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In this issue

Methicillin-resistant Staphylococcus aureus (MRSA) is a major problem worldwide, including in New Zealand. Zena Fadheel and colleagues found a MRSA prevalence in health workers from a hospital setting of 4% compared to zero in a general population.

Anna Denholm and colleagues present a case study of a pregnant woman with a family history of abnormal bleeding who initially presented with a raised PFA-100, normal FVIII:C and vWF:Ag, but a slight reduction in vWF:RCo and vWF:CB indicating a possible functional vWF discordance. A possible diagnosis of Type 2 vWF was considered, but in-depth laboratory testing confirmed this to be a case of pseudo von Willebrand disease.

It has been slightly over 10 years that the first graduates of the Bachelor of Medical Laboratory Science degree programmes entered the workforce. In this issue Caroline Knight shares her experience of studying medical laboratory science at the Auckland University of Technology.

In this issue the Editor discusses authorship (and non-authorship) criteria for papers submitted to the Journal. A new criteria for submitting, namely for authors to state what their contributions to the paper were, will apply from now together with one author taking responsibility for the integrity of the work as a whole.

Med-Bio Journal Award

Med-Bio offers an award for the best article in each issue of the New Zealand Journal of Medical Laboratory Science. All financial members of the NZIMLS are eligible. The article can be an Original, Review or Technical Article, a Case Study or a Scientific Letter. Excluded are Editorials, Reports, or Fellowship Treatises. No application is necessary. The Editor and Deputy Editor will decide which article in each issue is deemed worthy of the award. If, in their opinion no article is worthy, then no award will be made. Their decision is final and no correspondence will be entered into. Winner of the Med-Bio Journal Award from the November 2007 issue was Gloria Evans from the Laboratory for Cell and Protein Regulation, University of Otago, Christchurch for her article "Review of molecular methods for medical microbiology testing". N Z J Med Lab Sci 2007; 61 (3): 72-83.

Editorial

Authorship (and non-authorship) criteria and new author requirement

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Recent studies have shown that many scientists are unaware of what criteria constitutes authorship of articles submitted to peer reviewed biomedical journals. For instance, Dhaliwal et al found that, although 65% of academics were aware that authorship criteria existed, only 44% correctly identified a source (1). A previous editorial in this Journal gave basic guidelines for authorship (2). The World Association of Medical Editors (WAME), of which the Editor is a Board Director, recently published its criteria for authorship and is, in main, reproduced below.

Basically, the three main criteria for authorship are:

- Substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data
- Drafting the article or revising it critically for important intellectual content
- Final approval of the version to be published

Authors must meet all three above criteria.
Non-authorship criteria are:

- Performing technical services
- Translating text
- Identifying patients for study
- Supplying materials or equipment
- Providing funding or administrative oversight over facilities

These contributions should be acknowledged in the manuscript with the contributors knowledge and permission.

Many journals nowadays require all authors of submitted articles to specifically state what contributions they have made. As from now, that also will be a requirement for submission to the Journal. This information will be published with the accepted paper. Additionally, one author must take responsibility for the integrity of the work as a whole. These criteria have now been added to the "**Brief instructions to authors**" published on the index page of each issue of the Journal.

Thus, in a covering letter when submitting articles to the Journal state:

- That the submitted work is original, has not previously been published nor is under consideration by another journal.
- That all authors justify authorship by supplying information on their contributions.
- That all authors approve submission of the final version.
- That all cited references have been checked against the original article.
- Provide details on contributions made by each author.
- Supply the name of the author who responsibility for the integrity of the work as a whole.

WAME criteria for authorship (as posted on www.wame.org)

Everyone who has made substantial intellectual contributions to the study on which the article is based (for example, to the research question, design, analysis, interpretation, and written description) should be an author. It is dishonest to omit mention of someone who has participated in writing the manuscript and unfair to omit investigators who have had important engagement with other aspects of the work.

Only an individual who has made substantial intellectual contributions should be an author. Performing technical services, translating text, identifying patients for study, supplying materials, and providing funding or administrative oversight over facilities where the work was done are not, in themselves, sufficient for authorship, although these contributions may be acknowledged in the manuscript, as described below. It is dishonest to include authors only because of their reputation, position of authority, or friendship ("guest authorship").

Many journals publish the names and contributions of everyone who has participated in the work ("contributors"). Not all contributors necessarily qualify for authorship. The nature of each contributors' participation can be made transparent by a statement, published with the article, of their names and contributions and WAME encourages this practice.

One author (a "guarantor") should take responsibility for the integrity of the work as a whole. Often this is the corresponding author, the one who sends in the manuscript and receives reviews, but other authors can have this role. All authors should approve the final version of the manuscript.
It is preferable that all authors be familiar with all aspects of the work. However, modern research is often done in teams with complementary expertise so that every author may not be equally familiar with all aspects of the work. For example, a biostatistician may have greater mastery of statistical aspects of the manuscript than other authors, but have somewhat less understanding of clinical variables or laboratory measurements. Therefore, some authors' contributions may be limited to specific aspects of the work as a whole.

Number of authors.

Editors should not arbitrarily limit the number of authors. There are legitimate reasons for multiple authors in some kinds of research, such as multi-center, randomized controlled trials. In these situations, a subset of authors may be listed with the title, with the notation that they have prepared the manuscript on behalf of all contributors, who are then listed in an appendix to the published article. Alternatively, a "corporate" author (e.g., a "Group" name) representing all authors in a named study may be listed, as long as one investigator takes responsibility for the work as a whole. In either case, all individuals listed as authors should meet criteria for authorship whether or not they are listed explicitly on the by-line. If editors believe the number of authors is unusually large, relative to the scope and complexity of the work, they can ask for a detailed description of each author's contributions to the work. If some do not meet criteria for authorship, editors can require that their names be removed as a condition of publication.

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Disputes about authorship are best settled at the local level, before journals review the manuscript. However, at their discretion editors may become involved in resolving authorship disputes. Changes in authorship at any stage of manuscript review, revision, or acceptance should be accompanied by a written request and explanation from all of the original authors.

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Comparison of methicillin-resistant *Staphylococcus aureus* (MRSA) carriage rate in the general population with the health-worker population

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Abstract
The emergence of antibiotic resistance in bacteria is becoming a widespread problem and a major health issue. Methicillin resistant *Staphylococcus aureus* (MRSA) is becoming increasingly frequent in hospitals. In this study we compared the carriage rate of MRSA in 100 health workers at North Shore Hospital with the carriage rate of MRSA in the general population (100 staff members and students of Auckland University of Technology) and found a prevalence of MRSA in the health workers of 4%, but none in the general population group. The implication of MRSA carriage in health workers is discussed.

Key words: *Staphylococcus aureus*, MRSA, methicillin, prevalence, health workers

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Introduction
Staphylococcus aureus is a virulent bacterium that can cause serious infections including skin and soft tissue infections, wound infection, bacteremia, pneumonia, and endocarditis (1). It is an organism that is renowned for its potential to acquire resistance to antimicrobial agents. In 1961 there were reports from the United Kingdom of *S. aureus* that had acquired resistance to methicillin (methicillin-resistant *S. aureus*) (2). The clinical significance of oxacillin-resistant (methicillin-resistant) *S. aureus* is heightened by the fact that these isolates are usually resistant to other anti-staphylococcal agents such as lindamycin, erythromycin, tetracycline, and sometimes trimethoprim/sulphamethoxazole, with the exception of vancomycin (4). Although oxacillin-resistant staphylococci appear susceptible in vitro to other β-lactam agents, (such as the cephalosporins) these are clinically ineffective. Therefore, all oxacillin-resistant staphylococci are reported as resistant to all β-lactam agents, including cephalosporins, β-lactam/β-lactamase inhibitor combinations, and imipenem (2).

Currently nearly 90% of *S. aureus* isolates are penicillin-resistant. Methicillin and other semi-synthetic penicillins were successful in treating penicillin-resistant *S. aureus* until the 1980s, when methicillin resistance emerged (3). Methicillin is no longer commercially available, and in many laboratories testing for methicillin resistance has been replaced by oxacillin and/or cefoxitin. Cefoxitin gives clearer endpoints because it is a better inducer of the *mecA* gene (2). The genetic basis of methicillin resistance in MRSA is the acquisition of *mecA* gene, that renders MRSA resistant to all β-lactam antibiotics (2,3).

The methicillin resistance gene (*mec*A) encodes a methicillin-resistant penicillin-binding protein (PBP2a) that is not present in susceptible strains and is believed to have been acquired from a distantly related species. MecA is carried on a mobile genetic element, the staphylococcal cassette chromosome *mec* (SCC*mec*), of which four forms have been described that differ in size and genetic composition.

