

QML PATHOLOGY

newsletter February 07

>> Microbiological Review of *Trichomonas vaginalis*

Dr David Drummond, Pathologist, Infectious Diseases & Immunology

Trichomonas vaginalis is the most prevalent non-viral sexually transmitted infection globally, yet remains the 'poor cousin' of the family of sexually transmitted infections. It has not received the same degree of interest as compared to its stable mates such as *Chlamydia trachomatis* or *Neisseria gonorrhoea*. Never the less it is an important pathogen and one that deserves respect.

Whilst the protozoan was recognized for many years, it was not until 1939 that the complete pathological picture was detailed following human volunteer studies. In more recent times consideration has been given to attempting to develop a vaccine. However, we currently understand very little of the fundamental immunological processes relating to the infection and so progress is limited.

The genome of *Trichomonas vaginalis* is currently being sequenced.

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Epidemiology

The World Health Organization estimates that there is between 170 million to 190 million cases worldwide each year. In the Australasian region the estimate is of approximately 610,000 cases per annum. These estimates may be low as they are based on an assumed sensitivity of wet mount microscopy of 60-80%. In reality the sensitivity is probably closer to 35-60% in an ideal setting.

More precise data for the Australian population is limited as the infection is only required to be reported in the Northern Territory. What data there is indicates that the infection is more common in lower socio-economic groups and the indigenous population. A number of studies have demonstrated that the prevalence of this infection in Aboriginal and Torres Strait Islander women is up to 30 times higher than in non Aboriginal and Torres Strait Islander people.

Life Cycle and Pathogenesis

There is no cyst stage in the life cycle of this fragile, motile protozoan, reflecting its adaptation to the genital tract without an intermediary host. Sexual transmission is followed by expansion of the protozoan population via binary fission as part of the infective process (see life cycle illustration).

The trophozoite possesses specific adhesions for squamous epithelial cells. In women the infection can involve the exocervix as well as the vagina. Once attached to an epithelial cell, *Trichomonas* releases a range of cytolytic chemicals killing the affixed cell and liberating nutrients for the trophozoite to ingest. Following destruction of the cell the trophozoite detaches from the cell surface and moves to the next target cell.

The process not only leads to individual cell death but also disrupts the overall integrity of the tissue resulting in sloughing of the epithelium. The end metabolic products of *Trichomonas* may also enhance tissue necrosis. Molecular hydrogen is produced, which aside from producing the frothy appearance of the discharge may also act in a direct cytotoxic manner. Another metabolic product – putrescine – gives rise to the offensive odour of the discharge. Such destruction induces an intense inflammatory response, giving rise to the characteristic symptoms and signs.

Infection

Trichomonas vaginalis infection is associated with:

- Female morbidity
- Male morbidity
- Enhancement of HIV transmission
- Pregnancy complications
- Neonatal infection (rarely).

Only about 20% of females are asymptomatic, although this figure may be at the lower end as a result of sampling bias.

The remainder suffer a wide range of symptoms ranging from mild discomfort and dyspareunia, to incapacitating illness. Dysuria may also accompany the infection.

Infection in males is mainly asymptomatic and involves the prostate and related urinary - reproductive tract. In symptomatic males, prostatitis or urethritis are the principal findings. It is unknown why males should be spared the consequences of infection in comparison to females.

In populations where heterosexually transmitted HIV infection is an issue, the presence of *Trichomonas vaginalis* infection and the resulting denuded raw epithelium assists the transmission of HIV. The HIV viral load in seminal fluid and cervico – vaginal compartments of *Trichomonas* infected individuals is also significantly increased. This is important as the HIV viral load is the largest risk factor for HIV transmission in discordant HIV status couples. Consequently control and eradication of *Trichomonas vaginalis* is a key element in controlling HIV infection in at-risk communities.

Some recent studies have demonstrated an association with pregnancy complications including premature rupture of membranes, pre-term delivery and low birth weight. Rarely, neonates delivered through an infected birth canal can develop a respiratory tract infection. Urinary tract infection in female neonates has been described.

Diagnosis

Traditionally the diagnosis of *Trichomonas vaginalis* infection has been based principally on the clinical history and examination.

