QML Pathology
# Newsletter

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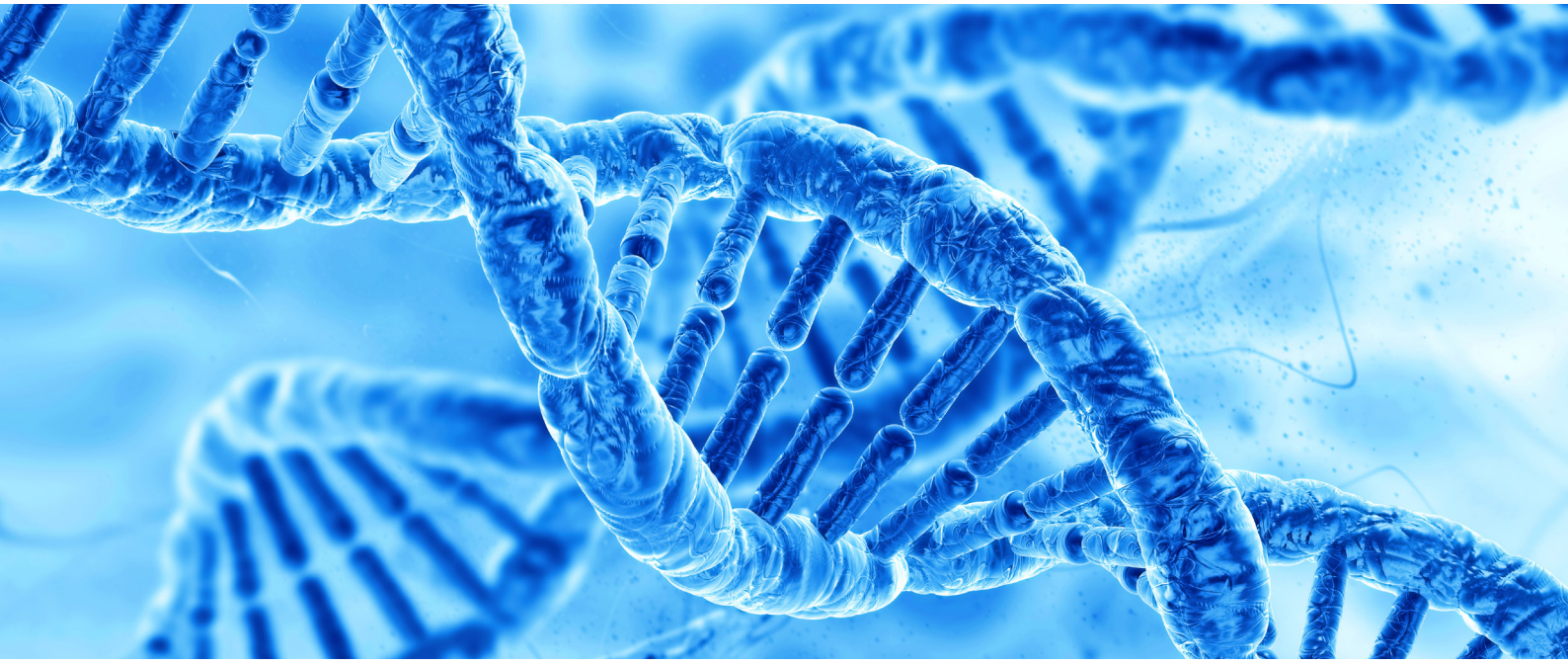
ISSUE 2, 2018



## Genomics in General Practice

### Dr Melody Caramins

Genomic medicine is the use of genomic information about an individual as part of their clinical care (e.g., for diagnostic or therapeutic decision-making). Genetic and genomic testing have for many years been part of mainstream diagnostic testing for specialists, but increasingly are being incorporated into everyday patient care in general practice.

The types of currently available genetic tests in human health are most usefully understood based on the affected clinical group and presentation (e.g. oncology, haematology, reproductive medicine, paediatrics), and the amount of genetic material studied and tested. Broadly speaking, genetic conditions are due to genetic variants, also known as mutations, that are either inherited, such as thalassaemia (known as germline mutations), or are acquired (known as somatic mutations). Acquired mutations often occur when there is rapid cell replication and division e.g. in cancer or during conception, leading to specific mutations in malignancies which may indicate appropriate treatments, or causing conditions such as Down syndrome in a fetus.