The origins of the major MRSA strains are still poorly understood. It has been proposed that all MRSAs were descended from a single ancestral *S. aureus* strain that acquired *mec*A, but more recent studies show that some MRSAs are very divergent, implying that
*mec*A has been transferred between *S. aureus* families (2,4).

In the past three decades, MRSA has become wide spread in many hospitals (2) and *S. aureus* (including MRSA) is commonly found in two main carriage sites, the nose (20%) and the perineum (3%). The skin, including the hands, can be transiently contaminated (5). The major form of spread is hand borne transmission (2).

Hospital infection control staff need to limit the spread of MRSA for several reasons. There have recently been reports of strains of MRSA that have intermediate resistance to vancomycin. This is an important concern since the already limited treatment options for serious MRSA infections may become more limited due to the increase in resistance to vancomycin. Limiting the transmission of MRSA might reduce the potential for these strains to spread (6).

Another concern is the simultaneous spread of MRSA and vancomycin-resistant enterococci (VRE), possibly resulting in the transfer of the vancomycin-resistance gene from VRE to MRSA, rendering MRSA fully resistant to vancomycin. The first such isolate was detected in the United States in 2002 (7). The cost of treating an MRSA infection is another concern because vancomycin, the antibiotic most commonly used to treat MRSA infection is expensive.

Epidemiological studies have shown that since the mid 1990s, the incidence of MRSA has been increasing in New Zealand (6). In 2006 the incidence of MRSA amongst hospital patients and staff was approximately 0.17% (10). Amongst *S. aureus* isolates in N.Z, 7% were resistant to oxacillin/methicillin in 2005.

There are four main strains of MRSA, each of which has distinguishing characteristics (11). In New Zealand, different strains of MRSA can become epidemic in different geographical regions (10). EMRSA -15 is predominately isolated in hospitals, whereas WSPP is found more frequently in the community.

The aim of this study was to compare the carriage rate of MRSA in 100 health workers at North Shore Hospital with the carriage rate of MRSA in a general population of 100 staff members and students of the Auckland University of Technology.

Methods
Nasal swabs from 200 participants were collected, and inserted directly into 7% salt broth for 24 hr incubation at 37°C (12). After 24 hr the broths were sub-cultured onto mannitol salt agar (MSA, Fort Richards, USA)) and examined after a further 24 hr and 48 hr for yellow colonies. Any yellow colonies on MSA plates had a DNA

test and purity plate on Columbia human blood (Fort Richards, USA). DNA positive isolates had a coagulase test performed. All presumptive *S. aureus* isolates (coagulase positive, DNA positive yellow colonies) were subjected to testing with MRSA screen slide latex agglutination kit (Pro-Lab Diagnostic) and sensitivities by disc diffusion on Mueller-Hinton agar. The antibiotics tested were penicillin, cefoxitin, erythromycin, tetracycline, clindamycin, ciprofloxacin, gentamycin, fucidic acid, rifampicin, mupirocin and vancomycin. Zone size for determining sensitivity or resistance is shown in Table 1. Isolates of *S. aureus* showing resistance to penicillin and cefoxitin were considered positive for MRSA in this study.

Previous history for MRSA was determined where available. If a new isolate demonstrated resistance to more than penicillin and cefoxitin, a slope was sent to ESR for phage typing to assist in strain identification. PCR typing was not performed. Infection control nurses were notified of isolates of MRSA.
Positive participants were treated and followed up by infection control and occupational health nurses. The Waitemata DHB protocol for MRSA positive staff is as follows:

- Nasal: apply bactroban/fucidic acid to nostrils twice a day x 5days.
- Body wash: chlorhexidine 4% washes (shower) daily x 5 days.
- Hair wash: chlorhexidine 4% hair washes x per week 3 sets of swabs to be taken 48 hr apart.
- Follow up: swabs taken monthly for 6 months, then 6 monthly for 18 months.

Due to the small number of positive tests, statistical analysis was not carried out. The study was approved by the Northern Regional Ethics Committee.

Table 1. Interpretation of antibiotic sensitivities

	mcg	Resistant	Intermediate	Sensitive
Pencillin	10U	£28mm		≥29mm
Cefoxitin	30	£19		≥20
Erythromycin	15	£13	14-22	≥23
Cotrimoxizole	25	£10	11-15	≥16
Tetracycline	30	£14	15-18	≥19
Clindamycin	2	£14	15-20	≥21
Ciprofloxacin	5	£15	16-20	≥21
Gentamycin	10	£12	13-14	≥15
Fucidic acid	10	£18	19-20	≥21
Rifampicin	5	£16	17-19	≥20
Mupirocin	5	£13		≥14
Vancomycin	30			≥15

Results
The main finding of our study was that the carriage rate of MRSA in health workers was 4% compared to 0% in the control population. The cohorts were well matched for age while there were more females than males in both groups. Average age of the health workers and control population were 42 yr and 40 yr respectively while there were 85 females in the health workers group and 64 females in the control group.

All four individuals in whom MRSA was isolated were nurses with more than 2 years of clinical experience. Three of the MRSA positive nurses were strain EMRSA-15 and one WSPP1. All four subjects showed resistance to cefoxitin and penicillin, three showed additional resistance to erythromycin, two additional resistance to ciprofloxacin and one additional resistance to clindamycin. One of the four nurses had previously isolated MRSA. Three of the four nurses were treated with the standard Waitemata DHB protocol and subsequently showed negative results for MRSA (Table 2).

Table 2. Characteristics of the MRSA positive nurses

Age	44	30	56	59
Length of service in the hospital	4 years	2 years	7 years	15 years
Strain	EMRSA-15	EMRSA-15	EMRSA-15	WSPP1
Antibiotic resistance	cefoxitin, penicillin and erythromycin-	cefoxitin, penicillin, ciprofloxacin and erythromycin	cefoxitin penicillin, ciprofloxacin, erythromycin and clindamycin	cefoxitin and penicillin
Results after treatment for 5 days	Neg results for 3 swabs following treatment	Neg results for 1st &2nd swab and positive for 3rd swab	Neg results for 3 swabs following treatment	Not treated

Discussion
This study, although small in size, found that 4% of health workers in the Waitemata DHB hospital carried MRSA, compared to none of the healthy volunteers in the wider Auckland community. Our figure of 4% is higher than the national reported incidence of 0.17% amongst hospital patients and staff (10), but compares with studies conducted on patient cohorts in the United Kingdom, where MRSA incidence ranged between 1.6% and 5.3% (15). However, our study is novel, because we compared carriage rate of MRSA in New Zealand health workers with the general community. We could not find any published studies with which to directly compare our results.

Detecting or identifying a MRSA can be done in several ways. Detecting the presence of *mecA* gene using PCR is the gold standard for identification and confirmation of MRSA isolates (13). Detection of the altered protein PBP2a using commercially available MRSA screen slide latex agglutination kits is a highly specific and sensitive method. In this test, latex particles sensitized with a monoclonal antibody against cell wall PBP2a specifically react with methicillin-resistant staphylococci to cause agglutination. In this study, the latex agglutination, together with antibiotic sensitivity by disc diffusion was used. Several studies (5,11,14) have shown that cefoxitin is superior to oxacillin in the detection of MRSA, and cefoxitin was used in our study.

It is interesting to note that the first MRSA positive health worker was sensitive to ciprofloxacin as EMRSA-15 strains are often resistant to ciprofloxacin (11). The concern is that these health workers could transmit MRSA to vulnerable patients. Patients are at higher than normal risk of acquiring *S. aureus* infection particularly as the in-patient population tends to be older, sicker and weaker, making them more vulnerable to infection.

Various strategies exist for controlling the spread of MRSA within healthcare settings. Preventative measures include laboratory surveillance and screening for MRSA (5), promoting careful hand washing with soap and water rather than the antibacterial gels in common use, gowning and gloving by staff and eradication of MRSA from colonized people (decolonization therapy). Most institutions use a combination of these strategies. Potential side effects associated with the use of eradication therapy include the development of further antibiotic resistance or the possibility of adverse reaction to the antibiotic. Although clinical trials of eradication therapy in colonized healthcare workers (healthy adults) exist, in practice healthcare workers are not always systematically screened for MRSA and offered eradication therapy (3). In contrast, many hospital patients are routinely screened and offered antibiotics or drugs if they are found to be colonized (5,9). Currently at WDHB all staff members have a pre-employment nasal swab to detect MRSA but no further screening if the staff member tests negative. Patients admitted to the hospital will be screened if they are perceived to be at higher risk of MRSA carriage.

The results from this study suggest a small, but significant MRSA carriage in health workers that could be transmitted to vulnerable patients. This does raise the question whether all health workers should be screened at regular intervals for MRSA, as well as the usual pre employment screen .