Vaginal discharge is present in 50 to 75% of diagnosed women, but the classical description of a coloured frothy blood- stained offensive discharge is not universal. In only about 10% of cases is the discharge described as offensive, and in about 8% of cases is the discharge frothy. In the majority of cases, the discharge can be described as being purulent – a description that can be applied to vaginal discharge arising from many other causes.

The classic signs reflect the infective process and resultant impact on the vaginal and exocervical tissue. The thick copious discharge is highlighted against a background of vulval and vaginal erythema. The cervix may have the colpitis macularis ('strawberry cervicitis') appearance.

Classically, confirmation of the diagnosis of *Trichomonas* infection was a 'side room' test, whereby a microscopic examination of a freshly obtained specimen of vaginal discharge revealed the 'bustling' motility of the trophozoite. The fragile nature of these trophozoites means that any

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additional delay results in their death and dissolution. This combined with the absence of a durable cyst stage greatly hinders the ability of delayed microscopic examinations to identify the pathogen.

In attempting to overcome this limitation a number of techniques were developed either to improve the viability of the trophozoite, the detection of specific antigens or antibodies, or the sensitivity of microscopy. Unfortunately many of these techniques resulted in little overall improvement in detecting infection. This was clearly an infection whose diagnosis could be improved by the application of the high sensitivity and viability - independent techniques associated with molecular technology. Nucleic acid amplification techniques (NAAT) have now been developed for the detection of *Trichomonas vaginalis* in vaginal discharge specimens.

QML pathology now does not perform 'wet preparation' microscopy of vaginal or endocervical swabs, but will undertake a *Trichomonas vaginalis* PCR on all requests indicating that *T. vaginalis* infection is suspected. For details regarding the investigational requirements please see the section at the end of the article.

Therapy

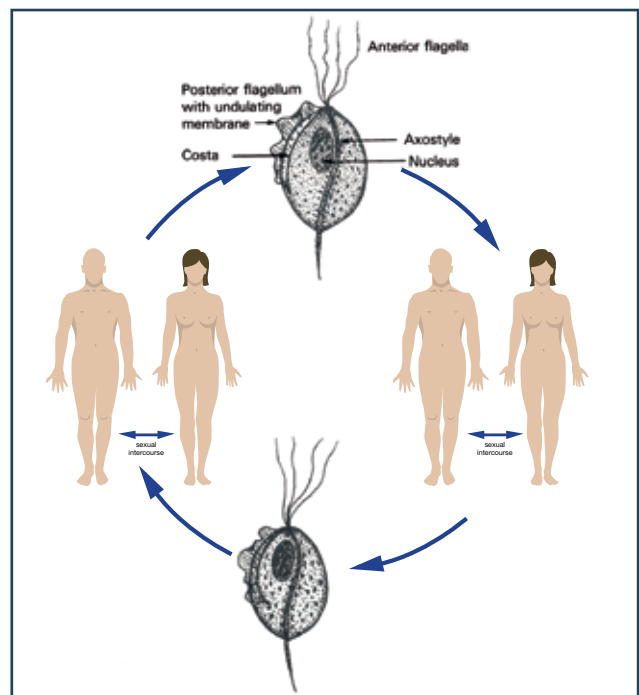
The drug of choice is metronidazole, given orally as a 2g single dose. Intra vaginal suppository formulations containing metronidazole have not proven as effective in clinical trials compared to oral delivery. Tinadazole 2g orally as a single dose is equivalent to metronidazole.

In pregnancy, the benefits of treatment are viewed as more important than the risks but not without caution. *Trichomonas vaginalis* infection has been associated with adverse pregnancy outcomes such as premature rupture of membranes, pre-term delivery and low birth weight. Some trials, however, have associated increased prematurity with treatment in the first trimester. The exact mechanism of action of metronidazole in regard to *Trichomonas vaginalis* is still not understood. The end result, however, is that the growth of the trophozoite is inhibited.

Resistant strains (approximately 2 – 5%) have a sequence of metabolic deficiencies that preclude any action of metronidazole. The alternate therapy for apparent resistance is a prolonged course of metronidazole 400mg b.d orally for 5 days.

Male sexual partners obviously require treatment concomitant with their sexual partner. Most apparent treatment failures are the result of re-infection by an untreated partner, rather than overt antimicrobial resistance.

