGNANCY

Recently concerns have been raised that Zika virus infection in pregnancy can lead to congenital abnormalities (microcephaly) in the fetus.<sup>1,7</sup> However this epidemiological link needs to be investigated and confirmed by further studies. Until this link is confirmed women who are pregnant (in any trimester) or who plan to become pregnant should consider postponing travel to any area where Zika virus transmission is ongoing.<sup>3</sup>

CDC has issued an update on its interim guidelines for health care providers caring for pregnant women with Zika virus exposure.<sup>12</sup> Infection with Zika virus in pregnancy can occur in any trimester<sup>8</sup>. Laboratory testing for Zika virus should be performed on all pregnant women with recent travel to an area of ongoing transmission and who present with symptoms consistent with Zika virus infection during or within 2 weeks of returning from travel<sup>4</sup>. CDC in its recent update has included serologic testing for Zika virus on asymptomatic pregnant women 2- 12 weeks after return from travel to an area of local transmission of Zika virus<sup>12</sup>. Once laboratory infection is confirmed, the patient should be referred to a specialist for further management. PCR testing for Zika virus RNA can be performed on amniotic fluid.<sup>7</sup> However, the sensitivity and specificity of the PCR test performed on amniotic fluid for diagnosing congenital infection is not known<sup>4</sup>. For more detailed information on management and treatment of pregnant women with Zika virus refer to CDC update: interim guidelines for health care providers caring for pregnant women and women of reproductive age with possible Zika virus exposure.<sup>12</sup>

## TREATMENT

There are no antiviral drugs available for treatment. Treatment is for symptomatic relief including rest, fluids, and use of analgesics and antipyretics. Dengue should be ruled out before prescribing aspirin and other non steroidal anti-inflammatory drugs to reduce risk of haemorrhage.<sup>8</sup>

## PREVENTION

Because there is neither a vaccine nor prophylactic medications available to prevent Zika virus infection, travellers are advised to strictly follow steps to avoid mosquito bites. Mosquitoes that spread Zika virus bite both indoors and outdoors, mostly during the daytime.<sup>1</sup>

General mosquito avoidance strategies include wearing clothes that cover arms and legs, using insect repellents and staying in accommodation that have screens in doors and windows to keep mosquitoes out.

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12. Oduyebo T, Petersen EE, Rasmussen SA et al. Update: Interim Guidelines for Health Care Providers Caring for Pregnant Women and Women of Reproductive Age with Possible Zika Virus Exposure — United States, 2016. *MMWR Morb Mortal Wkly Rep* 2016; 65(05):1–6. [http://www.cdc.gov/mmwr/volumes/65/wr/mm6505e2er.htm?s\\_cid=mm6505e2er.htm\\_w](http://www.cdc.gov/mmwr/volumes/65/wr/mm6505e2er.htm?s_cid=mm6505e2er.htm_w)

For further information, please contact

**Dr Renu Vohra MBBS MD FRCPA**

Pathologist in Charge - Microbiology & Immunology

**P: (07) 3121 4436**

**E: DrRenu.Vohra@qml.com.au**

## Faecal Haemoglobin Testing (Faecal Occult Blood Testing)

QML Pathology has introduced an automated analyser, OC Sensor Diana, to detect faecal occult blood (FOBT).

The OC Sensor Diana uses an immunochemical based FOBT method for detection of colorectal cancer. Immunochemical testing which utilises antibodies against human haemoglobin has now superseded the old Guaiac method of FOBT.

The OC Sensor Diana requires special collection tubes (green top) that will be available to the patients from their nearest QML Pathology collection centers from 1st March 2016. The kit contains three green top collection tubes with an instruction flyer (see below). The specimens after collection can be dropped off at any QML Pathology collection centre.

Specimen Collection Flyer / Faecal Occult Blood Collection (as below).

### Faecal Occult Blood Collection

Your doctor has requested a sample of faeces to be sent to the laboratory for examination.