Genetics and genomics have a role in human health from pre-birth to death. In the diagnostic setting, this involves the detection of pathogenic genetic variation, to determine:

- Risk of future disease (e.g. carrier screening, predictive testing, PGS/D)
- Diagnose and classify current disease (e.g. oncology, adult and paediatric syndromes)
- Determine treatment (e.g. companion diagnostics, pharmacogenetics)

Therefore, the detection of pathogenic genetic variation in an affected person can have a multidimensional impact, by providing a molecular diagnosis and potentially negating further unnecessary tests, procedures and appointments; changing patient management plans; and, suggesting prognosis and/or treatment options. This impact extends to implications for other family members if testing for inherited (rather than acquired) genetic variation.

**>>> CONTINUED OVERLEAF**

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Some forms of testing may be associated with complex issues related to the potential for the identification of results with no clear significance, as well as to various ethical, legal and social issues. The probability of these issues is higher with some tests (e.g. predictive testing for Huntington disease or inherited cancer) and benefits and harms must be balanced by the provision of non-directive counselling and informed consent.

There are a number of clinical settings where genetic testing is more relevant. The following is a list of some of these settings, including tests that are more commonly encountered by GPs:

- Reproductive medicine – preconception carrier screening, non-invasive prenatal testing (NIPT), diagnosis of genetic causes for infertility or recurrent miscarriage.
- Paediatric disease: diagnosis of developmental delay – sometimes through specific mutation/gene testing (e.g. Fragile X), or through genomic tests, such as chromosome testing by microarray.
- Pharmacogenetic/pharmacogenomics tests. These have implications for treatment choice in neurology, pain management, oncology, and in the prediction of adverse drug reactions.
- Adult medicine – specific mutation/gene testing for inherited diseases of adult onset (many neurodegenerative disorders, familial cancer syndromes), or for acquired disorders in oncology and haemato-oncology. These tests are generally ordered by specialists, but GPs will be familiar with them (e.g. BCR/ABL translocation testing in chronic myeloid leukaemia (CML), predictive testing for Lynch Syndrome, and genetic testing for Huntington Disease).

Currently, very few genetic tests are funded through Medicare in Australia. Tests are accessed by patients in a variety of ways, although all Australian accredited laboratories are required to perform testing only in the context of a medical consultation – be this general or specialist medical practitioners.

GPs most often come into contact with genetic testing in the context of reproductive health, with NIPT and pre-conception carrier screening being most frequently encountered.

Carrier screening involves testing individuals or couples, ideally prior to pregnancy, to determine if they have a genetic variant that may affect their chance of having a child with a genetic condition. This testing has received increasing media coverage of late, due to announcement in February 2018 by the Minister for Health, Greg Hunt, indicating that the government intends to invest tens of millions of dollars to improving access to this testing for prospective parents. Clinical guidelines recommend testing for three of the more common conditions as a basic screen:

- Cystic Fibrosis, which has a carrier frequency in Australia ~1/25
- Fragile X Syndrome, with a carrier frequency in Australia ~1/150
- Spinal Muscular Atrophy, with a carrier frequency in Australia ~1/40

These conditions have been recommended because they are common, severe, and the tests to detect them use mature techniques which are robust, specific and sensitive. Most of the time, a family history will not be informative, due to the inheritance patterns and the rarity of these conditions individually. And yet, together, the combined collective carrier rate for these three disorders in the Australian population is 5%, or 1/20; with the combined affected pregnancy rate in Australia from these three disorders being equivalent to the population risk of Down syndrome.

Testing for carrier screening should ideally be discussed with patients and performed by GPs during the preconception period, in order to have the most time to deal with all possible testing outcomes. The recommended testing pathway is to initially test the female partner, and to only test her partner if she is found to be a carrier. If the couple are shown to be carriers for any of the conditions, then genetic counselling is recommended so that they can accurately discuss their risk, as well as get detailed information about all their reproductive options.

## TAKE HOME MESSAGES:

- Genetic and genomic testing is increasingly part of the landscape of testing in general practice, particularly in the reproductive health setting.
- GPs should discuss the option of carrier screening for common genetic disorders (SMA, Fragile X, Cystic fibrosis) as part of preconception planning with their patient;
- GPs ordering testing should ideally do this prior to pregnancy, with the female partner undergoing carrier screening first. Her partner should be tested if she is found to be a carrier of a serious genetic disorder.
- Genetic counselling is recommended to assist couples to help understand their risks after testing, and to enable them to make informed reproductive choices

## FURTHER INFORMATION

For further information, please refer to the RACGP Genomics in General Practice publication at

**www.racgp.org.au/your-practice/guidelines/genomics**

# Genetic Carrier Screen

## A test for Cystic Fibrosis (CF), Fragile X (FXS), and Spinal Muscular Atrophy (SMA)

The Genomic Diagnostics Carrier Screen tests for three genetic conditions that are relatively common in general populations: Cystic Fibrosis (CF), Fragile X (FXS), and Spinal Muscular Atrophy (SMA). These conditions were chosen for patients considering conception based on local and international genetic screening recommendations[1,3]

Many children affected by these conditions are born to families with no history of disease due to the relatively rare nature of the conditions and autosomal recessive or X-linked inheritance patterns. The value of carrier screening for CF and SMA has therefore been recognised for all patients in some countries such as the United States[1], with the value of Fragile X also being recently recognised in Australian research[2].