In conclusion, *Trichomonas vaginalis* is a significant sexually transmitted infection. Aside from the associated morbidity in infected females, there is increasing concern over its association with complications of pregnancy and HIV transmission. It is only now with the advent of, and routine application of molecular diagnostic techniques that we will begin to obtain a clearer understanding of the extent of this infection in our communities, and the appropriate measures to ensure control of this important infection.



Life Cycle Illustration



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References:

1. Despommier D D, et al eds. *Trichomonas vaginalis*. In *Parasitic Diseases 5th edition*. Appletree productions L.L.C. NY. 2006.
2. Burgess D E. *Trichomonas infections*. In *Topley and Wilson's Microbial Infections 10th edition Parasitology*. Hodder Arnold 2006.
3. *Therapeutic Guidelines Antibiotic version 13*. Therapeutic Guidelines Melbourne. 2006.
4. *National Sexually Transmissible Infections (STIs) Strategy 2005 – 2008* Canberra

>> NAAT Testing for *Trichomonas vaginalis*

Dr David Drummond, Pathologist, Infectious Diseases & Immunology

QML Pathology will now perform testing for *Trichomonas vaginalis* by nucleic acid amplification technology (NAAT), specifically Transcription Mediated Amplification (TMA) methodology. We are adopting this approach for the following reasons:

- 1: The significantly increased sensitivity of the test compared to wet preparation examination – see table 1
- 2: The test is independent of viability of the *Trichomonas vaginalis* and therefore, there is no special transport or storage requirements
- 3: Testing can be performed in conjunction with testing for *Chlamydia trachomatis* and *Neisseria gonorrhoea* using the same specimen
- 4: Increasing concern regarding the morbidity associated with infection.

Useful For:

Detection of *Trichomonas vaginalis* infection of the female and male genitourinary tract.

Interpretation

- A negative result usually excludes infection with *Trichomonas vaginalis*.
- An indeterminate result indicates that there may be unidentifiable chemical compounds in the specimen that are inhibiting the assay.
- A positive result indicates the presence of RNA from *Trichomonas vaginalis* in the submitted specimen. Please note that specific nucleic acid residues for *Trichomonas vaginalis* could be detected for up to five weeks following successful therapy.

Cautions

- The test has only been validated for vaginal or urine specimens collected in the appropriate manner using the specific collection technique(s).
- Vaginal specimens from women should not be collected during menstruation, or if topical creams or douches have been used in the preceding 24 hours.
- The assay has not been evaluated for seminal specimens or respiratory samples from neonates.

References

1. Hobbs, M. et al. *Evaluation of Real-Time PCR and Transcription Mediated Amplification for Detection of Trichomonas vaginalis in Urine*. University North Carolina. Poster C-095. American Society for Microbiology Annual Scientific Meeting 2006.
2. Sitay, A. et al. *Rapid Detection of Trichomonas vaginalis from Vaginal specimens by Transcription Mediated Amplification*. Gen-Probe Inc, San Diego. Poster C-120 American Society for Microbiology Annual Scientific Meeting 2003.

Method

Target capture, Transcription-Mediated Amplification (TMA) and Hybridization Protection Assay (HPA).

Target capture uses specific DNA capture oligonucleotides and magnetic beads for separation of target ribosomal Ribo Nucleic Acid (rRNA) from clinical specimens. TMA amplifies a specific region of the target rRNA. HPA uses a chemiluminescent probe in a homogenous assay format whereby probe binds specifically to *Trichomonas vaginalis* amplicon and is induced to emit light.

Specimens

- First pass urines collected into plain urine containers or Aptima urine collection tubes.
- Aptima swab from endocervix / high vagina.
- Aptima swab from urethra.

Further details about the Aptima collection system can be found in the Aptima collection brochure.

Assay Frequency

Assays are performed daily, Monday to Friday. Specimens received by the morning will be processed the same day and results will generally be available the following morning.

Table 1: Comparison of PCR detection compared to current detection methods

Technique	Source	Sensitivity (%)	Specificity (%)
Pap smear		24	99
Wet prep	Discharge	40-60	-
PCR (female)	Urine	90-97	90-100

Rh (D) Antenatal Prophylaxis Program and Red Cell Antibody Screening

Current Australian Guidelines recommend that all Rh (D) negative women should have red cell antibody screening performed at their initial antenatal visit and at least once between 28 and 36 weeks gestation.