PLEASE USE THE FOLLOWING INSTRUCTIONS TO MAKE YOUR COLLECTION:



Collection Tube

**DO NOT collect faecal sample during menstrual cycle or if bleeding hemorrhoids present.**

1. Place a clean holder/container in the toilet bowl to catch faeces.
2. Once you have your faeces sample, open the green cap on the tube provided. Keep tube upright and take care to avoid spilling agent inside.
3. Using the threaded end of the sampler, poke faeces 5 times, in a different point each time.
4. Put the sampler back into the tube, push the cap on tightly to click shut and shake vigorously.
5. Ensure tube is labelled with: • Full Name • Date of Birth • Date & Time of Collection.
6. Keep sample at room temperature.
7. Repeat this procedure for multiple faecal occult blood tests on 3 separate consecutive days. Please use a fresh tube for each sample.
8. Return to doctor's surgery or QML Pathology Collection Centre.

**PLEASE NOTE:** If your request includes faeces test(s) other than for Occult Blood, a faeces specimen must be submitted in a separate provided container.

- The faecal occult blood test looks for blood present in faeces.
- Your doctor will require 3 samples collected over 3 days (one sample per day).
- When all 3 samples have been collected they **MUST** be returned to QML Pathology on the 3rd day.

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QML Pathology











Specialists in Private Pathology since the 1920s

# Skin Biopsies – a Quick Reference Guide





This guide helps explain how your biopsy technique can influence the accuracy of your patient's histology diagnosis.

Skin biopsy is a rapid procedure useful in the diagnosis of many neoplastic and inflammatory conditions, and therefore a valuable tool when the clinical differential diagnosis encompasses different treatment options.

The value of the biopsy may be limited by its size, the site selected for sampling, superimposed inflammatory changes, the application of topical agents, or concurrent use of medications. One of the major limiting factors is the lack of sufficient clinical information, with a clinical diagnosis frequently useful in the diagnosis of tumours, and essential in the diagnosis of rashes.

SELECTING THE BIOPSY SITE		
	Incision Biopsy	Punch Biopsy
Blotchy, macular		
Annular		sometimes unsuitable for punch
Discoid, plaque		
Papular		
Vesicular, bullous		unsuitable for punch
Nodule, tumour		
<p>* If food dependent, exercise induced anaphylaxis (FDEIA) is suspected</p> <p>** If tick –meat allergy syndrome (mammalian meat allergy) is suspected</p>		

## WHAT TO CALL YOUR SPECIMEN

	The pathologist performing the macroscopic examination needs to know whether a piece of skin this shape is an excision biopsy or an incision biopsy.
	Most specimens require division before being processed. A piece of skin this shape: should be divided this way if it is an excision specimen (as these transverse sections will display the margins of excision in relation to a tumour).
	If it is an incision specimen, it should remain whole so that sections display the full length of the specimen.
	If it is more than 3 mm wide, it should be divided longitudinally.

- For technical reasons QML Pathology reduces tissue blocks to about 2 mm thickness.
- To avoid a good incision biopsy being partly wasted or an excision biopsy that cannot be assessed for completeness of tumour removal, please specify excision or incision biopsy.

## CLINICAL NOTES

A clinical description (including clinical diagnosis or differential) is frequently useful in the diagnosis of tumours, and is usually essential in the diagnosis of rashes. Information should include:

- Exact site
- Size
- Duration
- Appearance
- Symptoms
- Drugs
- Clinical diagnosis

For further information, please contact the QML Pathology Histology Department on **07 3121 4495**

## Holter and ABP Monitoring

**Both Holter monitoring and Ambulatory Blood Pressure monitoring are now available at a number of QML Pathology Collection Centres.**

The Holter monitor is used to diagnose arrhythmias, or for investigation when patients have experienced dizziness, light-headedness, blackouts or fainting spells. The monitor can also be used to analyse patients with a pre-determined cardiac condition and to ascertain the effects of cardiac medications.