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Accurate diagnosis of high-affinity vWF-platelet disorders: a case study of pseudo von Willebrand disease

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Abstract
A pregnant woman with a family history of abnormal bleeding was referred to our laboratory for investigation. Abnormal PFA-100 results were initially found and von Willebrand Factor (vWF) studies indicated a functional discordance (ie functional assays of vWF being significantly reduced as compared to vWF:Ag) suggestive of type 2 von Willebrand Disease (vWD). Further testing raised doubts that this was a 'typical' case of type 2B vWD and may in fact be pseudo (platelet type) vWD. Pseudo vWD was confirmed after in-depth laboratory testing and this case study demonstrates that this very rare sub-type of vWD may be under diagnosed due to its phenotypic similarities to type 2B vWD.

Key words: pseudo von Willebrand disease; platelet aggregation; PFA-100; RIPA; platelet glycoprotein-1b receptor.

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Case history
A 24 year old woman presented at 36 weeks of pregnancy for investigation due to a maternal history of bleeding. The patient's mother had suffered a post partum haemorrhage controlled with utero-tonic medication and also a maternal cousin resident in Australia had been diagnosed with vWD (no data available)

Initial investigation included a PFA-100 where results were abnormal (Table 1). A full von Willebrand Factor (vWF) screen was then performed which showed a normal FVIII:C and vWF:Ag but a slight reduction in the assays which measure vWF function (vWF:RCo and vWF:CB) indicating a possible functional vWF discordance. Type 2 vWD was considered as a possible diagnosis and platelet aggregation studies were considered appropriate to test at this stage. Testing platelet response to the agonists ADP, Collagen, Epinephrine and Ristocetin showed an increased response to low concentration Ristocetin (Figure 1a) which is consistent with type 2B vWD (1). (A normal control response to low concentration Ristocetin is shown in Figure 1b). The patient had a normal platelet count which is not usually the case in type 2B vWD. vWF multimer analysis (Figure 2) showed that high, intermediate and low molecular weight multimers were present which also was not consistent with type 2B vWD. With vWF levels rising during pregnancy it was decided to repeat the assays post partum for confirmation.

Due to a risk of bleeding during childbirth it was recommended that 1-deamino-8-D-arginine vasopressin (DDAVP) 0.3mcg/kg be given prior to delivery and if abnormal bleeding occurred despite this an infusion of 1500 units of Biostate (a plasma-derived concentrate containing vWF and factor VIII) would be given. The patient went on to receive DDAVP prior to epidural catheter insertion and some hours later underwent caesarean section for failure to progress. There was some excess incision site bleeding noticed in the post-operative period and Biostate was administered with good results. The patient then demonstrated an uneventful recovery and the child was well.
Six weeks post partum the patient returned for further investigations. PFA-100, vWF studies, vWF multimers and Ristocetin Induced Platelet Aggregation (RIPA) were repeated with results confirming the previous findings (Table 1) except that vWF parameters were now more significantly reduced but still demonstrated the functional discordance.

Type 2B vWD was a likely diagnosis although this pattern of results is also found in pseudo vWD. To differentiate these two disorders RIPA mixing studies were performed. RIPA mixing studies involve performing RIPA on a combination of:

(i) patient platelets + patient plasma
(ii) patient platelets + control plasma
(iii) control platelets + control plasma
(iv) control platelets + patient plasma

Enhanced response to low concentration Ristocetin was found only in the presence of patient platelets indicating an intrinsic platelet defect was the cause of the high affinity platelet–vWF interaction. Therefore the diagnosis was pseudo (platelet type) vWD which is a rare disorder arising from a gain of function mutation in the gene for the platelet glycoprotein-1b receptor (*GP1BA*).

To confirm this diagnosis mutation analysis of the *GP1BA* gene was performed. Coding regions and flanking intronic sequences of the *GP1BA* gene were amplified by PCR and analysed by bi-directional automated sequencing. The sequence was then compared to the reference genomic DNA sequence for *GP1BA* (GenBank NT010718). A mutation c.746G>T was identified which results in the substitution p.G249S (G233S in old nomenclature) and is known to cause pseudo vWD (2).

Subsequent genetic analysis was performed on the patient's child and the mutation found in the mother was also found in the child. Due to the large amount of blood required phenotypic analysis has not been performed yet.

Some details of this case can also be found in another publication on a review of pseudo vWD and its diagnosis (3).

Discussion
Platelet–vWF interaction in the process of platelet adhesion involves optimal binding of the A1 domain of vWF protein with the GP Ib in the platelet membrane GPIb-IX-V receptor complex. Abnormally increased affinity between this receptor and ligand results in spontaneous binding of high molecular weight multimers (HMWM) of vWF to platelets. Increased HMWM vWF clearance, spontaneous platelet agglutination and variable thrombocytopenia ensue. When considering a high affinity vWD with discordant parameters for vWF:Ag and function, the diagnostic strategy should follow a logical stepwise sequence. The RIPA procedure using patient platelet rich plasma should be performed using standard (1.5mg/ml) and low (0.5mg/ml) concentration Ristocetin. Aggregation to 0.5mg/ml Ristocetin is abnormal confirming a high affinity interaction. The next step involves mixing studies, designed to separately explore the two components of the high affinity interaction: the patient's platelets and vWF plasma protein, as a mutation in either can result in this phenotype.

Pseudo vWD is a rare disorder arising from a gain of function mutation in the gene for *GP1BA* as first described in 1982 (3). Type 2B first described in 1980 (4) is caused by functionally defective vWF with high affinity for *GP1BA* resulting from a mutation in the vWF gene located on chromosome 12.
Patients with these bleeding disorders have similar phenotypic parameters and clinical features, ie functional VWF discordance, increased response to low concentration Ristocetin in RIPA testing and in most cases although not ours thrombocytopenia and loss of HMWM. Incidence of pseudo vWD is unknown but it is feasible that under diagnosis may be evident (5) as some patients previously classified as type 2B vWD may in fact have pseudo vWD.

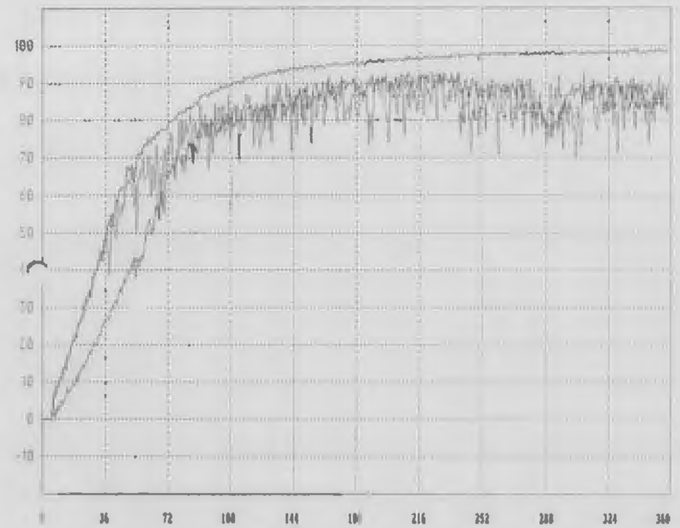
Correct distinction between these two disorders has important clinical implications. Treatment of type 2B vWD is usually with a virucidally–treated plasma derived vWF/FVIII concentrate; treatment of pseudo vWD may require platelet transfusion.

Conclusion
This case study highlights the need that if a patient presents with a functional vWF discordance and enhanced response to low concentration Ristocetin in RIPA testing, a diagnosis of type 2B vWD should not be automatically assumed. Further testing by RIPA mixing studies and appropriately targeted gene mutation study is necessary for correct diagnosis.