Importantly, if couples are found to be carriers of these conditions, they can consider several reproductive options including:

- natural pregnancy, with or without prenatal diagnosis
- preimplantation genetic diagnosis (PGD) with in vitro fertilization (IVF) to test and then transfer embryos that are free of the condition
- adoption
- the use of a sperm or egg donor

### BACKGROUND

**What is Cystic Fibrosis?** Cystic Fibrosis is an autosomal recessive genetic condition caused by a malfunction in the exocrine system responsible for producing saliva, sweat and mucus. This can result in a variety of symptoms, from mild (pancreatic sufficient) to severe (pancreatic insufficient), affecting mostly the respiratory and digestive systems.

**What is Fragile X syndrome?** Fragile X is an X-linked genetic condition causing intellectual disability, behavioural and learning challenges and various physical characteristics. It is also the most common single gene cause of autism worldwide (accounting for up to 5% of all cases) and the most common genetic cause of intellectual disability in males. Although Fragile X Syndrome occurs in both sexes, males are generally affected with greater severity than females.

**What is Spinal Muscular Atrophy:** Spinal Muscular Atrophy (SMA) is an autosomal recessive condition that results in the loss of motor neurones in the spinal cord and is classified as a motor neurone disease. The primary symptom is weakness of the voluntary muscles. In the most common form of SMA, due to mutations on chromosome 5, there is wide variability in age of onset, symptoms and progression rate.

### HOW COMMON ARE THESE CONDITIONS?

These three conditions combined are amongst the most commonly carried mutations in European populations.

| | CARRIER FREQUENCY | NUMBER OF LIVE BIRTHS |
|---|---|---|
| Cystic Fibrosis | 1 in 25 | 1 in 2,500 |
| Fragile X | 1 in 150 | 1 in 4,000 males<br>1 in 8,000 females |
| Spinal Muscular Atrophy | 1 in 40 | 1 in 6,000 – 10,000 |

### MODE OF INHERITANCE

| | MODE OF INHERITANCE |
|---|---|
| Cystic Fibrosis | Autosomal recessive |
| Fragile X | X-linked |
| Spinal Muscular Atrophy | Autosomal recessive |

**Autosomal recessive inheritance:** A mode of inheritance such that an individual must have a mutation in both copies of the specific disease gene, usually one inherited from each parent, to express the genetic condition.

**X-linked inheritance:** A mode of inheritance in which a mutation on the X chromosome causes the expression of the genetic condition. Males are typically affected, as they only have one X chromosome, whereas females may show variable expression of the condition due to differences in X chromosome inactivation. As Fragile X is a dominant mutation, females can be affected, but at approximately half the rate of males (50% chance of female receiving normal allele from mother). Mutations are typically inherited from a mother who is an unaffected carrier of the mutation.

### ASSAY PERFORMANCE

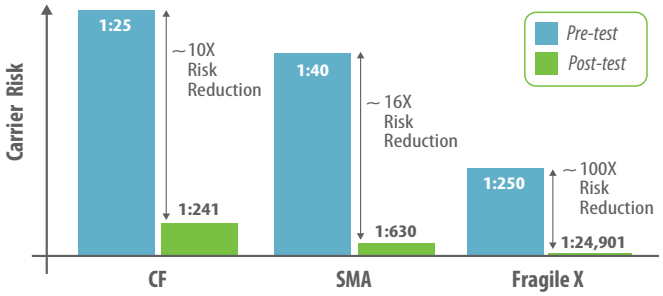
This assay tests for the most common genetic changes associated with FXS, CF and SMA. The assay can detect:

- 90% of Cystic Fibrosis carriers*
- 99% of Fragile X carriers
- 95% of Spinal Muscular Atrophy carriers

The test cannot detect everyone who is a carrier as rarer mutations cannot be detected by the assay. Therefore, the use of this assay in a carrier screening setting can significantly reduce the risk of a couple having an affected child but cannot remove this risk completely.

*Based on European population data

## EXAMPLE CARRIER RISK REDUCTION




1. Caucasian population - frequencies will vary for other ethnic groups.
2. Calculations are based on reported test sensitivity CF: 90% SMA: 95% and Fragile X: 99%

## WHICH CYSTIC FIBROSIS MUTATIONS ARE TESTED FOR BY THE ASSAY?

Since the discovery of the CFTR gene in 1989, more than 1900 mutations and variants have been described. Many of these mutations have been described only in one patient and/or family. Routine testing for all possible mutations is neither feasible nor cost effective and therefore testing is confined to the most common mutations.