ABP monitoring is used to survey ambulatory pulse waves, peripheral blood pressure, central haemodynamics, and arterial stiffness. ABP assessment allows the analysis of clinical effects, drug-related effects and work influence to be studied over a period of 24 hours rather than at isolated clinic blood pressure readings.

The Holter and ABP monitor cuff remains in situ for a 24 hour period.

Following both tests an extensive overview is developed by a cardio specialist who will provide a high quality Holter report and/or ABP profile.

Patients currently taking prescribed medications should continue to do so throughout the test unless otherwise directed by their physician.

Holter monitoring performed by QML Pathology will be bulk billed. Bulk billing is subject to Medicare guidelines and criteria. Ambulatory blood pressure monitoring will attract a fee of \$88.00.

For further information, please contact your Medical Liaison Officer.

## C-Urea Breath Testing

**The <sup>14</sup>C-Urea Breath Test is considered to be the 'gold standard' non-invasive diagnostic method for detection of the presence of current infection with *H. pylori* in the stomach.**

The <sup>14</sup>C-Urea Breath Test offers highly accurate and reliable diagnosis of *H. pylori* infection with a positive predictive value of 100% and a negative predictive value of 98%. It is valuable in initial diagnosis as well as post-treatment follow-up to demonstrate the eradication of *H. pylori*.

*H. pylori* infection is significantly underdiagnosed as the majority of individuals with uncomplicated infection are asymptomatic. Testing is currently recommended for: patients with known or suspected *H. pylori* infection, gastritis, peptic ulcer disease or gastric malignancy; patients with epigastric pain, discomfort or non-ulcer dyspepsia; patients experiencing unexplained anorexia, weight loss, nausea or vomiting; patients receiving or about to receive long-term proton pump inhibitor therapy; first degree relatives of patients with gastric cancer; and 4-6 weeks after treatment in all patients treated for *H. pylori* infection to confirm eradication of infection.

Prior to undertaking the test, patients must discontinue certain medications that have the tendency to cause false-negative results. It must be ensured that the patient has not had antibiotics and bismuth-containing products for 4 weeks prior to the test; cyto-protective medicines (e.g. sucralfate) for 2 weeks prior; and Losec, Zoton or Somac (or any other proton pump inhibitors) for 1 week prior to the test. Finally, the patient must not consume any food or drink (including water) for 6 hours prior to taking the test.

It must be noted that although the radiation dose is extremely small, this test has not yet been sufficiently tested in children or pregnant females. Therefore it is advised that the test not be performed in these groups.

For any further enquiries regarding the <sup>14</sup>C-Urea Breath Test please contact QML Pathology on **07 3121 4420**.

## Diagnosis of Dengue Virus

With over half of the world's population living in regions at risk of potential dengue transmission, dengue is the most important arboviral disease worldwide in terms of morbidity and mortality.

Dengue is a mosquito-borne viral disease (arbovirus) that is increasing in prevalence worldwide. It is caused by infection with any one of four antigenically distinct dengue virus serotypes (DENV 1-4) belonging to the family Flaviviridae.

Dengue is transmitted in Queensland primarily by the *Aedes aegypti* mosquito. Over half the world's population lives in regions at risk of potential dengue transmission, making dengue the most important arboviral disease worldwide in terms of morbidity and mortality. Significant global expansion and urbanisation has been coupled with rapid increase in incident cases, epidemics and disease severity<sup>1</sup>.

The clinical manifestations of the dengue virus are varied, ranging from sub-clinical to fatal. Symptomatic infection with dengue can result in dengue fever, dengue haemorrhagic fever/dengue shock syndrome (DHF/DSS) and death<sup>2</sup>.