Table 1. Laboratory results

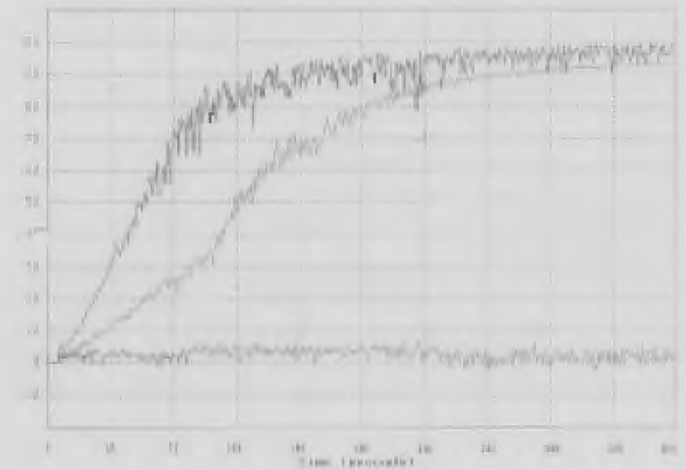
Assay	Antenatal	Post partum	Reference range
PFA-100 (COL/EPI)	247	>300	85 – 160 sec
PFA-100(COL/ADP)	197	>300	60 – 120 sec
FVIII:C	180	70	50 – 200%
vWF:Ag	98	47	50 – 200%
vWF:RCo	39	19	50 – 200%
vWF:CB	47	27	50 – 250%
Platelet count	206	229	150 – 450 X 10^9/L
VWF Multimers	High, intermediate and low molecular weight multimers present	High, intermediate and low molecular weight multimers present	High, intermediate and low molecular weight multimers present

Figure 1a. Patient RIPA results – shows abnormal response to 0.5mg/mL Ristocetin



Green tracing – Ristocetin 1.5mg/mL. Red tracing – Ristocetin 1.0mg/mL.
Blue tracing – Ristocetin 0.5mg/mL

Figure 1b. Control RIPA results - shows normal response to 0.5mg/mL Ristocetin



Green tracing – Ristocetin 1.5mg/mL. Red tracing – Ristocetin 1.0mg/mL.
Blue tracing – Ristocetin 0.5mg/mL

Figure 2. VWF Multimer Analysis

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A new graduate's experience: the challenges and advantages

Caroline Knight, BMLSc

Biochemistry Department, Counties Manukau District Health Board, Auckland

For me, choosing to study the Bachelor of Medical Laboratory Science programme at AUT felt almost like a natural choice. After speaking with my guidance councillor this was the course she most recommended, taking into account my love of science, hospitals and shift work options. She was also able to sell me on the idea due to the degree being job specific.

After applying to AUT I was accepted before sitting my bursary exams (as they still were when I was a young'n). The change from high school to university was marked, and I found that having a regimented programme of classes was great. There was no having to find a timetable to suit the papers I chose, the entire schedule was mapped out already without containing any clashes. All lectures preceded the practical classes meaning I had the understanding required to get the "lab" done. AUT gives its BMLSc students exposure to all the disciplines it offers in the first two years before having to make a specialist decision. This means as a student I acquired basic skills in all facets of medical laboratories and I was able to make an informed decision before specialising in the third year.

The knowledge that I was expected to gain on the BMLSc programme is very vast and comprehensive. The textbooks are HUGE! It was expected that I would understand entire diseased states, not just how results will be affected in the laboratory. I should also know how a given condition can be treated and the advantages and pitfalls of all options. This is beneficial as a student, but it also makes one become the first point of call for any medical condition relating to a family member (not the best job to have)!

In the BMLSc course there are five routine subjects (microbiology, biochemistry, haematology, immunology and transfusion science), with every second year cytology and histology being offered. AUT does not provide a specific genetics speciality paper, but students are allowed to undertake the Massey University paper and cross-credit back to AUT. Students must pick two specialist subjects from these. The lecturers at AUT are all very friendly and approachable. Any question asked of them will be answered to your level of understanding and they all have years of experience working in the laboratory industry. For specific topics in which they don't have the experience, visiting lecturers are brought in from hospital or community laboratories to aid in the teaching process and ensure that students are gaining knowledge on current workplace practices.

To my mind the biggest perk to studying at AUT was that all my relevant study was completed before going onto my placement, so I knew what I was doing when I got to "the real world". Assignments issued during my final year were all relevant to the work I was exposed to as a student employee in my placement year. AUT gets special bonus points in my mind as they allowed me to sit four examinations overseas. After applying to the examination board I was able to courier my completed exam papers back for marking. One disadvantage of the BMLSc degree at AUT is that placement exams are only undertaken at the end of the year. I acknowledge that this is so all students are expected to have the same knowledge and understanding, but it is always harder to recall accurately all steps involved during my first semester speciality. The hardest part of placement year for me was being required to move away from home. There is strong competition for placements being offered in Auckland, with those first being offered to students with children, as it would be harder to uproot them from their current education and homes. Other considerations are given to those students with a spouse and/or a mortgage. I can understand why the people with children are given priority, but I do feel that in beginning this degree there is an understanding that you may have to leave the greater Auckland area, and this rule should be applied fairly to all students.

Being required to leave Auckland for the placement year is stressful, as there is no aid from AUT in finding accommodation. I became isolated from the support system I had in place and generally part time employment is hard to find as one is in each city for such a short time frame. AUT is vigilant in monitoring the progress of each student. I was required to submit my logbook fortnightly so they could see the progress being made. The programme leader makes scheduled phone calls and/or visits to see how students' placements are going.

Textbooks seemed like the single biggest expense I had to pay, and as textbooks can cost up to $200 each, they make the first week of any semester VERY costly. The AUT prescribed textbook editions I found, were more recent than those used in the laboratory libraries that I encountered. Textbooks are worth the initial expense though as they are invaluable learning tools and I still refer to mine.

My first placement was with NZBS at Wellington Hospital. I opted for working full time, in order to get my placement done as quickly as possible so I could return home to Auckland where I didn't have to pay accommodation expenses. During my time in Wellington I got to work both in the hospital Blood Bank and at the processing centre. Upon returning to Auckland I did a further three week part time placement in the accreditation area.

The initial reality shock of working in a laboratory was obvious. For example, at University doing a practical, a student is given 3 samples and completes them. In the hospital laboratory there is at least 10 times that volume of work just in the first three hours of a day. There were also more anti-natal titres than I would have guessed. I seemed to do numerous panels during my placement. There was also a roster system that I had to come to terms with. Staff starts the day where they are rostered, but as the day moves on they seem to move around and work in all areas.

I have to say a big thanks to all the staff of Wellington NZBS, they were very welcoming to me. They are really supportive and it was liking working with friends, it never seemed like going to work was a chore. All the staff function well together, and interacted in a highly professional manner with all hospital staff.

My second placement was also at Wellington Hospital working in the immunology and serology departments. This was another reality shock, as work was batched and done daily or weekly, so that meant larger batch sizes. As these are smaller departments I was only able to work four days a week. There were only 2 or 3 staff who could supervise me. Both the serology and immunology departments had a closer interaction with the doctors than in blood bank, and weekly meetings were scheduled between laboratory staff and department doctors.

The workflow in immunology was regularly interrupted by bone marrow extractions, as these could never be pre-planned until the day before, so it was hard to get a consistent workflow. It also meant 2 of the staff would be out of the laboratory collecting samples leaving only 1 person to supervise me. A positive aspect was that I got to go and observe them preserving the bone marrow and the facilities used for long term storage.

Assays were done in larger batch sizes, the purpose of which was cost savings. Testing for HIV viral loads were done weekly and were only performed by the scientists, not myself, as kits were $10,000 each! The DHB could not afford for a student to undertake a batch and make a costly mistake. The serology and immunology departments have very skilled staff. They are interpreting many results visually and must be accurate and consistent. Reading of

results is restricted to those who are qualified and all results that require interpretation are read by at least two independent staff to ensure agreement before reporting.

So, I completed my life in Wellington safely, came back to Auckland and sat my final exams successfully which meant I got to graduate in March 2005. Next on the agenda was to gain employment. The "fortunate" employer was Counties Manukau District Health Board. After being interviewed for the biochemistry department I was offered a job. This was another shock, as neither of my placements were in this speciality. The department was larger than any I had worked in before. They have many part-time staff whose schedules vary. This meant inconsistencies in my day-to-day inauguration of specific areas. I had to find out who held the knowledge to best answer the questions I had and most importantly I needed to observe the team dynamics and fit in with them.

I felt like I was still a student, as I was being rostered around all the areas of the department with a scientist who could supervise and explain everything to me. To ensure I was gaining the correct knowledge I was given a logbook to fill out with details of pertinent tests that are preformed here and the understanding of why the test is done and how the result is obtained.

The biggest change that I noticed after my first roster through the department was that responsibility was now on me. I did not have to get everything I did signed off, and I was able to authorise others' work. My name would be released with patient results if I authorised the work. The year I started as a scientist was also the year that the CPD programme came into being. There were still a few questions surrounding the programme, especially for me being on an interim Practicing Certificate. We were unsure if I needed all 100 points that first year as I didn't start work until April and wasn't actually a scientist until October. No matter what ruling was made on that front I did not need to worry as for my year from April to December I earned 99 points.

As I was now an employee I realised that to extend myself I needed to ask for things, such as getting internet access, going on training courses and getting supplies. If I asked I did receive, and this has helped me in doing my job well. The biggest shock to being an official employee and having the "buck stop with me" was being rostered on phones, talking to doctors and nurses and answering their questions, such as what colour tube, how much longer until tests are completed, where does the test get sent, how much sample needs to be collected, and why did we reject a previous sample? This reinforced the knowledge I gained while being rostered throughout the department.