The Genomic Diagnostics' carrier screening test identifies 50 of the most common mutations in European populations. This exceeds both Human Genetics Society of Australia (HGSA) and American College of Medical Genetics (ACMG) guideline recommendations for the 29 most frequently occurring mutations.

## WHEN SHOULD PATIENTS BE TESTED?

The ideal setting for carrier screening is preconception, in order to have the most time to deal with all possible testing outcomes. However, testing can also be used in an antenatal setting.

## GENETIC COUNSELLING

If the couple are shown to be carriers for any of these conditions, then genetic counselling is recommended so that they can get more information and discuss in detail their options and potential impacts of their situation.

## HOW TO ORDER?

The recommended testing pathway is to initially test the **female partner**, and to only test her partner if she is found to be a carrier.

**Please note:** This test is not covered by private health insurance or Medicare.

1. **Considering genetic testing**
   Discuss with the patient whether or not to have the test as recommended by clinical guidelines.
2. **Request the test**
   If you would like to order the test, you can use your standard pathology request form. Simply write a request for a 'Genetic Carrier Screen' and return to us.
3. **Patient Pays for the Test**
   The patient calls our customer care team on **1800 822 999** to pay for the test and is given a receipt number and a collection location
4. **Results return**
   It will take approximately 14 working days (from receipt of sample) for results to be available. Results will be returned by your preferred method.

## FURTHER INFORMATION

Please call 1800 822 999 or
email info@genomicdiagnostics.com.au

## REFERENCES

1. ACOG Committee Opinion, No. 690, March 2017. "Carrier Screening in the Age of Genomic Medicine".
2. Metcalfe, S.A. Genetics In Medicine, 2017. "Informed decision making and psychosocial outcomes in pregnant and non-pregnant women offered population Fragile X carrier screening".
3. Prenatal screening and diagnosis of chromosomal and genetic abnormalities in the fetus in Pregnancy. RANZCOG. C-obs 59.

# Fragile X

## Dr Melody Caramins

## WHAT IS FRAGILE X?

Fragile X syndrome is the second most common genetic cause of intellectual disability after Down syndrome. It is a syndrome characterised by a pattern of physical, behavioural and/or intellectual signs and symptoms, which together describe the features of this condition. Fragile X syndrome is caused by the expansion of three DNA letters – CCG – (this is also known as a DNA triplet), occurring near a gene called the FMR-1 gene (Fragile X Mental Retardation), which is on the X chromosome. The number of times that the 'CGG' triplet is repeated creates different lengths of the repeat sequence and contributes to different presentations. The syndrome is an example of X-linked inheritance, where the disease presents differently in men and women, as men have one copy of the gene (and one X chromosome) and women have two copies (and two X chromosomes).

The differing CGG triplet repeat lengths are categorised as follows:

- **A short repeat sequence** is seen in most people, and is considered the "normal" length. The 'CGG' triplet is repeated between 6 and 54 times; the repeat numbers are variable in different families, with the most commonly seen repeat length being about 30. A high normal repeat length (between 44-54 repeats), is also known as "the grey zone". These repeat lengths were not thought to be associated with any clinical features. More recent studies suggest this may not always be the case, although reports have been inconsistent and research in this area continues.
- **A medium repeat sequence**, known as a pre-mutation, is seen in some men and women where the 'CGG' triplet is repeated between about 55 and 200 times. People with a pre-mutation are not affected intellectually, although they are at higher risk of having neurological or reproductive sequelae, such as Fragile X Tremor/Ataxia Syndrome (FXTAS), and Premature Ovarian Insufficiency (POI). FXTAS is a progressive neurological condition which generally develops after the age of 50, with increasing risk with age. The risk of developing FXTAS in men with pre-mutations is approximately 50% by the time they reach 80. The risk of premature ovarian failure and early menopause for women who carry a pre-mutation is ~20%. In addition to this risk, pre-mutation carriers are also at risk of having children with Fragile X Syndrome, and should always be referred for genetic counselling with a qualified professional.
- **A long repeat sequence**, also called a full mutation, is where the 'CGG' triplet is repeated more than 200 times. This inactivates the FMR-1 gene, leading to Fragile X Syndrome. Males will generally be more severely affected and have the following features, which may vary in severity: Developmental delay, behavioural/emotional problems, defining physical characteristics such as large ears and a long face, and other medical conditions such as epilepsy. In females, due to the second "normal length" copy of the FMR-1 gene, features are much milder, but may include mild intellectual disability (up to 60%), as well as some emotional or behavioural disturbances.