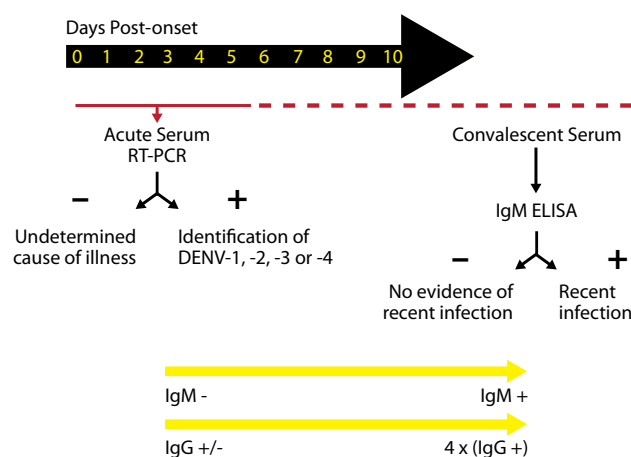
Prompt case detection and appropriate clinical management allows earlier implementation of supportive therapy. Rapid detection and reporting of dengue cases to public health centres is critical in managing an epidemic as effective vector control is the mainstay of dengue prevention.

Detection of virus or viral antigen or detection of the immune response will enable diagnosis of dengue. Acute dengue infection is characterised by fever and viraemia and may be diagnosed by testing for NS1 antigens (a non structural protein of the dengue virus) or dengue virus PCR. IgM and IgG antibodies to dengue are formed following infection and increase to detectable levels within 2 weeks (Figures 1 and 2). Understanding the two stages of dengue infection is critical to accurate diagnosis.

Patients with dengue fever usually seek medical attention within the first two days of symptoms as the onset of fever and malaise is acute. At this stage diagnosis is only possible by genome and/or antigen detection (PCR or NS1 rapid test).

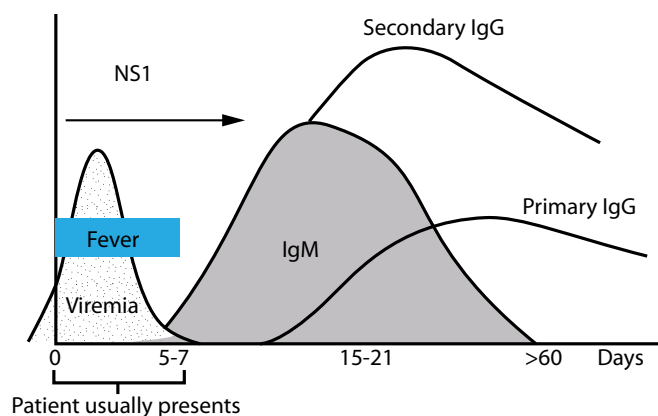
Serological diagnosis is most accurate once fever subsides. Post fever, the IgM serological tests will be positive however tests to detect dengue proteins such as the NS1 antigen and viral PCR will be negative.

PCR can be used to confirm the presence of the dengue virus and will validate NS1 and serology findings, as well as provide information about the infecting serotype.



**Figure 1: Dengue Diagnostic Process**

<http://www.cdc.gov/dengue/clinicalLab/diagnosticProcess.html>



**Figure 2: Markers for Dengue Diagnosis**

Guzman MG et al. Laboratory Diagnosis of Dengue. Dengue and Dengue Hemorrhagic Fever 2nd Edition. Eds Gubter et al. Chapter 10 Pg 185 – 209.

To order, request Dengue NS1, Dengue Serology and Dengue PCR.  
Medicare rebates apply. NS1-Same day result.

1. Murray NEA, Mikkil BQ, Walter-Smith A. Epidemiology of Dengue: past, present and future prospects. Clin Epidemiol. 2013; 5: 299-309.

2. Halstead SB. Dengue. Lancet 2007; 370: 1644-52.

3. Huang S, Williams F, Clements ACA, Wenbio H. Imported Dengue Cases, Weather Variation and Autochthonous Dengue Incidence in Cairns, Australia. PLOS 2013;8 Issue 12 e81887.