The final shock came when I began participating in the 24 hour roster system, working weekends and night shifts. Prioritising work, control of many analysers at once, embracing quality control protocol and having a basic ability to fix errors as they occur on the analysers without calling in help was expected of me.

Overall my experience in the laboratories I have worked in has been very positive. There does need to be a profile lift of "what we do" to the general public. We are highly skilled specialists who do have a great responsibility in patient care even though we mostly never see the patients directly. I appreciate that the NZIMLS is working to raise the profile of medical laboratory scientists, the "backroom girls and boys" of the diagnostic world of medicine, through the career expos held at colleges throughout the country.

Acknowledgement

I wish to thank Jacquie Case of Counties Manukau District Health Board for her guidance in preparing this article for publication.

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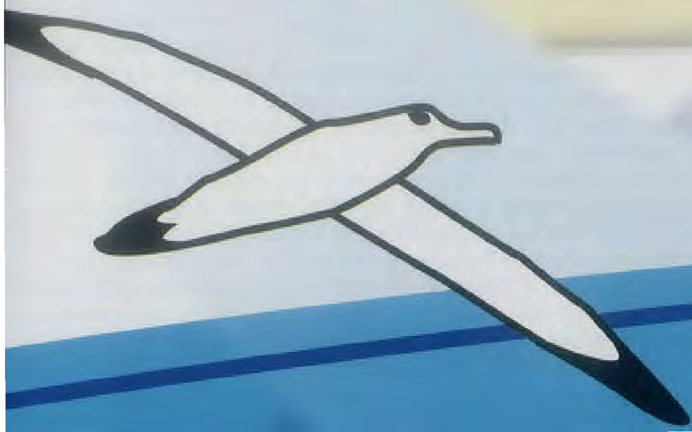

Invitation to attend:

On behalf of the New Zealand Institute of Medical Laboratory Science and the New Zealand Society of Cytology, I would like to invite you to join us for the 2008 combined Annual Scientific Meeting in Dunedin. The conference is being held at the St David's Lecture Theatre complex at the University of Otago.

The theme for the conference is 'Southern Most Science". The scientific programme will have a broad appeal and will be stimulating and challenging. The conference is an opportunity to assemble some of the vast and cultivated minds from the Otago Medical School, complimented by national and international speakers as well as the energy and vitality that we have within our own profession.

To complement the scientific programme is our industry exhibition. Our exhibiting companies support our conferences each year and our exhibitors will be part of the conference within the St David's Lecture Theatre Complex.

The poster session has become part of our event and this will be held again this year. We encourage medical laboratory scientists and technicians to take part by producing a poster and being eligible to be chosen for the Hugh Bloore Memorial Prize. We wish to also encourage our colleagues to proffer papers.

Dunedin is a very special place and we hope that many of you will choose to take advantage of its delights by attending the 'Southern Most Science' conference in our beautiful city.

Trevor Rollinson
Conference Convenor

Social Programme

Icebreaker - Opening of the Exhibition

The welcome function will give you the opportunity to catch up with friends, new and old whilst enjoying drinks and nibbles. It is also a special time for our exhibitors to host this function around their exhibits.

Conference Dinner

The stunning modern facilities make the Otago Museum, Dunedin's most versatile and unique venue for functions. This is the venue for the conference dinner. The theme is a 'touch of tartan' with an "to after five" dress code.

Come and enjoy this evening with pre-dinner music being provided by 'one of our own' followed by a delicious meal and entertainment provided by the X-Factor (a powerful mix of entertainment catering for both a dancing crowd and those who prefer to watch the performance).

Tickets will be limited so please don't be disappointed!

For information and registration visit www.nzimls.org.nz

New Zealand Institute of
Medical Laboratory Science

3rd NZIMLS Nth Island Seminar & Phlebotomy/Specimen Services SIG Meeting

Call for presentations

Place:	**Novotel, Palmerston North**
Date:	**10th May 2008**
Registrations:	**9 - 10.00am**
Commencing:	**10.00am**
Finishing:	**5.00pm**
Seminar Dinner:	**7.00pm**

Organisers are now accepting presentations for the North Island (NI) Seminar and Phlebotomy/Specimen Services SIG meeting. If you have a case study, evaluated some new lab equipment or method, conducted some MLS research, or if you have something that may be of interest to seminar or SIG meeting attendees, then please consider presenting. With the ASM in Dunedin this year, this meeting provides a great opportunity to earn CPD points for 2008.

Make plans to come to Palmy, meet with colleagues, enjoy the hospitality and take in the atmosphere at the Novotel!

Enquiries to:

Registrations & accommodation:	**fran@nzimls.org.nz**
NI seminar scientific programme:	**c.j.kendrick@massey.ac.nz** **jandm@medlabcentral.co.nz**
Phlebotomy/SS programme:	**janek@medlabcentral.co.nz**

www.nzimls.org.nz

Biochemistry Special Interest Group Meeting

Saturday June 14th 2008
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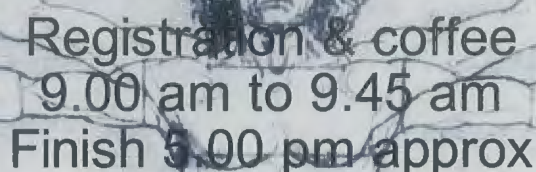

Registration & coffee
9.00 am to 9.45 am
Finish 5.00 pm approx

Dinner 7.00 pm

WANTED

Presentations on Specialist Biochemistry, Method Development, Point of Care, Automation, Information Technology, Quality Management, Laboratory Management, Case Studies and Process Improvement

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Contact Ross Hewett,
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Auckland City Hospital,
Email: rossh@adhb.govt.nz

Microbiology Update Course.
A Microbiology Update Course was held at the PPTC during November 2007.There were six participants: Andrew Tekanene and Barieti Itaaken from Tungaru Central Hospital in Kiribati, Geoffrey Wuatai from Rarotonga Hospital in the Cook Islands, Aldrin Solomon from Pohnpei State Hospital, Pohnpei, Federated States of Micronesia, Herjet Lomae from Majuro Hospital Marshall Islands and Likiak Alik from Arthur Sigrah Memorial Hospital, Kosrae, Federated States of Micronesia.
The course had both theoretical and practical sessions on current aspects of Microbiology. Lectures were given to the participants by John Elliot, Director of the PPTC and from both Pathologists and Laboratory Scientists of Wellington Hospital's Microbiology Department.
Brian Currie from Fort Richard Laboratories visited the PPTC and spoke to the participants about new developments in culture media and Rapid Spot Identification methods.
Dr Ron Mackenzie Chairman of the PPTC presented certificates to the participants at the end of the Course.
A Certificate presentation was also held for Rosemary Tekoaua, from Kiribati who had been on a 3 month attachment in the Cytology Department at Aotea Pathology, Wellington.
During one weekend John Elliot drove the Course participants to Hawkes Bay where they got a chance to see another part of New Zealand apart from Wellington.

Participants of the 2007 Microbiology Course and friends of the PPTC.

PPTC 2008 TRAINING COURSES:

Haematology and Blood Cell Morphology, 31 March – 24 April 2008
During the first part of the course students will study general haematology procedures and methods and the interpretation of the common haematological parameters. They will also discuss the use of automated cell counters and their maintenance and the importance of routine use of quality control samples in haematology. In the second part of the course participants will be provided with guidelines for the objective microscopic evaluation of white cells, red cells and platelets in both health and disease. Trainees will learn to correlate the blood film findings with results obtained from manual and/or automated methods for red and white cell parameters.

The origin of all blood cells will be discussed from the common stem cell through all stages of development. The course is designed to give trainees confidence in the examination of blood films and to be able to recognise the abnormal findings in common blood cell disorders.

Blood Bank Technology, September 2008
This course will include units of study covering the theoretical and practical aspects of routine blood grouping, blood group antigens, crossmatch techniques, antibody detection, transfusion reactions, haemolytic disease of the newborn, screening blood for infectious agents, blood donor selection, organisation of a blood bank and the appropriate use of blood components in transfusion medicine.

Online Distance Learning Courses
Early in 2008 Immunology, the last module of the Certificate in Medical Laboratory Technology course will be run through WHO's POLHN. Later in the year a Laboratory Management and Quality Systems course will be available to senior laboratory staff also through POLHN.
It is hoped that the Certificate in Medical Laboratory Technology modules will be repeated starting in the second half of the year and in addition other courses are being planned for offering through POLHN.

