

Interpretation of Iron Studies

Dr Fraser Wright, Dr Polly Higgins and Dr Yin Yuan

INTRODUCTION AND IRON METABOLISM

Iron is an essential element that serves many important biological functions. It acts as a co-factor for many enzymes, participates in electron transfer reactions, and plays a role in host defence. One of the critical functions of iron is the transport of oxygen as part of haemoglobin in red cells and myoglobin in muscles – principally skeletal and cardiac muscle. In contrast, excess iron can be harmful and so tight regulation of iron balance is required.

The average human body contains approximately 3-4g of iron, of which 1.5-2g is found in erythrocytes (blood and marrow erythroid cells) as haemoglobin. Under normal conditions dietary iron is usually 15-25mg daily of which 5-10% (1-2mg) is absorbed in the duodenum. It is transferred in blood bound to the transport protein transferrin. Excess iron can be stored in the liver as ferritin. The reticuloendothelial system (RES) acts as a secondary storage site and is involved in the recycling of iron. Outside of erythrocytes approximately 1g of iron is stored in the liver, and 0.4g as myoglobin¹ (See figure 1).

The hormone regulating systemic iron homeostasis is hepcidin, which is produced by the liver. Hepcidin down-regulates the cellular iron exporter ferroportin, which is found in enterocytes, hepatocytes and macrophages. Ferroportin controls the release of iron into the circulation. When it is downregulated iron absorbed into enterocytes will remain within the enterocytes until they are lost by desquamation. Low ferroportin also prevents release of iron stores from the RES. Thus, hepcidin acts as a negative regulator of iron by decreasing the absorption of dietary iron and decreasing the flow of stored and recycled iron into plasma. Hepcidin is up-regulated in states of iron excess and inflammation and down-regulated in states of hypoxia and increased erythropoiesis. There is no mechanism to control iron excretion, with iron losses occurring primarily through the sloughing of gastrointestinal mucosal cells or in the form of blood loss such as menstruation in women² (See figure 2).

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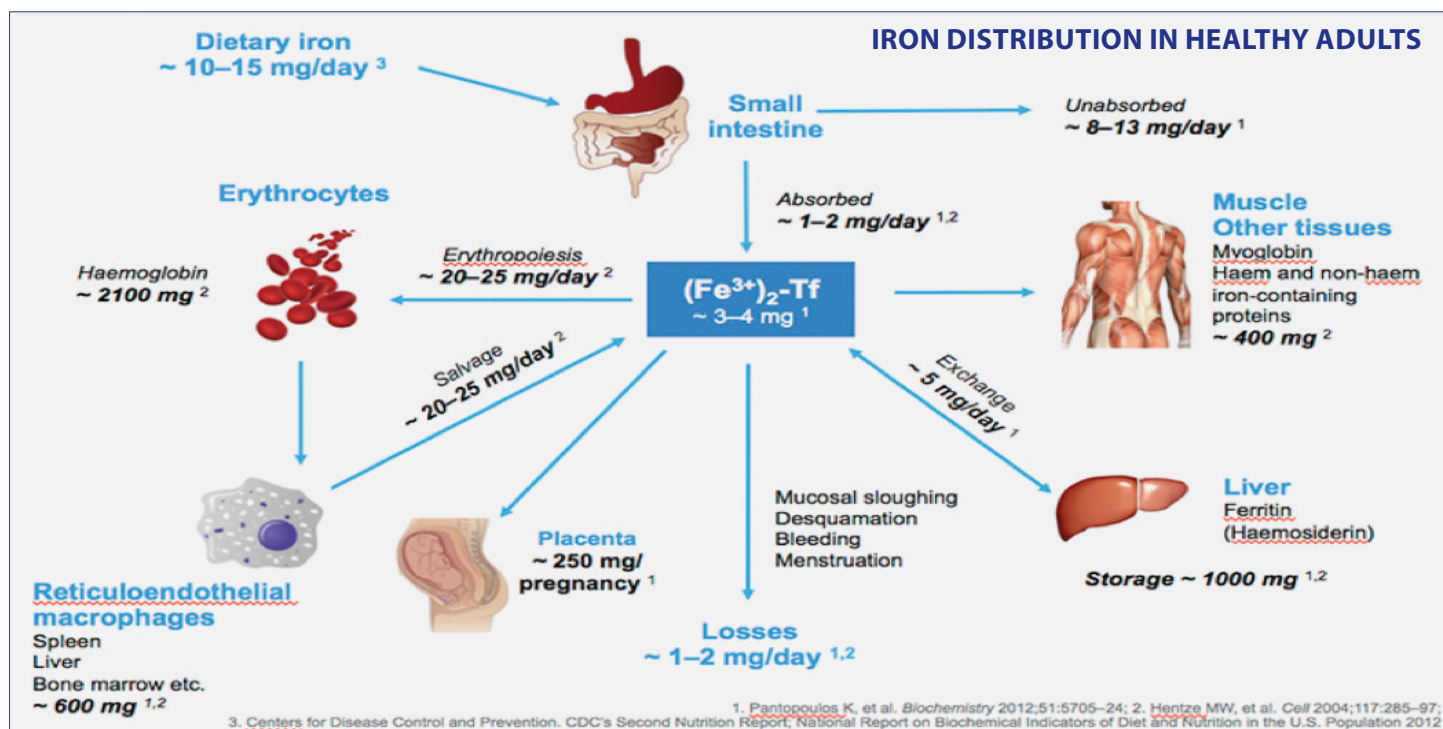