## From the Education Desk of QML Pathology

### Welcome to 2016 and the last year of the RACGP Triennium

**Do you have your QI (Quality Improvement) completed? At least one of your category 1 activities must include a quality improvement component for the RACGP 2014-2016 Triennium.**

The RACGP states that inherent quality improvement includes the following

- Clinical audit (40 points)
- PDSA cycles (40 points)
- Small group learning (40 points)
- Evidence based medicine journal club (40 points)
- Supervised clinical attachment (40 points)
- GP research (40 points).

Both of our QML Pathology audits are quality improvement approved so if you have been amongst the thousands of Queensland & NSW doctors taking part in the Surgical Skin and Cytology Pap Smear Audit and have achieved your Cat 1 points then your QI component for the 2014-2016 triennium has been completed.

#### HELPFUL HINTS FOR AUDITS

If you have not been receiving your monthly reports for either audit please contact the QML Pathology Education department at [education@qml.com.au](mailto:education@qml.com.au) as we may need to check your registration particulars.

- Both Surgical Skin and Cytology Pap Audits have specialised request forms. Please ensure these requests are utilised for specimen inclusion and please use your stores request forms to order.
- The Surgical Skin Audit requires completion of the reverse of the request form.
- The Cytology Pap Smear Audit requires all relevant clinical boxes be selected.
- These aspects need to be adhered to as this data takes a different computer pathway through our system to give you all of your statistical information.
- **Please note** with the 2017 changes to cytology screening rapidly approaching, the QML Pathology Cytology Pap Smear Audit was designed from the outset to report your patients +HPV, Chlamydia, Gonorrhoea and Trichomonas so please include all relevant clinical pathology and include in the clinical tick box area.
- If you relocate surgeries or commence in a new practice please let the education team know so we can adjust/ add the audits to your QML Pathology Dr code.

- Please make sure we have your RACGP & ACRRM numbers at registration. Having your numbers at registration will guarantee a smooth transition for processing your achievements.

#### UPCOMING

Previously mentioned throughout 2015 we had quite a lot of computer changes behind the scenes to streamline and enhance our internal process. Some of our reporting was held up for a short time to allow the IT transition process to proceed. I would like to thank all clinicians for bearing with us over those couple of months whilst correlation and checking was carried out to ensure all statistical information was correlating. Along with these changes comes the very exciting move towards digital correspondence for all QML Pathology Education. Please notify us of 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 a hard copy is still available through QML Pathology.

Plans are already laid down for events throughout the state this year so please keep a look out for notifications in your area. QML Pathology looks forward to producing topical, appropriate and first hand medical education for our community.

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](mailto: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](mailto: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](mailto: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: \_\_\_\_\_

# Flu Season 2016 Fluvax<sup>®</sup> In Stock Order Yours Now!



Two types of influenza virus cause serious disease – influenza A and influenza B.

Disease caused by influenza A is typically more common, more severe and affects people of all ages. Importantly, influenza A tends to peak earlier in the season with influenza B being most dominant every 4-5 years.

Health Authorities have noted increasing episodes of influenza and emphasised the need for persons to be fully vaccinated against all circulating strains in the community, even if they have been vaccinated in previous years<sup>1</sup>.

## ABOUT QML PATHOLOGY VACCINES DEPARTMENT

We stock an extensive range of premier vaccines and offer exceptionally competitive pricing, including generous discounts for bulk purchases. As a certified cold chain distributor our integrated network ensures the integrity and quality of our products. We also provide next day delivery to doctors located within metropolitan and regional areas in Queensland and Northern New South Wales.

## TRIVALENT INFLUENZA VACCINE (TIV)

The 2016 Fluvax<sup>®</sup> Trivalent influenza vaccine is available from QML Pathology from the end of February 2016. With other brands of vaccinations not available until April, Fluvax<sup>®</sup> is one of the first vaccinations available to your patients for the 2016 flu season.