PPTC Board
The PPTC is governed by a Board of Management made up of four people elected at the AGM. Currently these are Dr Ron Mackenzie, Chairman [who along with Assoc Professor Sandie Ford was the co-founder of the PPTC], Mike Lynch, Treasurer; Marilyn Eales and Rob Siebers. In addition, the two PPTC staff are ex-officio members.
It is interesting to note that all four elected Board members are Life Members of the NZIMLS an honour that has been granted them for services to the profession.

Elected members of the PPTC Board
Left to Right: Rob Siebers, Mike Lynch, Marilyn Eales, Ron Mackenzie.

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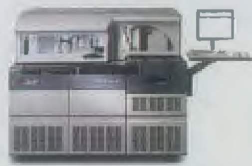
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Journal-based questionnaire

Below are 10 questions based on articles in this, the April 2008 issue of the Journal. Read the articles fully and carefully, most questions require more than one answer.

Answers are to be submitted through the NZIMLS web site. Make sure you supply your correct email address and membership number. The site will remain open until Friday 30 May 2008. You must get a minimum of 8 questions fully right to obtain 5 CPD points.

Journal questions:

1. What are the main criteria for authorship.
2. What is the genetic basis of methicillin resistance of MRSA and what does it render MRSA resistant to.
3. What does the methicillin resistance gene encode and what is it carried on.
4. In how many health workers was MRSA isolated and which strains were they.
5. What is the 'gold standard' for identification and confirmation of MRSA isolates.
6. Why are patients at a higher than normal risk of acquiring *S. aureus* ***infection.***
7. Pseudo (platelet type) von Willebrand disease is a rare disorder arising from what.
8. What is the recommended drug therapy for risk of bleeding during childbirth and if bleeding occurs despite this, what further treatment is recommended.
9. To differentiate Type 2B vWD from pseudo vWD which mixing studies are performed.
10. What is Type 2B vWD caused by.

Questions and answers for the November 2007 journal-based questionnaire.

1 Name the basic steps for the extraction of DNA/RNA from commercial columns. **Lysis. Precipitation. Binding to membrane/washing. Elution of purified DNA/RNA.**

2 What is required to synthesise new dsDNA and what does it facilitate.
Taq **DNA polymerase. Binding and joining of the complementary nucleotides.**

3 What steps does the reverse transcription PCR process consist of.
Annealing of the primer(s) to the single stranded RNA. RT enzyme catalysing primer extension to create first strand DNA.

4 Which instruments are combined into one for real time PCR.
PCR thermal cycler and an integrated fluorescence detection device.

5 Name an example of strand displacement amplification.
BD Probe Tec™ assay for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoea*.

6 How does the ligase chain reaction differ from PCR.
It ligates the probe molecule.

7 Which resistant strains belonging to the *Eschericia* genus were isolated from chickens in Millar's study.
E. coli **and** ***E. fergusonij.***

8 What are the possible transmission routes of antibiotic resistant bacteria.
Food-borne transmission. Surface water via eating crops. Meat pet food via pets.

9 What is the unique characteristic of the sideroblastic anaemias and what is it the result of.
Amorphous iron deposits in erythroblast mitochondria. Ineffective insertion of iron into the developing haem molecule.

10 What are the effects of alcohol on haematopoiesis.
Directly toxic to developing cells. Causes lowering of the plasma concentration of pyridoxal phosphate.

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NZIMLS Journal Prize

Council of the NZIMLS has approved an annual Journal prize for the best case study accepted and published in the Journal during the calendar year. The prize is worth $200.

Case studies bring together laboratory results with the patient's medical condition and are very educational. Many such studies are presented at the Annual Scientific Meeting, SIG meetings, and the North and South Island Seminars, yet are rarely submitted to the Journal for wider dissemination to the profession. Consider submitting your case study presentation to the Journal. If accepted, you are in consideration for the NZIMLS Journal Prize and will also earn you additional CPD points. Please contact the Editor or any Editorial Board Member for advice and help. Contact details are on the NZIMLS web site (www.nzimls.org.nz) as are instructions to authors.

No formal application is necessary but you must be a financial member of the NZIMLS during the calendar year to be eligible. All case studies accepted and published during the calendar year (April, August and November issues) will be considered. The Editor, Deputy Editor and President of the NZIMLS will judge the eligible articles in December each calendar year. Their decision will be final and no correspondence will be entered into.

Winner of the 2007 NZIMLS Journal Prize was Rossi Holloway, formally from PathLab Bay of Plenty, now LabPlus Auckland for her article "Sideroblastic anaemia secondary to chronic alcoholism: a case study and review", N Z J Med Lab Sci 2007; 61 (3): 69-70.

Reviewers for 2007

The Editors would like to thank various individuals for refereeing submitted articles to the Journal during 2007. All submitted articles undergo peer review in order that the Journal maintains its high standard since its inception in 1948. Additionally, thoughtful comments and suggestions made by referees help authors in ensuring that their paper, if accepted, is put in front of the reader in the best possible light. Not all papers submitted to the Journal are accepted and published. In the last five years about 20% have been rejected as being either scientifically unsound, not novel enough, or not applicable to the broad subject of medical laboratory science. Additionally, one submitted paper from overseas was found, upon data base checking, to have been published previously in another journal. Duplicate publication is definitely not allowed.

Guidelines are given to referees. Below are the main guidelines and we advise authors to take these into account when writing articles. Put yourself in the shoes of a potential referee and ask, is what I wrote good science, carefully argued, and does it make sense. Additionally, we would encourage authors to ask colleagues not involved with the manuscript to read and comment

Guidelines to referees:

- Identify and comment on the major strengths and weaknesses of the study design and the methodology.
- Comment accurately and productively upon the quality of the author's interpretation of the data, including acknowledgement of its limitations.
- Comment on the major strengths and weaknesses of the manuscript as a written communication, independent of the design, methodology, results, and interpretation of the study.
- Provide the author with useful suggestions for improvement of the manuscript.
- Ensure comments to the author are constructive and professional.
- Comment on whether cited references are appropriate, and/or whether other references are more appropriate.

The Editors are not and cannot be experts in the many different disciplines of medical laboratory science and thus rely on quality peer review by referees. The following have generously and professionally given their time and experience in peer reviewing articles submitted to the Journal during 2007:

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School of Medicine and Health Sciences, University of Otago Wellington

Journal article questionnaire

for the Haematology Special Interest Group

Spuriuos elevation of automated platelet counts in secondary acute monoblastic leukaemia associated with tumour lysis syndrome (TLS). Pathol Lab Med 1999; 123: 1111-1114.

Questions
True or False (Questions 1-6)

1. TLS can lead to life-threatening metabolic derangements including acute renal failure.
2. TLS was originally described in patients with Burkitt's Lymphoma following chemotherapy.
3. TLS occurs in other types of leukaemias, lymphomas and solid organ tumours.
4. Cytoplasmic fragments of the white cells can lead to falsely elevated platelet counts.
5. The two cases discussed in this journal article are therapy related acute monoblastic leukaemia complicated by disseminated intravascular coagulation and TLS.
6. DIC is common in Acute Promyelocytic leukaemia and in approximately 20% of patients with acute monoblastic leukaemia.
7. What is the mechanism of Tumour Lysis Syndrome?
8. What is TLS characterised by?
9. In the first case how many hours after the initiation of therapy did the peripheral blood smears reveal apoptotic tumour cells with pyknotic nuclei and cellular fragments of the neoplastic cells?
10. In the two cases what type of special stain was used to confirm the postivity of the cellular fragments of the neoplastic cells?

Prepared by Kamla Prasad, Cellular Morphology Technical Specialist, Haematology Lab, LabPlus, Auckland Central Hospital. Email: kamlap@adhb.govt.nz or Ph 3074949 Ext 7580.