Figure 1: Compact Renal Learning Module 1: Physiology of iron metabolism 2014

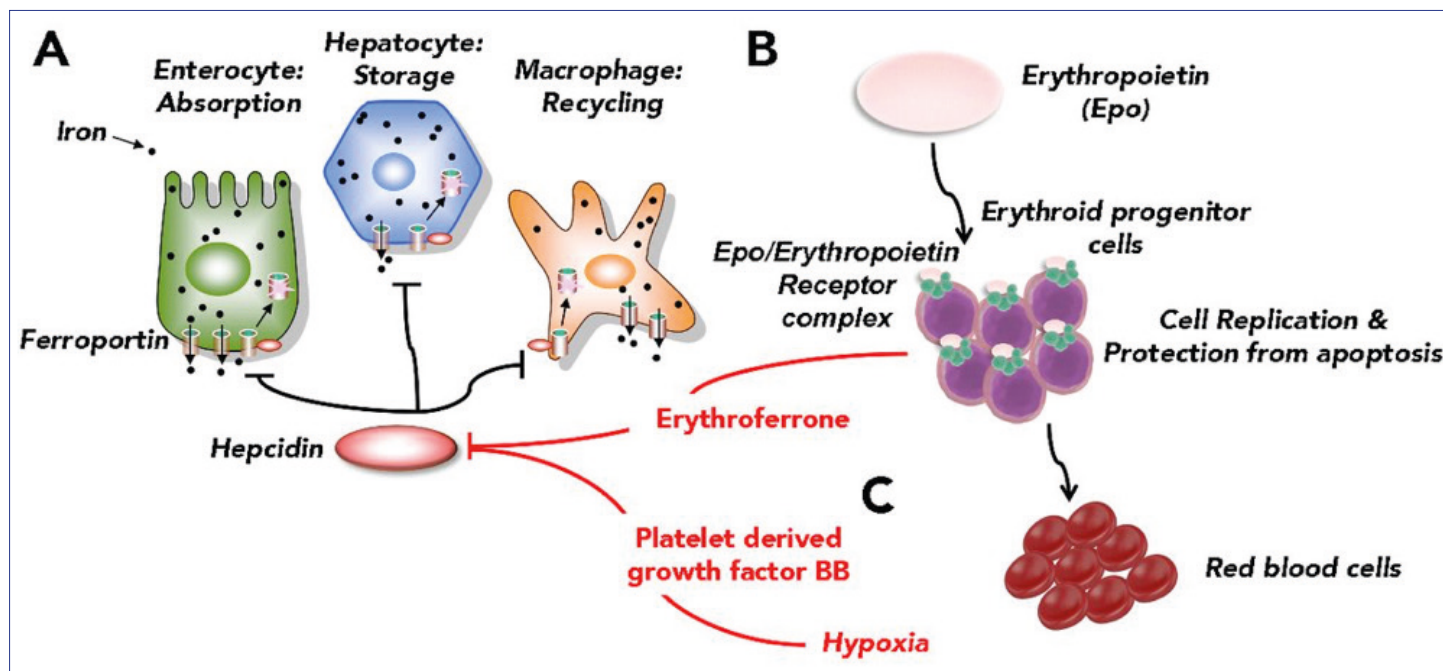


Figure 2: Stefano Rivella Blood 2019 133:51-58

IRON DEFICIENCY

Iron deficiency is the most common cause of anaemia worldwide. In Australia, 10% of women of child bearing age are iron deficient. Males have a third of the risk as females. Indigenous and refugee populations are also at higher risk of iron deficiency anaemia. Iron deficiency is also common in children especially in the toddler age group (1-6%).¹

