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The *New Zealand Journal of Medical Laboratory Science* (the Journal) is the official publication of the New Zealand Institute of Medical Laboratory Science (NZIMLS). The Journal is peer reviewed and publishes original and review articles, case studies, technical communications, and letters to the Editor on all subjects pertaining to the practice of medical laboratory science. The Journal is open access (www.nzimls.org.nz/nzimls-journal.html) and is published three times per year in April, August, and November. Hard copies are circulated to all NZIMLS members and universities and research units in New Zealand and overseas. Current circulation is about 2,200 copies per issue. Printing is by PWA Griffin, Christchurch on environmentally responsible paper using elemental chlorine free third party certified pulp sourced from well managed and legally harvested forests. The Journal is indexed by CINAHL, EMBASE, SCOPUS, Informit, Thomson Gale, EBSCO and Biosis Citation Index, and the Journal Editors are members of the World Association of Medical Editors (www.wame.org).

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In this issue

Rob Siebers, Editor

Priapism is a rare disorder, defined as a persistent erection of the penis that continues more than four hours beyond, or is unrelated to, sexual stimulation. In this issue, Christian Christian from SCL Dunedin presents a case study of priapism where the initial physical examination suggested ischaemic priapism and the diagnosis was later confirmed by cavernous blood gases results. The cavernous blood gases showed hypoxia, hypercapnia, and acidosis, which confirmed the diagnosis of ischaemic priapism.

Congenital antithrombin deficiency is associated with a high risk of deep vein thrombosis and pulmonary embolism. The antithrombin heparin binding site defect subtype is generally a mild form of antithrombin deficiency, carrying a lower risk of thrombosis than other antithrombin deficiencies and this form of antithrombin deficiency may, therefore, be inappropriately treated with anticoagulant therapy. A two dimensional crossed electrophoresis method to detect the heparin binding site defect is used in some countries, however, this method is expensive and time consuming. Janko Legner and colleagues developed a single dimensional gel electrophoresis method with immunofixation, with and without heparin, to detect the heparin binding site defect in patients with antithrombin deficiency. Their results indicated that it was possible to distinguish antithrombin heparin binding site defect from normal antithrombin and from antithrombin Type I defects using this method and has potential for routine use in diagnostic medical laboratories.

In this issue are three Editorials. The first, by Ross Hewett from LabPLUS Auckland, discusses a case brought against a medical laboratory worker for breach of privacy by the Health Practitioners Disciplinary Tribunal. Ross points out that Membership of the NZIMLS is subject to a Code of Ethics and

breach of those ethics will be subject to review by NZIMLS Council, and in proven cases, their membership is far from certain.

The second Editorial by the Editor asks whether the Journal is still alive after 70 years of continuous publication. He points out that the popular Journal questionnaire, undertaken by nearly 1000 participants each issue for valuable CPD points, is totally dependent on articles published in that issue. Lately, not many articles have been submitted from New Zealand medical laboratories, despite a number of excellent and interesting presentations at SIG or other scientific meetings that are worthy of publication.

The third Editorial by the CPD Co-ordinator and the Editor discusses ten years of the Journal questionnaire. They point out that the Journal questionnaire has been a popular and successful method for members to use as part of their CPD points' activities with between 900 and 1000 submissions received for each edition of the journal. It is currently the fourth most popular means of earning CPD points They further express concern regarding a number of instances in the past of collusion of participants with others in answering the questionnaire. Finally they plead for more articles submission from New Zealand medical laboratories as the sustainability of the journal questionnaire is solely dependent on there being enough suitable articles published in the journal to base the questions around.

Finally, a new series start in this issue, namely Science Digest, in which Associate Professor Michael Legge presents snippets of interest to the scientific community from the world literature. Journal questions may be derived from this.

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Patient information, a sacred trust

Ross Hewett, LabPLUS Auckland

The role of Laboratory Manager can be a varied one and a bit like Forest Gump's box of chocolates, you just don't know what you are going to get.

The recognition of Medical Laboratory Science within our community is too often unrecognised and the public's general perception of our profession is usually based on their experiences with a Phlebotomist. They have very little understanding of the science and technology that supports the analysis of their samples and with the results of the analysis conveyed to them via their health practitioner, we are unseen in the process.

So our interface with the public and patient population is very limited and it's only when there is some sort of media attention, usually some adverse event, do we become known.

In early 2015 I had the unfortunate task of reporting one of our colleagues in LabPLUS to the Medical Sciences Council of New Zealand for breach of privacy after this employee had been dismissed from our organisation in December 2014. Subsequent to the reporting to the MSCNZ, an investigation was commenced resulting in the Health Practitioners Disciplinary Tribunal laying charges against this practitioner. The hearing occurred in May 2016 and the findings published on July 8th 2016. The full transcript of the hearing and its findings can be found on the Health Practitioners Disciplinary Tribunal's website.

The case was reported via a number of different media channels including the New Zealand Doctor and the official summary from the Health Practitioners Disciplinary Tribunal outlining the actions of this practitioner and those of us involved in the investigation and our subsequent actions.