Delayed diagnosis is common, with many individuals not being diagnosed until school age or later. It has been estimated that in 50% of families, by the time a child is diagnosed, the family has already had a second affected child.

## HOW COMMON IS FRAGILE X?

The prevalence of the Fragile X pre-mutation is more common, occurring in one in 800 males, and more often in females, with one in 150 in the general population being at risk of having a child with the full mutation. The estimated prevalence of individuals with the full mutation is about one in 3600 males and one in 4000–6000 females.

## TESTING: WHEN, HOW, AND WHAT TO DO ABOUT RESULTS

Fragile X testing should be considered/offered in the following clinical situations:

Diagnostic testing should be offered to patients who have any of the symptoms consistent with Fragile X; any individual, male or female, of any age with intellectual disability, developmental delay, or learning disability and features of FXS including anxiety, ADHD or autism spectrum disorder. Additionally, adult patients with ataxia, or women with premature ovarian insufficiency should also be tested. In all these patients, testing is funded by Medicare, and clinical details should be provided on the request form.

Cascade testing of at-risk relatives should also be offered to asymptomatic relatives who have a family member with an FMR-1 mutation. This testing is also rebated under Medicare.

Carrier screening, the option of testing for carrier status for Fragile X has been recommended by RANZCOG for all women prior to, or early in pregnancy, in order to provide the widest range of family planning options. Knowledge of carrier status offers the full spectrum of reproductive options, including the choice of becoming pregnant naturally (with or without testing in pregnancy), adoption, or using assisted reproduction techniques including egg or embryo donation and pre-implantation genetic embryo testing.

Testing for carrier status where there is no family history of Fragile X is not funded by Medicare. Carrier screening can be undertaken for Fragile X only, or can be combined with screening for other common genetic disorders (such as cystic fibrosis and spinal muscular atrophy).

Pre-implantation or prenatal testing involves testing of the embryo or fetus of women who are carriers of a Fragile X mutation (either a pre-mutation or a full mutation). This testing is generally requested in a hospital or IVF setting by a specialist team, although GPs should be aware of these options and a discussion around them should be part of genetic counselling for women who are found to be carriers.

>>> CONTINUED OVERLEAF

The pathology request should indicate 'DNA testing for Fragile X syndrome', and any relevant clinical and family history. In addition, any individual with developmental disability who has not previously been fully assessed should also be tested by 'chromosome analysis by genome-wide microarray'. This test detects other, chromosomal causes of developmental delay. This test does not detect Fragile X syndrome, although it may detect other causes of developmental and intellectual delay when being assessed for the first time.

Fragile X is an inherited condition, likely to affect multiple family members. GPs should identify all at-risk patients in a family using genetic testing. This especially applies to females of child-bearing age, who will need to know their carrier status in order to restore reproductive confidence and choice in decisions about future pregnancies. As indicated above, genetic counselling is strongly recommended for patients whose results indicate a longer than normal triplet repeat length.

### TAKE HOME MESSAGES

- Fragile X is the most common known inherited cause of developmental disability.
- Presenting features can include anxiety and autism spectrum disorder.
- Although males and females of all ages can be affected, in females the presenting symptoms are often attenuated by the presence of a second gene, on the other X chromosome, with a normal length CGG repeat.
- Long expansions (full mutations) of the CGG triplet repeat are associated with Fragile X syndrome, whereas shorter expansions (pre-mutations) are associated with FXTAS and POI, and a risk of having children affected with Fragile X syndrome or associated conditions.
- Ensure DNA testing for Fragile X is included in assessment of all cases of developmental disability and autism.
- Genetic counselling is recommended for all patients who are found to carry a longer than normal length triplet repeat.

## Doctor Noticeboard

**Dr Mahomed Khatree**
MBChB FCOG (SA) FRCOG (London) FRANZCOG

Dr Khatree (Obstetrics, Gynaecology and Fertility) would like to inform colleagues that he has relocated his Sunnybank consulting suites to Suite 211, Level 2, Times Square, 250 McCullough Street, Sunnybank. Appointments are usually available within a week and there is no gap payment for privately insured patients for deliveries or gynaecological operations.

P: 07 3345 8384 / F: 07 3344 3429

**Dr Andrea Stimming** FRANZCP FAChAM

Dr Andrea Stimming is a consultant psychiatrist and addiction medicine physician who admits to the Damascus Unit at Brisbane Private Hospital. She understands substance use disorders can be counterintuitive and frustrating to those that suffer and their supports. She takes a holistic approach to understanding her patients and what treatment is most appropriate for their life. She has an interest the treatment of young adults as well as the co-occurrence of addictions and other mental health problems.