## WHAT IS IN THE VACCINE?

Each year Health Authorities recommend that 3 influenza strains are included in all influenza vaccines based on ongoing surveillance of the circulating virus<sup>1</sup>.

Over the past 10 years the 3 recommended strains have accounted for an average of 94% of all influenza cases reported in Australia<sup>2</sup>.

## FURTHER INFORMATION

For further enquiries regarding the influenza vaccination, please contact **QML Pathology Vaccines department** on **(07) 3121 4523**.

1: Australian Government Department of Health 'Clinical advice for immunisation providers regarding the administration of 2015 seasonal influenza vaccines' February 2015 [www.health.gov.au](http://www.health.gov.au)

2: Australian Government Department of Health Australian Influenza Surveillance Report 1 August to 14 August 2015 [www.health.gov.au](http://www.health.gov.au)



## Warfarin registrations over the Easter period

QML Pathology wishes to advise that the Warfarin Care Clinic will not be accepting any NEW REGISTRATIONS as from 5pm Wednesday, 16th March 2016.

This service will re-open from 8am Tuesday 29th March 2016.

Patients who are currently monitored by QML Pathology and are being discharged from hospital will still be accepted during this time.

## Doctors' Noticeboard

### Dr Fabio Brecciaroli

MBBS, FRACGP, Grad Dip Pall Med, FACHPM

After 40years I have retired from my full time hospital position. I will continue to be available for my private practice as a Palliative Care consultant.

### Sunshine Coast Palliative Care Consultancy

PO Box 966, Caloundra 4551

M: 0419722764 / F: 54923560

E: fabiopallcare@bigpond.com

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](mailto:info@qml.com.au)

### Dr Amy Tang

Dr Tang is a subspecialist gynaecologist and certified gynaecological oncologist. Dr Tang has recently opened her own private practice in the city.

### Gynaecology @ The Mill

Suite 116, Ground Floor, Watkins Medical Centre, 225 Wickham Terrace, Spring Hill, QLD 4000

P: 07 3319 1515 / F: 07 3319 1500

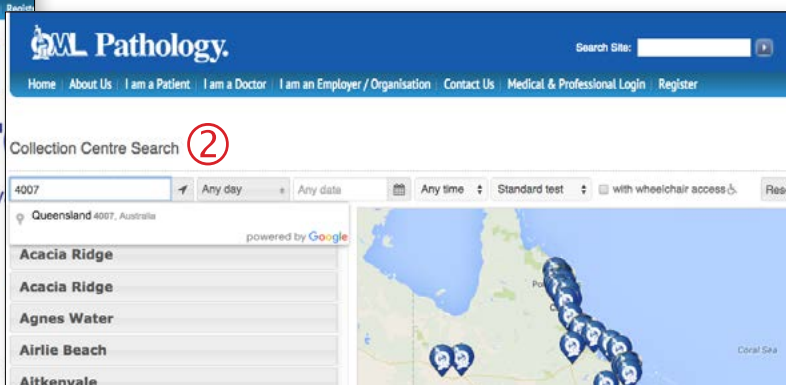
E: [info@themillgyn.com.au](mailto:info@themillgyn.com.au) / W: [themillgyn.com.au](http://themillgyn.com.au)