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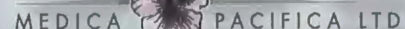
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British Journal of Biomedical Science Abstracts

Amess JA, O'Neill W, Giollariabhaigh CN, Dytrych JK. A six-month audit of the isolation of *Fusobacterium necrophorum* from patients with sore throat in a district general hospital. Br J Biomed Sci 2007; 64 (2): 63-5.
Fusobacterium necrophorum is an obligate anaerobe believed to be a member of the normal flora of the human oropharangeal and urogenital tract. It has been associated with deep-seated infections and was first described in 1936 by Lemierre, a French microbiologist. There is now strong evidence to suggest that it is also a cause of recurrent sore throat and persistent sore throat syndrome (PSTS) without leading to full systemic infection. It is considered to be the second most common cause of sore throat after group A beta-haemolytic *streptococci*. This study was performed over a six-month period (October 2004 to March 2005) at the Eastbourne District General Hospital. All throat swabs received in the laboratory are cultured routinely for haemolytic group *A streptococci* and pathogenic *Corynebacteria* spp. During the study period an extra fastidious anaerobic blood agar plate with neomycin was inoculated, with a 30 microg vancomycin disc placed at the junction of the second and third streaks. This was examined after 48 h for the presence of *F. necrophorum*. A total of 1157 swabs were processed during the study period: 156 were positive for haemolytic group A *streptococci*, 57 were positive for *F. necrophorum*, 47 for group C haemolytic *streptococci*, nine for group G haemolytic *streptococci*, and one was positive for *C. ulcerans*. Patient age ranged from less than a year old to 88. The majority of *F. necrophorum* isolates were from patients in the 11-25 age group, with an isolation rate of 9.48% (44/464). This age group accounted for 40% (464/1157) of the swabs received during the study period and 77% (44/57) of these were positive for *F. necrophorum*. Group A haemolytic *streptococci* showed an overall isolation rate of 13.5%, with peaks of 23% in the 0-10 and 26-35 age ranges. Together, these two organisms were responsible for 18.4% (213/1157) of all throat infections in this study. The results presented here indicate that *F. necrophorum* is second to group A haemolytic *streptococci* as a cause of sore throat, especially in the young adult, and introduction of routine culture should be considered.

Evans GE, Anderson TP, Seaward LM, Murdoch DR. Evaluation of the Mycoplasma Duo kit for the detection of *Mycoplasma hominis* and *Ureaplasma urealyticum* from urogenital and placental specimens. *Br J Biomed Sci* 2007; 64 (2): 66-9.
This study compares the Mycoplasma Duo kit for the detection of genital mycoplasmas with conventional culture using A7 differential agar for the detection of *Mycoplasma hominis* and *Ureaplasma urealyticum* in clinical samples. Detection of the mycoplasmas is based on the specific metabolic properties of each organism to hydrolyse either arginine or urea. The Mycoplasma Duo test showed a significantly higher detection rate than did culture, although many of the culture-negative results may have been due to the presence of bacterial overgrowth.

Ito T, Sekizuka T, Murayama O, Moore JE, Millar BC, Taneike I, Matsuda M. Cloning, sequencing and molecular characterisation of a cryptic plasmid from a urease-positive thermophilic *Campylobacter* (UPTC) isolate. *Br J Biomed Sci* 2007; 64 (2): 70-3.
Cloning, sequencing and molecular characterisation of a cryptic plasmid, pUPTC237, from a urease-positive thermophilic *Campylobacter* (UPTC) isolate obtained from the natural environment in Northern Ireland is reported in this study. Based on the determined DNA sequence, the pUPTC237 DNA was identified as a circular molecule of 3828 bp with a G+C content of 29.5%. As with other plasmid DNAs from Gram-negative bacteria, pUPTC237 contained an A+T-rich region (A+T content: 95%), followed by multiple direct tandem repeat units of 22 bp, characteristic of a replication origin and iteron sequence. A possible open reading frame (ORF)-1 was located upstream of the A+T-rich region and the iteron sequence that encoded a 460 amino acid protein similar to the mobilisation (mob) protein and two putative promoter structure sequences at the -35 and -10 regions and a possible ribosome binding site occurred upstream of the start codon for the ORF-1. Moreover, three possible ORFs (a short ORF-2 encoding 26 amino acids, similar to repA; an ORF-3 encoding 341 amino acids, similar to repB; and an ORF-4 encoding 96 amino acids with unknown function) were also identified. There are also two putative promoter structures for these three ORFs at the -35 and -10 regions upstream of the possible ORF-2. A possible transcription termination region was identified downstream of ORF-4. Northern blot hybridisation analysis suggested that these four ORFs constitute an operon and generate a messenger RNA (mRNA) transcript.

Nagano Y, Watabe M, Porter KG, Coulter WA, Millar BC, Elborn JS, et al. Development of a genus-specific PCR assay for the molecular detection, confirmation and identification of *Fusobacterium* spp. *Br J Biomed Sci* 2007; 64 (2): 74-7.
A genus-specific polymerase chain reaction (PCR)-based assay is developed for the detection and identification of clinically relevant *Fusobacterium* species, including *F. nucleatum* and *F. necrophorum*. Two 16S ribosomal DNA (rDNA) primers, FUSO1 (forward primer: 5'-GAG AGA GCT TTG CGT CC-3' [17-mer]) and FUSO 2 (reverse primer: 5'-TGG GCG CTG AGG TTC GAC -3' [18-mer]) are designed to target conserved regions of the 16S rDNA gene for *Fusobacterium* spp. Subsequent proof-of-principle studies employing this assay detected *Fusobacterium* spp. in the faeces of eight (10%) out of 80 patients with suspected gastrointestinal infection. This assay may be used for the genus-specific detection of *Fusobacterium* spp. from clinical specimens and for subsequent species identification.

Dutta R, Jha R, Gupta S, Gupta R, Salhan S, Mittal A. Seroprevalence of antibodies to conserved regions of *Chlamydia trachomatis* heat shock proteins 60 and 10 in women in India. *Br J Biomed Sci* 2007; 64 (2): 78-83.
Persistent, untreated chlamydial infection causes chronic stimulation of the host immune system against immunogenic antigens such as chlamydial heat shock proteins (cHSP) 60 and 10. In order to find the seroprevalence of antibodies to cHSPs, enzyme-linked immunosorbent assay (ELISA) is performed using specific peptide sequences to measure antibody response against major outer membrane protein (MOMP), cHSP60 and cHSP10 in patient sera. In this study, 255 patients attending the gynaecology out-patient department (March 2004 to August 2005) of Safdarjung Hospital were enrolled. Of these patients, 107 were diagnosed with cervicitis while 52 had pelvic inflammatory disease (PID)/infertility. *Chlamydia trachomatis* infection in endocervical specimens is diagnosed by a direct fluorescence assay (DFA) and the polymerase chain reaction (PCR). In 75 (29.4%) of the *C. trachomatis*-positive women, 50 (66.7%) were ELISA positive for MOMP 48 (64.0%) were positive for cHSP60 and 46 (61.3%) were positive for cHSP10. The anti-MOMP index correlated positively with anti-cHSP60 ($R = 0.522$, $P < 0.01$) and anti-cHSP10 ($R = 0.286$, $P < 0.05$). Antibody titre for MOMP was significantly higher than that for cHSP60 (1:5; $P < 0.01$ and 1:25; $P < 0.05$). Moreover, patients with PID/infertility showed significantly higher antibody titres for cHSP60 and cHSP10 when compared to patients with cervicitis at dilutions of 1 in 50, 1 in 250, 1 in 1250 ($P < 0.001$) and at 1 in 6250 ($P < 0.01$).

Moriarty TF, Elborn JS, Tunney MM. Effect of pH on the antimicrobial susceptibility of planktonic and biofilm-grown clinical *Pseudomonas aeruginosa* isolates. *Br J Biomed Sci* 2007; 64 (3): 101-4.
The pH at the site of infection is one of a number of factors that may significantly influence the in vivo activity of an antibiotic prescribed for treatment of infection and it may be of particular importance in the treatment of cystic fibrosis (CF) pulmonary infection, as acidification of the airways in CF patients has been reported. As *Pseudomonas aeruginosa* is the most frequent causative pathogen of CF pulmonary infection, this study determines the effect that growth at a reduced pH, as may be experienced by *P. aeruginosa* during infection of the CF lung, has on the susceptibility of clinical *P. aeruginosa* isolates, grown planktonically and as biofilms, to tobramycin and ceftazidime. Time-kill assays revealed a clear loss of tobramycin bactericidal activity when the isolates were grown under acidic conditions. MIC and MBC determinations also showed decreased tobramycin activity under acidic conditions, but this effect was not observed for all isolates tested. In contrast, growth of the isolates at a reduced pH had no adverse effect on the bacteriostatic and bactericidal

activity of ceftazidime. When the isolates were grown as biofilms, the pH at which the biofilms were formed did not affect the bactericidal activity of either tobramycin or ceftazidime, with neither antibiotic capable of eradicating biofilms formed by the isolates at each pH. This was in spite of the fact that the concentrations of both antibiotics used were much higher than the concentrations required to kill the isolates growing planktonically. These results show that growth in an acidic environment may reduce the susceptibility of clinical *P. aeruginosa* isolates to tobramycin.

Chaturvedi P, Machacha CN. Efficacy of *Raphanus sativus* in the treatment of paracetamol-induced hepatotoxicity in albino rats. *Br J Biomed Sci* 2007; 64 (3): 105-8.