Definitions

- **Iron Deficiency (ID)** - refers to a decrease in the amount of iron stored in the body and is defined by a serum ferritin level below 25ug/L in females (this also applies to pregnancy) or below 30mg in males, or less than 15ug/L in pre-pubescent children. Values between 25-30 ug/L in females should be considered as borderline deficiency with limited iron reserves.

- **Anaemia** - defined as Hb <130g/L in men and <115g/L in non-pregnant adult females. In pregnancy haemoglobins down to 105g/L are quite common. In the elderly a Hb < 125g/L and < 110 g/L in males and females respectively may be accepted as defining anaemia.
- **Non-anaemic Iron Deficiency (NAID)** - occurs when iron stores are low, however have not been depleted to the point where erythropoiesis is impaired. As the ferritin level falls, transferrin levels increase to improve iron uptake.
- **Iron Deficiency Anaemia (IDA)** - is diagnosed by anaemia in the setting of iron deficiency, usually accompanied by a drop in the mean cell volume (MCV).

Causes of Iron Deficiency^{3,4}

Increased iron loss (blood loss)

- GI sources: inflammatory bowel disease, malignancy, oesophagitis, gastritis, angiodysplasia
- Menstruation/ menorrhagia
- Pulmonary (haemoptysis, pulmonary haemosiderosis), urological disorders (haematuria) or nasal disorders
- Iatrogenic from repeated blood donation or clinical blood draws
- Dialysis
- Haemoglobinuria from intravascular haemolysis e.g. PNH, valve haemolysis

Nutritional deficiency (Decreased iron intake)

- Patients with inadequate dietary intake such as vegetarian/vegan diets, elderly, low socioeconomic status, excessive alcohol intake

Malabsorption (Decreased iron absorption)

- Gastrointestinal disease or surgery e.g. Coeliac disease, inflammatory bowel disease, achlorhydria, autoimmune atrophic gastritis/ H pylori colonization
- Congenital cause e.g. iron refractory iron deficiency anaemia, hereditary hypotransferrinaemia, DMT1 disease (all extremely rare)

Increased utilisation

- Growing infants, children and adolescents
- Expanded Red Cell Mass – e.g. EPO therapy or True Polycythaemias.

Please note: Dietary and GI causes of iron deficiency are also a cause of B12/Folate deficiency and these should always be assessed even if the FBC appears to be a case of classic iron deficiency.

Functional Iron Deficiency

Functional iron deficiency (FID) is a state in which there is insufficient iron incorporation into the haemoglobin of erythroid precursors in the face of apparently adequate body iron stores, as defined by the presence of stainable iron in the bone marrow together with a serum ferritin

value within normal limits. In a broader sense this may extend to inadequate iron incorporation into the heme complex in muscle myoglobin.⁸

Diseases that are affected by a state of FID include:

- Chronic Diseases – infectious and inflammatory;
- Chronic Dyserythropoietic Disorders – including some Thalassemias;
- Chronic Kidney Disease – especially with EPO/ESA therapy;
- Chronic Heart Disease – specifically Heart Failure;
- Malignant Disease

FID may be suspected to be present if the ferritin concentration is less than 100 µg/L, or if transferrin saturations (TSAT) of 20% or lower are present in the presence of a ferritin that is within the normal reference range, in the clinical context of such chronic diseases.