P: 0490 846 890 / F: 07 3532 5144
E: contact@drstimming.com.au

**Dr Justin Gundara** MANZES ANZHPBA

Dr Justin Gundara is an endocrine, HPB and general surgeon with a special interest in neuroendocrine disease. Having completed a PhD in thyroid cancer and two clinical fellowships, he has settled in Brisbane and maintains academic posts with UQ, Griffith University, membership of ANZ Endocrine Surgeons, ANZHPBA and sits on the RACS Endocrine Surgery Section Committee. While having a dedicated interest in endocrine (thyroid, parathyroid, adrenal, pancreas) surgery he is also happy to assist with all general surgical concerns through Specialist Services.

P: 07 3226 3800 / F: 07 3236 9485

**Dr Tzu-yang Wang** MMBBS (Hons) FRACP FRCPA B.Phty

Dr Wang is a Brisbane Haematologist, specialising in the diagnosis and management in both malignant and non-malignant haematological problems. Consulting at Greenslopes, Wesley and Sunnybank Private Hospitals. Access to day Therapy units with rapid and personalised management including iron infusion, blood transfusions, venesections and administration of chemotherapy. Dr Wang has a no- gap agreement with most health funds. Fluent in English and Mandarin.

P: 07 3833 6747 / E: info@hqclinic.com.au

**Dr Sudeep** MBBS FRACP

Dr Sudeep is a Paediatric Endocrinologist and General Paediatrician. She currently works as a staff specialist at the Caboolture Public Hospital and is looking forward to starting and establishing paediatric endocrine services at North Lakes. Sudeep has joined the practice of North Lakes Endocrinology allowing patients to access specialist care locally. She has a special interest in type 1 diabetes and insulin pump therapy. Her areas of expertise and interest include: growth disorders, thyroid hormone disorders, puberty and sexual development disorders, pituitary disorders and childhood obesity.

P: 07 3448 0195 / F: 07 3448 0196

**Dr Andrew Hadj** MBBS FRACS (Plast.)

Dr Andrew Hadj is a well-versed plastic surgeon who specialises in hand surgery, degenerative joint conditions, skin cancer surgery, breast and reconstructive surgery, restorative facial surgery and upper and lower limb reconstructive trauma surgery.

After completing his degree in medicine and medical training in Victoria, he went on to further his specialised training with multiple hospital positions.

Dr Hadj has travelled extensively for his training and worked across a variety of clinical settings, allowing him to become well-rounded and adaptable in a variety of situations.

P: 07 3488 8118 / E: info@valleyplasticsurgery.com.au

**Dr Fraser Wright** MBBS FRACP FRCPA BSc

Dr Wright is a Clinical & Laboratory Haematologist. He has a diverse interest in the diagnosis and management in both malignant and non-malignant haematological issues. Dr Wright combines laboratory sessions at QML Pathology as well as private consultation and access to day therapy units at the Wesley and Canossa Hospitals. He is a passionate and personable Haematologist whose priorities centre on providing his patients with the best possible care.

P: 07 3833 6747 / E: info@hqclinic.com.au

**Dr Davoren's Paediatric Practice**

Dr Davoren's Paediatric Practice at Stafford Heights would like to welcome the following sessional specialists to his rooms; Dr Christopher Whight, Paediatric Cardiologist, Dr Kerry Buchanan, Paediatric Endocrinologist, Victoria Alexander, Accredited Mental Health Social Worker for children, teens and families. Please contact Dr Davoren's rooms to enquire about availability and schedule appointments.

P: 07 3350 1722 / E: reception@nwpg.net.au

**Dr Vishnu Sannarangappa** MBBS FRACP

Dr Sannarangappa is an Endocrinologist and General Physician who trained in India in 1998. Vishnu is practicing at the North Lakes Endocrinology within the new North Lakes Specialist Medical Centre. He is also a staff specialist Endocrinologist and General Physician at the Caboolture Public Hospital as well as a Clinical Senior Lecturer at UQ. Dr Vishnu provides patient-centred care in all areas of general endocrine conditions including diabetes, metabolic bone diseases, thyroid and obstetric endocrinology.

P: 07 3448 0195 / F: 07 3448 0196
W: northlakesendocrinology.com.au

**Dr Rakesh Malhotra** MD DM MRCP (UK) FRACP

Dr Malhotra is a Consultant Endocrinologist who completed his training at the University of Mumbai, India. He has recently joined the practice at North Lakes Endocrinology within the North Lakes Specialist Medical Centre. He is also a senior staff specialist at the Caboolture Public Hospital and a Senior Lecturer in Medicine at UQ. Dr Halhotra strives to provide patient centred, evidence-based care to all of his patients with an interest in diabetes, thyroid and metabolic bone diseases.