In the present study, the efficacy of a methanol extract of *Raphanus sativus* root (RSME) is tested in albino rats that developed hepatic damage due to administration of paracetamol (100 mg/kg body weight) for 30 days. Twenty rats were divided into three experimental groups (E1, E2, E3) and one control group (EC). Two doses of RSME (80 and 120 mg/kg body weight) were administered orally to E1 and E2, respectively, and a mixture of RSME (120 mg/kg) and paracetamol (100 mg/kg) was administered to E3 for 21 days. Group EC and another group of normal rats (EN) that served as controls were administered distilled water. At the end of the experiment rats were bled to assay thiobarbituric acid reactive substances (TBARS), serum glutamate oxaloacetate transaminase (SGOT) and serum glutamate aspartate transaminase (SGPT), reduced glutathione (GSH) and catalase. Results indicated that RSME reduced the levels of TBARS, SGOT and SGPT, and increased the level of GSH and the catalase activity in E1 and E2 as compared to the EC group. Group E3 showed decreases in TBARS, SGOT and SGPT levels, but the results were not statistically significant compared with the EN group. There was also a marked depletion in GSH level and catalase activity in this group. RSME reduced lipid peroxidation induced by paracetamol and brought the levels of SGOT and SGPT to normal, indicating liver recovery. It also brought about repletion of GSH levels and recovery of catalase activity. Results for group E3 indicated that RSME was not able to reverse the effects of paracetamol if administration continued.

El-Bassiouni EA, Helmy MH, El-Zoghby SM, El-Nabi Kamel MA, Hosny RM. Relationship between level of circulating modified LDL and the extent of coronary artery disease in type 2 diabetic patients. *Br J Biomed Sci* 2007; 64 (3): 109-16.

The impact of diabetes on health is due almost entirely to a series of complications that characterise the disease. It is associated with an increased incidence of macrovascular complications including coronary artery disease (CAD). The aim of the present study is to evaluate the possible relationship between the circulating levels of the modified derivatives of low-density lipoprotein (LDL) and the development of angiopathy in type 2 diabetic patients with CAD. The status of the antioxidant defences and the role of supplementation with antioxidant combinations are also studied in these patients. The study was conducted on three groups: group I (controls); group II (type 2 diabetic patients without complications--CAD[-]); and group III (including type 2 diabetic patients with stable CAD - CAD[+]). Patients in group III received adjunct treatment of antioxidant tablets for three months. The results of the present study clearly indicated that there was excessive exposure to oxidative stress in diabetic patients. The increase in free radicals was coupled with disturbance in free radical scavengers, particularly the glutathione system. The disturbance was more prominent in CAD(+) patients. The study has shown alteration in the lipid profile in diabetic groups, where the oxidised LDL (ox-LDL) levels were significantly higher than in control subjects. Diabetics with CAD had higher levels of ox-LDL than did patients without CAD. The intima/media thickness (IMT) of the carotid artery was within clinically accepted normal values if the ox-LDL level was below 100-110 u/L. Once the ox-LDL exceeded this range, IMT increased sharply with the increase in plasma ox-LDL. It seems that the level of ox-LDL should be kept below an upper limit of the 100-110 u/L range in order to avoid the serious atherosclerotic effects of this factor. The results demonstrate that plasma levels of ox-LDL correlate with the extent of coronary artery disease in type 2 diabetic patients and suggest that elevated levels of ox-LDL, can serve as an independent and significant predictor for future cardiac events in type 2 diabetic patients with CAD.

Eshraghi P, Hedayati M, Daneshpour MS, Mirmiran P, Azizi F. Association of body mass index and Trp64Arg polymorphism of the beta3-adrenoreceptor gene and leptin level in Tehran Lipid and Glucose Study. *Br J Biomed Sci* 2007; 64 (3): 117-20.

In this study the association between beta3-adrenoceptor gene polymorphism and serum concentration of leptin with body mass index (BMI) is investigated. Using subjects in the Tehran Lipid and Glucose Study, genotyping of the Trp64Arg polymorphism of the beta3-adrenoreceptor gene was performed using a restriction fragment length polymorphism-polymerase chain reaction (RFLP-PCR) technique was used and the association with obesity was investigated. At total of 197 men and 204 women were divided into four groups (BMI<20, 20< or =BMI<25, 25< or =BMI<30, BMI< or =30) and 97, 98, 104 and 102 subjects, respectively, were placed randomly in the four groups. Leptin level was determined by an enzyme immunoassay (EIA) method and FBS, HDL-C, triglyceride and total cholesterol levels were determined by an enzyme colorimetric method. Body mass index (BMI) was also measured. The A (Arg) allele frequency was 0.08 among the population and its presence was significantly associated with increase of leptin level (AA/TA, 30.5+/-24.8 ng/mL; TT, 22.6+/-20.9 ng/mL; P=0.014) but there was no significant association with increased BMI (AA/TA, 27+/-5.6 kg/m2; TT, 25.4+/-5.5 kg/m2; P=0.072). These data show that the presence of the Arg64 allele at the beta3-adenoceptor gene locus is related to increase in leptin level in this population, but is not related to body mass index.

Kalansooriya A, Holbrook I, Jennings P, Whiting PH. Serum cystatin C, enzymuria, tubular proteinuria and early renal insult in type 2 diabetes. *Br J Biomed Sci* 2007; 64 (3): 121-3.

This study investigates the association between serum cystatin C, serum creatinine concentrations, N-acetyl-beta-D-glucosaminidase (NAG enzymuria), urine alpha1-microglobulin (alpha1-MG) and beta2-microglobulin (beta2-MG) levels in subjects with type 2 diabetes (n=40, 20M/20F, age range 25-65 years; duration of diabetes 8-10 years) and age- and gender-matched healthy controls (n= 20). Exclusion criteria were absence of gross proteinuria, hypertension, dyslipidaemia or cardiovascular disease. Fasting blood samples and mid-stream specimen of urine (MSSU) were collected and serum creatinine, cystatin C, urine creatinine, NAG enzymuria, alpha1-MG and beta2-MG were measured. Diabetic subjects were separated into two groups based on albumin:creatinine concentration ratio. Group A: <3.5 (mg/mmol creatinine), group B: 3.5-35 (mg/mmol creatinine). While serum creatinine concentrations remained within the laboratory reference range for all groups, serum cystatin C concentration (mg/L) was significantly increased in group B (1.79 +/- 0.42 [mean +/- SD] compared to both control [0.81 +/- 0.10] and group A values [0.95 +/- 0.10]; both P<0.001). NAG enzymuria (units/mmol creatinine) was increased in both diabetic groups compared to control values (group B: 122 +/- 7, group A: 70 +/- 5, controls 27 +/- 2, all P<0.001). alpha1-microglobulin (microg/mmol creatinine) concentrations, similar in both the control group and group A diabetics at 1.10 +/- 0.10 and 1.11 +/- 0.21, respectively, were significantly elevated in group B at 2.10 +/- 0.41 (both P<0.01). Similarly, elevated beta2-MG (microg/mmol creatinine) levels were also observed in group B compared to both group A and control values (3.20 +/- 0.21 vs. 1.80 +/- 0.51 and 0.91 +/- 0.11, respectively; both P<0.001). In addition, group B levels were significantly higher than group A (P<0.001). These observations suggest that serum cystatin C is a more appropriate and effective biomarker for the overall estimation of GFR than serum creatinine values. In addition, increased serum cystatin C values were also associated with early renal tubular insult in subjects with type 2 diabetes, as characterised by increased NAG enzymuria, alpha1- and beta2-microglobulin excretion.

Blann AD, Marwah SS, Cobley AJ, Bareford D. Increased levels of soluble P-selectin correlate with iron overload in sickle cell disease. *Br J Biomed Sci* 2007; 64 (3): 124-6.

Homozygous sickle cell disease (SCD) is characterised by increased soluble P-selectin (sP-selectin), suggesting increased platelet activation, and high non-transferrin-bound iron (NTBI), reflecting iron overload, possibly due to blood transfusion. Hypothesising a relationship between these processes, we measured both markers in 40 SCD patients and 40 age/gender/race-matched controls, finding increased levels of each marker in the patients (both P<0.001), but more pertinently a significant NTBI/sP-selectin correlation (r=0.52, P<0.001). Both indices were increased in the blood of 15 recently-transfused patients compared with 25 three-month transfusion-free patients (P<0.001), but only sP-selectin was higher in present sickle crisis (P<0.001). We suggest that increased NTBI associated with blood transfusion iron overload in SCD may promote platelet activation.

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Answers to HSIG questionnaire

1. True
2. True
3. True
4. True
5. True
6. True
7. Massive necrosis of malignant cells and subsequent release of intracellular degradation products such as purines, phosphate and potassium into the bloodstream.
8. Hyperuricemia, hyperphosphatemia, hyperkalaemia, lactic acidosis and hypocalcaemia
9. 10 hours
10. Alpha- naphthyl butyrate esterase

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