Anaemia of Chronic Disease

Anaemia of chronic disease (reticuloendothelial block) occurs when a persistent inflammatory state leads to the sequestration of iron, reducing iron availability for red cell production. This is mediated via the up-regulatory effects of inflammation on hepcidin.²

IRON OVERLOAD

Excess of iron can lead to a constellation of clinical manifestations known as haemochromatosis. The most common primary (hereditary) cause is *HFE*-related haemochromatosis. The most common mutation in the *HFE* gene is C282Y, which has a carrier rate of up to 5-12% in the Caucasian population. The second most common mutation is H63D and up to 24-31% of Caucasians are heterozygous for the allele. Generally only C282Y homozygotes (C282Y/C282Y) or C282Y compound heterozygotes (C282Y/H63D) are at risk of iron overload. H63D homozygotes (H63D/H63D) very rarely develop iron overload. Heterozygotes can occasionally develop clinical disease if there are other risk factors (e.g. excess alcohol consumption). Genetic testing can confirm the diagnosis. Other forms of hereditary haemochromatosis exist but are much rarer. Secondary iron overload can occur as a result of iron-loading anaemias (such as thalassaemia), parenteral iron excess (most commonly as a result of regular blood transfusions) and chronic liver disease^{5,6} (See figure 3).

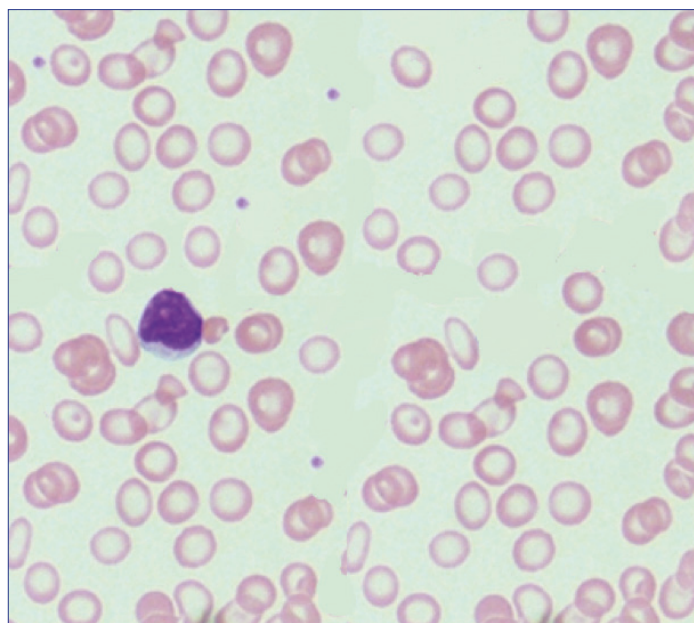
INVESTIGATIONS

- **FBC** - Low haemoglobin (Hb) occurs later in the course of iron deficiency and Hb can initially be in the normal range. MCV is usually low in iron deficiency. RDW- is the red cell distribution width and this is usually increased. The film may be reported as showing microchromic, microcytic red cells with elliptocytes (elongated red cells) and pencil cells (elongated cells with tapered ends). The FBC is usually normal in iron overload.

HH	HFE related HH (C282Y/C282Y, C282Y/H63D)
	Non-HFE related HH Juvenile Hemochromatosis Hemojuvelin related Hepcidin related Transferrin receptor-2 related HH
	Ferroportin related HH
Secondary Iron Overload	Iron loading anemia Thalassemia major Sideroblastic anemia Chronic haemolytic anemia
	Parenteral iron overload (multiple transfusions)
Others	Metabolic syndrome
	Chronic liver disease Hepatitis C Alcoholic liver disease Non-alcoholic steatohepatitis Porphyria cutanea tarda
	African Iron Overload
	Very low levels of ceruloplasmin
	Very low levels of transferrin
	Neonatal iron overload

Figure 3:

Izquierdo Alvarez et al. Journal of Genetic Syndromes and Gene Therapy 2013

**Figure 4:** Blood film showing iron deficiency anaemia, with hypochromic microcytic red cells

- **Serum Iron** - Serum iron is a measure of iron content in serum. It is affected by diurnal variation, recent iron intake and acute phase responses. It is therefore of limited utility in assessment of overall iron status, however is used in the calculation of transferrin saturation.
- **Ferritin** - In steady state ferritin levels are generally a good indicator of total body iron stores. Very low levels are diagnostic of iron deficiency. However a ferritin level within normal range but <100µg/L does not exclude

iron deficiency as ferritin is an acute phase reactant that can be increased in inflammatory states, liver disease and malignancy. Elevated levels may reflect iron overload, but again ferritin level alone can be misleading as it can be increased in all the above conditions. High transferrin saturation is required for the diagnosis of iron overload.