P: 07 3448 0195 / F: 07 3448 0196

**Dr Swapna Devadula** MBBS FRACP

Dr Devadula is a Rheumatologist and General Physician who trained in the UK prior to obtaining fellowship with the RACP in 2016 and specialist rheumatologist in 2017. She has recently joined the practice at North Lakes Endocrinology within the North Lakes Specialist Medical Centre. She is also a rheumatologist at the Caboolture Public Hospital. Swapna is interested in all aspects of rheumatology and provides a holistic approach to all age groups of patients, including young adults transitioning into adulthood as well as women of child bearing age.

P: 07 3448 0195 / F: 07 3448 0196

**Dr Sarika Bhadange** MBBS FRANZCOG

Dr Sarika Bhadange is an experienced Obstetrician & Gynaecologist who offers complete care for women of all ages. She has a special interest in managing high risk pregnancies and offers an evidence based obstetric care.

She is passionate about adolescent health and has completed a fellowship year in paediatric and adolescent gynaecology at the Lady Cilento and Royal Brisbane Hospitals. She operates through St Andrew's Ipswich Private and Mater Private Hospital, Springfield..

P: 07 3444 4870
W: http://www.excelwomancare.com.au/

# Infectious Diseases Report

## GEOGRAPHIC DISTRIBUTION - MAR 2017

| 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 |
| Adenovirus (not typed) | 1 | 11 | 11 | | | | 23 | | 11 | 11 | 31 | 7 | 1 | 2 | 1 | 110 | 79 | 85 |
| Adenovirus (typing pending) | | | 3 | | | | 1 | 1 | 4 | 1 | 1 | 1 | | | | 12 | 8 | 16 |
| Barmah Forest virus | 2 | | | | | | 2 | | | | 1 | | | 1 | | 6 | 1 | 5 |
| Bordetella pertussis | 4 | 17 | 9 | 2 | | | 15 | | 6 | 2 | 17 | 34 | 9 | 6 | 4 | 125 | 146 | 148 |
| Brucella species | | | 2 | | | | | | | | | | | | | 2 | 8 | 5 |
| Campylobacter jejuni | | | | | | | | | | | | | | | | | | |
| Chlamydia pneumoniae | | | | | | | | | | | | 1 | | | | 1 | | |
| Chlamydia trachomatis, not typed | 72 | 92 | 66 | 24 | 7 | 1 | 131 | | 84 | 50 | 266 | 78 | 37 | 40 | 16 | 964 | 966 | 977 |
| Coxiella burnetii | 2 | 1 | | 4 | | 1 | 1 | | 1 | 2 | | | 1 | | | 13 | 19 | 12 |
| Cryptococcus species | | 1 | 1 | | | | 1 | | | | 3 | | 1 | | | 7 | 3 | 7 |
| Cytomegalovirus (CMV) | 2 | 12 | 3 | 2 | | | 10 | | 18 | 2 | 18 | 7 | 1 | 6 | 5 | 86 | 54 | 64 |
| Entamoeba histolytica | | | | | | | | | | | | | | | | | | |
| Enterovirus - not typed | | 1 | | | | | | | | | | | | | | 1 | | |
| Epstein-Barr virus (EBV) | 7 | 28 | 11 | 4 | | | 30 | | 15 | 3 | 43 | 16 | 3 | 7 | 10 | 177 | 175 | 164 |
| Flavivirus unspecified | | | | | | | 3 | | 4 | | 1 | | | | | 8 | 9 | 14 |
| Hepatitis A virus | | 1 | | | | | | | 1 | | 4 | | | | | 6 | 4 | 2 |
| Hepatitis B virus | 9 | 10 | 7 | 1 | | | 17 | | 3 | 3 | 73 | 5 | | 5 | 1 | 134 | 158 | 144 |
| Hepatitis C virus | 25 | 44 | 36 | 9 | 2 | | 35 | | 40 | 11 | 104 | 32 | 13 | 9 | 19 | 379 | 398 | 339 |
| Hepatitis D virus | | | | | | | | | | | | | | | | | 1 | |
| Hepatitis E virus | | | | | | | 1 | | | | 1 | | | | | 2 | 2 | 1 |