With routine antenatal prophylaxis, the blood sample for antibody screening should be collected prior to giving the 28 week dose of Rh (D) immunoglobulin.

Two recent Australian cases of significant adverse foetal outcomes highlight the importance of ensuring the correct timing and interpretation of red cell antibody screening in the Rh (D) antenatal prophylaxis program.

These two cases have reportedly been attributed to a lack of clarity as to whether the presence of Rh (D) antibodies was due to active alloimmunisation or prophylaxis. In both cases, the Rh (D) immunoglobulin was given prior to blood sample collection thus further complicating laboratory findings.

In 2005, two cases were reported in which misinterpretation of the antenatal antibody investigation resulted in severe haemolytic disease of the newborn. The report recommended that implementation of routine Rh (D) antenatal prophylaxis must be supported by education.

As a consequence of the reported cases, the Australian and New Zealand Society of Blood Transfusion (ANZSBT) in conjunction with a Joint Rh (D) Consultative Committee (JCC) are performing a review of procedures. Until the outcomes of this review are completed, it is recommended that:

- When antenatal red cell antibody testing is indicated e.g. at 28 weeks gestation, the blood sample for the test should be collected prior to the administration of Rh (D) immunoglobulin
- If Rh (D) immunoglobulin has already been administered, the test should still be performed and the prior administration of Rh (D) immunoglobulin should be highlighted on the laboratory request form
- When a laboratory detects an Rh (D) antibody with a strong agglutination, even after the administration of Rh (D) immunoglobulin, quantification of the Rh (D) antibody should be performed.

Further information regarding Rh (D) immunoglobulin, the Rh (D) antenatal prophylaxis program and antenatal red cell antibody screening can be found at the following websites:

- RANZCOG website at www.ranzcog.edu.au/publications/statements/C-obs3.pdf
- ARCBS website at www.transfusion.com.au/RhD
- ANZSBT website at www.anzsb.org.au/publications/documents/ANGuidelines2004.pdf.

References

- 1) Australian Red Cross Blood Service, *Red Cell Antibody Screening in the Context of the Rh (D) Antenatal Prophylaxis Program*, January 2007

Testing and Condition	Timing
ABO and Rh (test for weak D optional)	
<ul style="list-style-type: none"> • All pregnancies • Other 	<ul style="list-style-type: none"> • Initial visit • For pretransfusion testing
Antibody screening	
<ul style="list-style-type: none"> • All pregnancies • RhD Neg pregnancies 	<ul style="list-style-type: none"> • Initial visit • Additionally at least once between 28 and 36 weeks. In the context of routine antenatal prophylaxis this should be undertaken prior to administration of first dose of RhD immunoglobulin at 28 weeks
<ul style="list-style-type: none"> • Other 	<ul style="list-style-type: none"> • For pretransfusion testing
Antibody identification	
<ul style="list-style-type: none"> • Unexpected antibodies present • Confirmatory testing 	<ul style="list-style-type: none"> • Upon initial detection • At time of titration
Antibody titration/quantitation	
<ul style="list-style-type: none"> • Rh antibodies 	<ul style="list-style-type: none"> • Upon initial detection. Repeat at 18-20 weeks gestation. Further testing will be influenced by titre/quantitation and specificity of antibody
<ul style="list-style-type: none"> • Other potentially significant antibodies 	<ul style="list-style-type: none"> • As above, with discussion with Obstetrician

Table 1: Recommended Prenatal Testing

Anti-D Administration

RhD Negative females only (with negative antibody screen)
Routinely at 29/40-33/40 (625 IU) AFTER antibody screening is performed at 28 weeks Postpartum 625IU.