- **Transferrin** - Transferrin is the iron transport protein. Total Iron Binding Capacity (TIBC) is an indirect measure of transferrin levels. Transferrin levels are increased in the setting of iron deficiency, as well as in pregnancy and with oestrogen administration. It is decreased in states of inflammation, chronic liver disease and malnutrition.
- **Transferrin Saturation (Tsats)** - % of transferrin's carrying capacity that is saturated with iron. This is a calculated measurement (serum iron/TIBC x 100). Decreased saturation is suggestive of iron deficiency or inflammation. Transferrin saturation is useful in the assessment of high ferritin. A high ferritin with a Tsat below 45% in women or below 50% in men is more likely to be secondary to another cause. Testing should be repeated and if the Tsat remains elevated genetic testing should be performed to investigate for haemochromatosis (Ideally measured on fasting sample as it may be falsely elevated by recent iron ingestion).
- **Zinc Protoporphyrin (ZPP)** - ZPP is a trace by-product of haem synthesis and any condition that limits iron supply to the erythroid marrow or stimulates porphyrin synthesis leads to an increased concentration of ZPP in circulating red cells. The measurement of ZPP concentration provides a reliable index of FID and may be used as an alternative to indices of red cell hypochromia or reticulocyte haemoglobin content, although it is less sensitive to acute changes in iron availability.
- **Soluble transferrin receptor** - Transferrin receptor is up-regulated in states of increased iron demand, such as iron deficiency, or dysregulated erythropoiesis (e.g. haemolysis, thalassaemia, MDS, B12 or folate deficiency etc.) and is thus inversely related to body iron stores. Unlike ferritin it is not an acute phase reactant and therefore may be useful to demonstrate iron deficiency in the presence of an acute phase response and to distinguish iron deficiency from anaemia of chronic disease. **It is however not routinely available, and performed as a send away test with an out of pocket fee.**

Important points when interpreting iron studies

- As well as reflecting total iron stores, serum ferritin is affected by the acute phase response and other inflammatory conditions within the body, particularly those affecting the liver or reticuloendothelial system.

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	IRON DEFICIENCY (NAID)	IRON DEFICIENCY ANAEMIA	FUNCTIONAL IRON DEFICIENCY	IRON DEFICIENCY AND ACUTE PHASE RESPONSE/ACD	ACUTE PHASE RESPONSE/ ANAEMIA OF CHRONIC DISEASE (ACD)	TRUE IRON OVERLOAD
Hb	Normal	Reduced	Normal	Reduced	Reduced	Normal
MCV	Low or Low-normal	Low	Normal	Low or Low-normal	Low or Low-normal	Normal
Serum Iron	Decreased	Decreased	Decreased/N	Decreased	Decreased	Increased
Transferrin or TIBC	Increased	Increased	Variable	Variable	Decreased	Decreased
Transferrin Saturation	Decreased	Decreased	Usually < 30%	Normal/Decreased	Normal/Decreased	INCREASED
Ferritin	Decreased - < 30	Usually < 15	30-299	30-100, but within normal range.	> 200. Usually increased	INCREASED
CRP	Normal	Normal	Possibly increased	Increased	Increased	Normal
Soluble Transferrin Receptor	Increased	Increased	Increased	Variable	Normal	Decreased
ZPP	Normal	Increased	Increased	Increased	Normal	Normal
Marrow Iron	Reduced	Reduced	Low-Normal	Low-Normal	Increased	High-Normal

Figure 5: Patterns of iron study results¹³

- Low serum ferritin is diagnostic of iron deficiency.
- High serum ferritin can have many causes. Suspect iron overload if transferrin saturation is >50% in men or >45% in women. Genetic studies for HFE gene mutations are appropriate in this group.
- High serum ferritin without raised transferrin saturation is seen in inflammatory conditions, malignancy, liver disease, metabolic conditions, or rarely haematological conditions.⁷
- Serum iron is measured to calculate transferrin saturation, and is not useful alone to assess iron status.
- A ferritin <100µg/L but within normal range does not exclude iron deficiency in patients with inflammation/ acute phase response.

MANAGEMENT OF IRON DISORDERS

Iron Deficiency

Aim to correct ferritin to levels of more than 40 ug/L or higher in patients with functional deficiency.

- Oral iron (usually 100-200mg elemental iron per day for at least 3 months after normalisation of Hb)
- IV iron for selected patients
- Optimise dietary iron (secondary prevention) and address underlying cause

Note: In patients with true polycythaemia inducing an iron depleted state is a goal of their management, and unless anaemic due to over-venesecting or other chronic states, iron replacement is not advised as this will only worsen the polycythaemia.