| Herpes simplex Type 1 | 12 | 58 | 29 | 10 | | | 45 | | 23 | 12 | 123 | 51 | 11 | 12 | 14 | 400 | 381 | 465 |
| Herpes simplex Type 2 | 12 | 33 | 18 | 2 | | | 33 | | 23 | 9 | 57 | 32 | 5 | 7 | 7 | 238 | 283 | 248 |
| Herpes simplex virus - not typed | | | | | | | | | | | | | | | | | | |
| HIV-1 | | 3 | | | | | 2 | | | | 4 | | | | | 9 | 26 | 21 |
| HTLV-1 | | | | | | | | | | | | | | | | | | |
| Human Metapneumovirus | 6 | 12 | 4 | 2 | 2 | | 12 | | 5 | 11 | 16 | 24 | 3 | 4 | 2 | 103 | 72 | 135 |
| Influenza A virus | 8 | 39 | 9 | 10 | | | 30 | 2 | 19 | 2 | 34 | 28 | 9 | 26 | 1 | 217 | 232 | 205 |
| Influenza B virus | 17 | 32 | 3 | 7 | | | 37 | | 16 | 5 | 45 | 33 | 5 | 7 | 1 | 208 | 170 | 167 |
| Legionella pneumophila (all serogroups) | | | 1 | | | | 1 | | | | | 1 | | | | 3 | 1 | 1 |
| Legionella species | | | | | | | | | | | | | | | | | 1 | 2 |
| Leptospira species | 2 | | 1 | | | | | | | | 1 | | | | | 4 | 1 | 2 |
| Measles virus | | | | | | | | | | | | | | | | | 2 | |
| Mumps virus | | | | | | | | | | | | | | | | | 3 | 1 |
| Mycoplasma pneumoniae | 2 | 17 | 2 | 2 | 1 | | 10 | | 4 | 4 | 22 | 8 | 1 | 1 | 4 | 78 | 86 | 94 |
| Neisseria gonorrhoeae | 11 | 5 | 9 | | 2 | | 17 | | 7 | 2 | 40 | 7 | 1 | 3 | 2 | 106 | 98 | 89 |
| Parainfluenza virus | 10 | 25 | 11 | 2 | 2 | | 25 | | 28 | 4 | 42 | 26 | 9 | | 7 | 191 | 109 | 147 |
| Parvovirus | | 4 | 2 | | | | 1 | | 1 | | 11 | 3 | | 2 | | 24 | 21 | 22 |
| Pneumocystis carinii | | | | | | | | | | | | 1 | | | | 1 | | 1 |
| Respiratory Syncytial virus | 31 | 50 | 22 | 5 | 1 | | 46 | | 39 | 14 | 92 | 57 | 4 | 16 | 2 | 379 | 222 | 153 |
| Rhinovirus (all types) | 10 | 51 | 15 | 11 | | | 59 | | 52 | 23 | 74 | 57 | 24 | 34 | 3 | 413 | 364 | 151 |
| Rickettsia - Spotted Fever Group | | | | 1 | | 1 | 1 | | 2 | | | | | | | 5 | 3 | 3 |
| Ross River virus | 12 | 10 | 4 | 1 | | | 8 | | 10 | 13 | 8 | 23 | 3 | 6 | 12 | 110 | 95 | 56 |
| Rubella virus | | | | | | | | | | | | | | | | | 2 | 1 |
| Salmonella paratyphi A | | | | | | | | | | | | | | | | | 1 | |
| Salmonella paratyphi B | | | | | | | | | | | | | | | | | | |
| Salmonella typhi | | 1 | | | | | | | | | 1 | | | | | 2 | | 1 |
| Streptococcus Group A | 2 | 10 | 4 | 3 | | 1 | 12 | 70 | 13 | 6 | 17 | 10 | 4 | 4 | 5 | 161 | 155 | 160 |
| Toxoplasma gondii | 2 | 6 | 1 | 2 | | | 7 | | | 1 | 3 | 8 | 1 | | 2 | 33 | 35 | 67 |
| Treponema pallidum | 41 | 13 | 11 | 5 | 9 | | 85 | 2 | 17 | 11 | 68 | 9 | 5 | 34 | 8 | 318 | 313 | 336 |
| Trichomonas vaginalis | 11 | 2 | 1 | | | 1 | 1 | 2 | 2 | 1 | 5 | 1 | | 5 | | 32 | 49 | 43 |
| Varicella Zoster virus | 19 | 50 | 35 | 4 | 1 | | 52 | 1 | 35 | 7 | 90 | 45 | 7 | 7 | 4 | 357 | 363 | 344 |
| Yersinia enterocolitica | | | | | | | | | | | | | | | | | | |
| TOTAL | 332 | 639 | 331 | 113 | 27 | 5 | 754 | 78 | 483 | 210 | 1316 | 605 | 158 | 244 | 130 | 5425 | 5119 | 4898 |

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.



Specialist Diagnostic Services Pty Ltd (ABN 84 007 190 043) t/a QML Pathology PUB/MR/001 (Apr-18)