ANC 1st Visit Group & Screen	18-20	28-30	30-34	34-36	Post-partum
Rh Pos/ No Ab		Optional Screen			
Rh Pos/ Pos Ab*	Antibody Titration				
Rh Neg/ No Ab		Antibody Screen	625 IU Anti-D		625 IU Anti-D + extra if Kleihauer dictates
Rh Neg/ Pos Ab#	Antibody Titration	Antibody Titration	625 IU only if NO allo anti-D		625 IU only if NO allo anti-D

* Antibodies detected will NOT include anti-D, but other antigen systems may be detected e.g. anti-K, anti-c

anti-D may be present or perhaps other antigen systems may be detected if anti-D is present it is important to be sure that it is true alloimmunisation and not passive due to blood testing within 6-8 weeks of Anti-D administration

clinical data feb 07

Infectious Diseases Report - Geographic Distribution - December 2006

SEROLOGY	Regions (as per key below)															Total Dec	Nov	Oct	Sep
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15				
Adenovirus (not typed)	1	1	0	0	0	0	1	0	4	0	1	1	0	2	0	11	14	23	34
Barmah Forest virus	2	2	0	2	0	0	2	0	1	1	4	6	0	1	3	24	44	91	49
Bordetella pertussis	2	3	2	0	0	0	10	0	7	0	9	7	0	0	1	41	72	79	67
Brucella species	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0
Campylobacter jejuni	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chlamydia pneumoniae	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chlamydia trachomatis, not typed	51	52	17	11	1	0	50	1	21	18	87	44	6	13	11	383	502	516	434
Coxiella burnetii	2	0	3	0	0	0	0	0	1	0	0	1	1	0	0	8	11	8	15
Cryptococcus species	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	3	1	1
Cytomegalovirus (CMV)	0	7	2	1	0	0	4	0	2	1	5	1	1	2	3	29	33	17	20
Entamoeba histolytica	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1
Enterovirus - not typed	0	0	0	0	0	0	2	0	0	0	0	0	0	0	1	3	1	5	9
Epstein-Barr virus (EBV)	6	9	6	6	0	0	24	0	13	3	18	15	4	4	2	110	149	169	123
Flavivirus unspecified	5	1	0	1	0	0	1	0	0	0	1	1	1	3	0	14	14	10	4
Hepatitis A virus	1	0	0	0	0	0	1	0	0	0	1	0	0	0	0	3	4	0	6
Hepatitis B virus	5	1	3	1	0	0	10	0	0	0	35	3	1	2	1	62	83	84	74
Hepatitis C virus	16	42	13	3	1	0	19	0	17	7	39	11	3	7	3	181	289	263	266
Hepatitis D virus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Hepatitis E virus	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0
Herpes simplex Type 1	10	26	8	0	0	2	34	0	22	8	49	13	4	7	7	190	208	233	188
Herpes simplex Type 2	15	22	4	0	0	0	18	0	15	6	35	12	3	4	1	135	207	174	156
Herpes simplex virus - not typed	4	8	0	3	0	0	4	0	2	1	8	3	0	5	1	39	63	58	34
HIV-1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	7	2	4
HTLV-1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Influenza A virus	0	1	0	0	0	0	2	0	2	0	0	2	0	0	0	7	11	16	96
Influenza B virus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	3	11
Legionella species	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Leptospira species	1	0	0	0	0	0	2	0	0	0	0	0	0	1	0	4	2	4	3
Measles virus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
Mumps virus	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1	0	2	0
Mycoplasma pneumoniae	3	12	3	0	0	0	17	2	11	6	13	5	1	0	4	77	104	116	195
Neisseria gonorrhoeae	2	1	1	1	0	0	9	0	0	0	6	3	0	2	0	25	36	27	28
Parainfluenza virus Type 1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Parainfluenza virus Type 2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1
Parainfluenza virus Type 3	0	2	0	0	0	0	2	0	1	0	3	0	0	1	0	9	14	27	18
Parvovirus	1	1	0	1	0	0	5	0	7	3	8	2	3	2	1	34	56	83	69
Pneumocystis carinii	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	3	0	0	4
Respiratory Syncytial virus	0	2	2	0	0	0	5	1	5	2	7	0	1	4	0	29	25	41	77
Ross River virus	4	1	1	0	0	0	2	2	0	2	4	3	0	1	0	20	23	22	26
Rubella virus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Salmonella paratyphi A	0	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	0
Salmonella typhi	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	2	0	0
Shigella dysenteriae	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Shigella flexneri	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Streptococcus Group A	10	7	1	1	1	2	10	0	12	5	10	15	3	1	4	82	88	114	104
Toxoplasma gondii	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	10	2	3	2
Treponema pallidum	14	7	3	1	3	0	17	0	3	1	13	4	1	8	0	75	122	105	107
Trichomonas vaginalis	2	0	0	0	0	0	3	0	1	0	0	0	0	1	0	7	9	3	6
Varicella Zoster virus	6	17	9	0	0	2	33	1	19	14	51	22	8	4	0	186	210	197	211
Yersinia enterocolitica	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
TOTAL	163	229	79	32	6	6	288	8	166	78	408	175	41	75	43	1797	2413	2499	2454