True Iron Overload

- Reduce dietary iron if appropriate
- Venesection regimen in the non-anaemic patients
- Chelation therapy in anaemic patients - i.e. venesection is contraindicated
- In all Iron overload patients it is prudent to provide liver protection and to ensure the patient is immunised against Hepatitis A and Hepatitis B

FURTHER INFORMATION

QML Pathology Procedural Unit offers Iron Infusion and a Venesection service in selected areas. Please contact your Medical Liaison Officer for more information.

REFERENCES

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Changes to Cervical Screening Collection Requirements

From the 15th May 2019 QML Pathology will no longer be accepting SurePath™ samples for Cervical Screening tests.

ThinPrep® is the only collection medium that will be accepted.

For further details about this change please contact your Medical Liaison Officer.

Please Note: Due to a recent large scale mail out of CST invitations by the National Cervical Screening Register, our turnaround time for CST's has extended, particularly for those requiring either reflex cytology or a co-test cytology.

We apologise for this delay and thank you for your understanding.

Pharmacogenomic (PGx) Testing

QML Pathology now provide Pharmacogenomic (PGx) Testing.

PGx is the study of genetic variations that influence response to medication; enabling you to test for specific genetic changes to predict whether a patient may have a normal response, a poor response, or a higher risk of side effects before prescribing a specific medication. This testing is recommended in the areas of Mental Health and chronic pain conditions, in some cases it can also assist in cardiac and gastrointestinal conditions.

For detailed information on testing options and cost, contact your QML Pathology Medical Liaison Officer.

From the Education Desk

We are well into the final year of the Triennium and with that in mind, please review your list of activities over the last 3 years with your Colleges to ensure you have all your necessary points including your mandatory Cat 1 QI activity. The hugely popular Cat 1 Skin ALM is scheduled for 27th July 2019. Due to the end of the triennium we have decided to only have one event for 2019, so please let your Medical Liaison Officer know of your interest and we will send you your personalised invitation. Please note, there are only 40 attendees for this event so register quickly so you don't miss out.

THE DYSGLYCAEMIC AND DIABETES MELLITUS AUDIT (Now Closed for Registrations)

This audit has seen a change for 2019 based on responses in evaluations and reflections in the first 18 months. The audit extended from 9 months to 12 months to better reflect the annual Diabetes Mellitus cycle of care. We would like to thank all doctors for their input, kind words and support in regards to this audit. Due to this change we are NO longer available to offer registration for this audit in this current triennium.

THE SURGICAL SKIN AUDIT (Open for Registrations)

You will soon notice a change to the Skin Audit report format; the revised report will better reflect changes in current practice and give you greater statistical information based on that month's participation. Whilst we have implemented these changes we are happy to let all know that we have been able to maintain the uncomplicated and easy to read report format.

Reminder: The Surgical Skin Audit requires specialised request forms. To order, please use your stores request forms via your local laboratory or via the website. Please use these requests with the reverse of the request form completed to ensure your specimen is included in your count.

For further information on all of our upcoming educational activities please visit our website www.qml.com.au, contact your local Medical Liaison Officer or email Education directly at education@qml.com.au.

Warm regards and hope to see you all soon at our events.
The QML Pathology Education Team,
Jo and Sharon

Doctor Noticeboard

Dr Georgie Lyons MBBS (Hons), BMedSci (Hons), MPH & Trop Med (Hons), FACD

Dr Lyons' special interests include Medical Dermatology, Women's and Paediatric Dermatology. Dr Lyons is a general dermatologist with an interest in the treatment of acne, psoriasis, eczema, connective tissue disorders, bullous dermatoses, vulval diseases, paediatric skin conditions and cutaneous malignancies. She has previously worked as a Consultant Dermatologist at The Royal Children's Hospital and The Western Hospital in Victoria. Dr Lyons is excited to have relocated to Toowoomba and is now available for referrals.

P: (07) 4638 1566 / F: (07) 4639 2446

Prof. David Coman MBBS MPhil FRACP

Professor David Coman is pleased to announce a change of location for his general paediatric, metabolic and clinical genetics private practice. Formerly at the Wesley Hospital, he is now at 55 Little Edward Street, Spring Hill 4000. Whilst still delivering babies at the Wesley and performing public clinics at the Queensland Children's Hospital, his new Spring Hill rooms allow more space for Star Wars figurines and support a close collaboration with Queensland Fertility Group.