## 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](http://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).



## Infectious Diseases Report

GEOGRAPHIC DISTRIBUTION - DEC 2015

ORGANISM	Regions (as per key below)															TOTAL			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	DEC	NOV	OCT	SEP
Adenovirus (not typed)	1	17	7	1	1		15		5	2	23	9	1		3	85	124	157	217
Adenovirus (typing pending)		3	1				1			2	4	3	1	1		16	9	16	17
Barmah Forest virus		1							1			1				3	2	1	1
Bordetella pertussis	6	61	30	7			75	1	42	13	78	74	7	6	6	406	448	1328	1871
Brucella species			2	1				1	1				1	1		7	6	3	7
Campylobacter jejuni																0	0	0	1
Chlamydia pneumoniae																0	0	2	0
Chlamydia trachomatis, not typed	52	133	47	22	3		124		77	25	188	79	25	31	15	821	958	860	848
Coxiella burnetii	1	4	1			1			4				2			13	12	10	20
Cryptococcus species			1				1									2	4	2	1
Cytomegalovirus (CMV)	3	8	4		2		4		6	1	15	3	2	2		50	57	50	37
Entamoeba histolytica																0	0	0	1
Enterovirus - not typed																0	0	0	1
Epstein-Barr virus (EBV)	7	32	5	4	1		21		17	6	41	21	6	4	2	167	181	173	179
Flavivirus unspesified	2	2		1						2	2	1		1		11	9	11	12
Hepatitis A virus		1					1					1				3	8	5	3
Hepatitis B virus	7	7	11		2		16		5	2	48	1	4	1	1	105	150	160	147
Hepatitis C virus	20	43	32	16		1	39		24	9	84	33	18	10	25	354	356	398	377
Hepatitis D virus																0	2	0	0
Hepatitis E virus			1													1	1	0	2
Herpes simplex Type 1	20	78	24	13			51		44	9	70	35	10	5	20	379	383	376	399
Herpes simplex Type 2	6	43	16	9			23		12	7	44	20	8	6	12	206	242	213	194
Herpes simplex virus - not typed																0	0	0	0
HIV-1	1	8		1			4		1		3		1			19	13	21	14
HTLV-1																0	0	1	0
Human Metapneumovirus	6	14	5				28		13	3	25	19	3	7	5	128	213	254	442
Influenza A virus		9	1			1	8	2	2		9	7	1	5		45	74	189	939
Influenza B virus		7					1				2	1	3	5		19	21	174	2016
Legionella pneumophila (all serogroups)		4														4	7	3	2
Legionella species							1									1	1	2	8
Leptospira species			2						1		1	1				5	5	1	2
Measles virus																0	0	0	0
Mumps virus										2						2	1	4	0
Mycoplasma pneumoniae	5	57	22	7			39		29	4	56	24	14	11	7	275	376	400	413
Neisseria gonorrhoeae	9	9	3	1			9		8		14	4		2		59	73	77	66
Parainfluenza virus	1	16	2	1	1		21	1	13	5	26	14		5	6	112	198	215	505
Parvovirus		1					1		2	1	1	2				8	17	12	6
Pneumocystis carinii																0	2	4	2
Respiratory Syncytial virus	3	9	5	2			13		12	1	13	13	6	2	3	82	67	59	155
Rhinovirus (all types)	4	48	21	4	2		35		32	12	79	38	5	8	8	296	499	428	707
Rickettsia - Spotted Fever Group																0	4	3	4
Ross River virus	3	11	4	2	1		11		16	8	9	13		4	5	87	136	116	99
Rubella virus																0	0	2	0
Salmonella paratyphi A																0	0	0	0
Salmonella paratyphi B																0	0	0	0
Salmonella typhi							2									2	0	1	7
Streptococcus Group A	3	3	9	2			9	64	11	2	13	15	5	2	6	144	147	161	160
Toxoplasma gondii	1	1	2				2		3	1	1	1			1	13	13	14	21
Treponema pallidum	22	16	7	1	9	1	43	1	18	1	41	7	8	28	3	206	219	237	238
Trichomonas vaginalis	10	1	2		2		1		1	1	2			6		26	34	53	48
Varicella Zoster virus	12	52	13	5			45		27	3	55	32	10	16	14	284	336	323	313
Yersinia enterocolitica																0	0	0	0
TOTAL	205	699	280	100	24	4	644	70	427	120	949	472	141	169	142	4446	5408	6519	10502

## 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.