REGIONS

1 Cairns
2 Gold Coast/Northern Rivers
3 Ipswich

4 Mackay
5 Mount Isa
6 New England
7 North Brisbane Suburbs

8 Northern Territory
9 Redcliffe
10 Rockhampton
11 South Brisbane Suburbs

12 Sunshine Coast
13 Toowoomba
14 Townsville
15 Wide Bay/Burnett

November 2006 and further historical clinical data can be obtained by contacting your local Medical Liaison Officer

This newsletter has been prepared and published by QML Pathology for the information of referring doctors. Although every effort has been made to ensure that the newsletter is free from error or omission, readers are advised that the newsletter is not a substitute for detailed professional advice.

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Queensland Cervical Screening Program Media Campaign

Queensland Health recently launched its Queensland Cervical Screening Program media campaign. The goal of the campaign is to increase participation of Queensland women in the program by improving awareness amongst women of the importance of regular Pap smears in reducing the incidence of cervical cancer. In particular the campaign will focus on those women who rarely have or who have never had a Pap smear.

By achieving high participation rates and standards across the cervical screening pathway the detection of cervical cancer precursors can be maximised. The program aims to promote access to information about cervical screening and follow-up services, and to promote quality management and high standards of service delivery throughout the cervical screening pathway. The program is continuously monitored and evaluated at all levels.

QML Pathology has an ongoing commitment to supporting the Queensland Cervical Screening Program and its goals by providing a high quality gynaecological cytology service staffed by an experienced team of cytopathologists and scientific staff. Our laboratory meets all national performance measures and adheres to all quality assurance practices required by NPAAC, including participation in the Royal College of Pathologists external Quality Assurance Program in Cytopathology.

For further information please contact our Cytology Department on (07) 3121 4009 or the Queensland Cervical Screening Program on (07) 3234 1596.

Doctor's Noticeboard

- Dr John Gibbons and Dr Kate Cayzer, Consultant Gastroenterologists, are pleased to announce the commencement of their new practice as of 1st December 2006. In addition to visiting Mater Private Hospital and Eastern Endoscopy Centre, they have expanded to take in Sunnybank Private Hospital. Consultations are available at Springwood, Sunnybank, Camp Hill, Stones Corner and Birkdale.

Dr Georgia Hume, Consultant Gastroenterologist, will continue her sessions at Mater Private Hospital and Eastern Endoscopy Centre. For more information please phone (07) 3207 1111 or visit our website www.gibbons-cayzer.com.

- Relocation
Chinatown Surgery - Doctor Dentist has relocated to:
Shop 206 McWhirters Building
Cnr of Brunswick and Wickham St,
Fortitude Valley QLD 4006
Telephone remains the same - (07) 3252 3288
Fax remains the same - (07) 3252 8383

The surgery will be renamed:
Chinatown - McWhirters Medical Centre

- Dr John Morris offers sessions suitable for a branch consulting practice (e.g. psychiatry) at Indooroopilly.
 - Easy access and parking
 - Reception
 - Consulting room.Phone Sue Brown on (07) 3870 2244.

New Collection Centres

Sinnamon Park

58 Oldfield Road, Sinnamon Park
(located inside Sinnamon Park Medical Centre)
Phone: (07) 3376 0262
Fax: (07) 3715 6360
Opening Hours:
Monday to Friday 8.00am - 4.00pm
(closed for lunch 12.30pm - 1.00pm)

Relocated Collection Centres

Beenleigh

Unit 2, 70-72 City Road
Phone: (07) 3287 5947
Opening Hours:
Monday to Friday 7.00am - 6.00pm
Saturday 8.00am - 12.00pm