P: (07) 3832 9876/ E: reception@drdavidcoman.com.au
W: www.drdavidcoman.com.au

Dr Maurice Stevens M.B.B.S. (Qld) F.R.A.C.S.

Otolaryngology Head & Neck Surgery

Dr Stevens is a highly experienced ENT surgeon who for over 20 years has been practising in The Gap area. Dr Stevens will be attending Harry St Monday afternoons and would be happy to see patients with general ENT problems, including children and he has a special interest in nasal surgery, be it reconstructive or pertaining to the sinuses. Dr Stevens continues to practice in Wickham Terrace, Belmont and Longreach.

P: (07) 3832 5422.

Dr Lauren Galt BBioMedSc, MBBS, FRACP

Dr Lauren Galt is a Thoracic and Sleep Physician now working collaboratively with Dr Alex Ritchie and Dr Ian Brown at the St. Andrew's War Memorial Hospital and the Wesley Hospital. Lauren is passionate about personalised patient care, and is adept at managing severe asthma, COPD, bronchiectasis, lung cancer, pleural disease, obstructive and central sleep apnoea, as well as complex ventilation in the setting of neuromuscular disease. She performs standard and complex bronchoscopy, including radial and convex EBUS, and cryobiopsy.

P: (07) 3832 7776 / E: lauren@galtrespiratory.com.au

Dr Kevin Chan B.Pharm MBBS FRACS

Dr Chan is a General Surgeon with a special interest in Upper GI surgery. He undertook subspecialty training in advanced Upper Gastrointestinal and Bariatric surgery through the Australian and New Zealand Gastric and Oesophageal Surgery Association (ANZGOSA) post-fellowship training program. He currently holds a VMO post at the Royal Brisbane & Women's Hospital and consults and operates privately at St Vincent's Private Hospital Northside, St Andrew's War Memorial Hospital and The Wesley Hospital. Appointments for patients can be made by contacting his rooms.

P: (07) 3350 2533 / F: (07) 3350 2511

E: info@totaluppergisurgery.com.au

Dr Gerald Tracey LRCP&SI MB, Bch NUI, MRCPI, MRCP (Rheum)

Dr Tracey has completed Specialist Rheumatology training at the Royal National Hospital for Rheumatic diseases in Bath, UK. He has special interest in Inflammatory conditions, Osteoporosis, Fibromyalgia and Giant Cell Arteritis. He is also trained in Musculoskeletal Ultrasound and has publications related to PMR, Rheumatoid Arthritis, Osteoporosis, Connective Tissue Disease and Ankylosing Spondylitis. Dr Tracey is interested in a holistic approach, creating a collaborative relationship with the patient including a strong emphasis on self-management and non-pharmacological strategies when appropriate. Dr Gerald Tracey has joined Paradise Arthritis & Rheumatology located in Nerang St, Southport.

P: (07) 5591 5542 / E: reception@panr.com.au

Dr Brenda Biggs BSc (Hons), MSc, MBBS, FRANZCOG

Dr Biggs has not retired from being a VMO Obstetrician at Mater Mothers' Private Hospital. Her rooms are located at Level 3, Mater Private Clinic and she shares on-call with the River City Obstetric Group. Dr Biggs is happy to receive referrals for antenatal care and management of deliveries (including high risk pregnancies) and office Mirena insertions.

P: (07) 3010 3344 / F: (07) 3010 3338

Infectious Diseases Report

GEOGRAPHIC DISTRIBUTION - MAR 2019

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)	21	76	25	16	0	0	57	0	45	14	101	65	12	23	22	183	115	179
Adenovirus (typing pending)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Barmah Forest virus	1	0	0	0	0	0	0	0	2	1	1	0	0	1	1	3	2	2
Bordetella pertussis	4	50	21	4	1	0	32	0	53	11	89	43	12	5	19	100	104	140
Brucella species	0	0	1	0	0	0	0	0	0	0	0	2	4	2	0	2	6	1
Campylobacter jejuni	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chlamydia pneumoniae	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
Chlamydia trachomatis, not typed	147	356	177	77	10	2	449	5	227	106	786	252	80	135	71	1024	921	935
Coxiella burnetii	1	3	9	4	0	1	1	0	2	3	1	9	10	5	3	26	10	16
Cryptococcus species	1	2	1	0	0	0	1	0	0	0	5	2	0	0	0	7	3	2
Cytomegalovirus (CMV)	5	16	6	4	0	0	27	0	25	7	32	13	3	4	4	46	58	42
Entamoeba histolytica	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Enterovirus - not typed	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Epstein-Barr virus (EBV)	7	57	35	11	0	0	80	0	58	10	134	64	11	11	14	192	148	152
Flavivirus unspecified	8	7	0	2	0	0	8	0	4	0	9	7	1	3	2	17	19	15
Hepatitis A virus	0	5	1	0	0	0	3	0	2	0	6	0	0	1	0	7	7	4
Hepatitis B virus	25	35	22	3	2	0	59	2	12	8	244	16	9	4	7	170	155	123
Hepatitis C virus	38	133	86	19	5	0	128	0	93	28	335	85	48	31	48	388	357	332
Hepatitis D virus	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1	0
Hepatitis E virus	0	0	0	0	0	0	2	0	1	0	1	0	0	0	0	3	1	0
Herpes simplex Type 1	58	158	91	37	9	0	186	0	132	35	383	165	36	59	42	479	422	490
Herpes simplex Type 2	34	150	43	11	1	0	113	0	66	13	169	88	12	20	12	251	259	222
Herpes simplex virus - not typed	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
HIV-1	2	6	2	1	0	0	12	0	3	0	14	2	0	2	0	15	15	14
HTLV-1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Human Metapneumovirus	13	32	6	2	0	0	27	0	29	6	41	43	5	13	7	57	74	93
Influenza A virus	184	349	102	62	12	1	350	7	230	41	471	242	43	182	34	850	717	743
Influenza B virus	6	25	3	0	5	0	23	0	15	2	34	15	6	6	3	54	39	50
Legionella pneumophila (all serogroups)	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1	0
Legionella species	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0
Leptospira species	1	0	0	1	0	1	0	0	0	1	0	0	1	0	0	2	2	1
Measles virus	0	0	0	0	0	0	2	0	0	0	1	0	0	0	0	1	2	0
Mumps virus	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0
Mycoplasma pneumoniae	2	40	10	5	0	2	28	0	24	6	60	19	5	6	7	73	62	79
Neisseria gonorrhoeae	33	40	20	1	4	0	69	1	31	4	124	22	9	5	10	107	112	154
Parainfluenza virus	12	88	41	10	2	0	84	0	64	30	129	71	8	19	16	266	156	152
Parvovirus	1	9	2	0	0	0	7	0	3	0	17	8	2	0	1	15	12	23
Pneumocystis carinii	0	0	0	0	0	0	2	0	0	0	0	1	0	0	0	1	1	1
Respiratory Syncytial virus	35	163	26	36	1	0	119	0	94	38	195	139	12	81	43	520	265	197
Rhinovirus (all types)	50	231	103	40	4	2	194	1	236	93	348	179	64	104	52	753	553	395
Rickettsia - Spotted Fever Group	0	4	1	1	0	0	1	0	0	1	0	0	2	1	0	5	1	5
Ross River virus	21	22	5	14	3	0	15	0	15	24	22	24	7	31	22	103	85	37
Rubella virus	0	1	1	0	0	0	0	0	0	0	1	0	0	0	0	1	1	1
Salmonella paratyphi A	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Salmonella paratyphi B	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Salmonella typhi	0	0	0	0	0	0	2	0	0	0	1	0	0	0	0	0	1	2
Streptococcus Group A	14	30	32	5	2	3	45	214	21	6	57	28	9	15	14	187	163	145
Toxoplasma gondii	6	17	5	1	0	1	18	0	9	3	26	9	6	3	7	25	39	47
Treponema pallidum	127	47	42	7	41	2	276	5	51	41	267	36	25	75	20	379	317	366
Trichomonas vaginalis	37	6	5	2	8	0	5	4	3	5	19	5	2	17	5	47	35	41
Varicella Zoster virus	48	147	75	16	2	0	187	0	117	29	295	137	15	31	36	410	370	355
Yersinia enterocolitica	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
TOTAL	942	2308	999	392	112	15	2612	240	1667	566	4419	1791	460	895	522	6772	5612	5556

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.