The various interpretations and reporting of this event would have certainly influenced the reader's perception of our profession and those of us working in it. I will leave it to the readers of this editorial to access these various reports via the links above and make your own judgement on how they would have been perceived by the public at large.

It is always a sad occasion when any adverse event occurs in healthcare and especially when a fellow practitioner takes it upon themselves to consciously deviate from process for whatever reason they feel legitimates their course of action. The wider impact of that decision affects the whole profession with the potential of bringing the profession into disrepute.

We all sign confidentiality clauses in our contracts and we have within the practice of Medical Laboratory Science the trust of our patients and the public at large to uphold that trust and to ensure we behave in a way that maintains the integrity of their lab results and our profession as a whole.

When a fellow practitioner breaks that trust and regardless of it becoming public knowledge or not, as guardians of our profession, we have no choice but to take a course of action that censures that practitioner and ensures the patient information remains confidential.

Membership of the NZIMLS is subject to a Code of Ethics and as a Council, we have a choice as to who we admit as members and also who we revoke membership. Regardless of the outcome of the Health Practitioners Disciplinary Tribunal in this or any other case, the future membership of the NZIMLS of a practitioner found in breach of the Privacy Act or any other Legislation will be subject to review by Council and their membership far from certain.

Within the NZIMLS Code of Ethics, the obligations and duties of all our members is clearly stated and in particular clause 1 where it is our duty to uphold the dignity and honour of our profession and clause 9 where we are bound by the Privacy Act.

It is hoped in the future, any publicity about Medical Laboratory Science is seen by our patients and our community in a positive light where the dedication and expertise of all practising Medical Laboratory Scientists and Technicians is widely recognised, understood and embraced by all.

NZIMLS Code of Ethics

- 1. It is the duty of Members to uphold the dignity and honour of the profession, to accept its ethical principles and not engage in any activity that would discredit the profession.*
- 9. All members are bound by the Privacy Act and shall respect the confidential and personal nature of patient records and shall not disclose information to anyone without patient's consent except where the best interests of the patient requires or the law demands.*

AUTHOR INFORMATION

Ross Hewett, MNZIMLS, President¹ and Laboratory Manager²

¹NZIMLS, Rangiora

²LabPLUS, Auckland

Correspondence: rossh@adhb.govt.nz

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70 years old and still alive?

Rob Siebers, NZIMLS

No, it's not about me as Editor (not quite 70 years old yet!), but about our Journal. Upon the birth of our professional organisation in 1945 and incorporation on the 9th of April 1946, members of the New Zealand Association of Bacteriologists unanimously decided that a professional journal was necessary for the dissemination of scientific knowledge thought to be of interest and use. From the 1st issue in April, 1946 the Journal has featured peer-reviewed articles, technical notes, and editorials pertaining to all medical laboratory science disciplines and matters.

Our professional journal was initially known as the *Journal of the New Zealand Association of Bacteriologists* from 1946 to 1960, briefly changing its name from 1961 to 1962 to the *Journal of the New Zealand Institute of Medical Laboratory Technology*, renamed the *New Zealand Journal of Medical Laboratory Technology* from 1963 to 1990, and finally from 1991 to its present title, the *New Zealand Journal of Medical Laboratory Science*.

Douglas Whillans from Auckland was the Journal's 1st Editor. In fact he was more than just the Editor, also being the printer, publisher, and advertising manager. In 1951 AM Murphy from Auckland took over, followed by JJ Cannon from Christchurch in 1956, Lois Evans and Gilbert Rose from Christchurch as co-Editors in 1958, John Case from Dunedin in 1963, RD (Bob) Allan from Dunedin in 1971, Hugh Matthews from Dunedin in 1981, Dennis Dixon-McIver from Auckland in 1983, Maree Gillies from Auckland in 1990, and finally myself from Wellington in 1994. Today the Journal is ably supported by the Editorial Board and formatted by Sharon Tozer at the NZIMLS Office.

For many years articles for the Journal predominantly came from our colleagues and pathologists in New Zealand medical laboratories, although lately more overseas articles have been submitted and published since our Journal became open access in 2011. Indeed, if it was not for overseas contributions, the recent April 2016 issue would not have had any articles in it. Looking at author contributions over the years, there have been a total of 897 authors or co-authors contributing to 1150 scientific articles since 1946. However, the majority of these, namely 638 (71.1%) have only contributed once.

So why do the majority of our colleagues not contribute? A common theme I hear when approaching potential authors is "do not have the time"; "too difficult"; or "not part of my job". Certainly, given the massive changes in the medical laboratory field over the last decade, it is understandable that laboratory personnel do not see writing articles for the Journal as a priority. This, I believe is represented by the steady decline in published articles over the years as exemplified in the Table below. However, I believe that most of us have a 'duty' to write articles for the Journal. Many excellent presentations are made by members at SIG meetings, company symposia, the North and South Island seminars, and the Annual Scientific Meetings. Many such presentations have technical gems that are worthy of wider dissemination and use, and a large number of presented case studies are educational in nature. Importantly, the Journal is reliant on such articles for providing the Journal questionnaire in each issue. Currently, close to 1,000 members rely on the Journal questionnaire in each issue for valuable CPD points.

Table 1. Publication output over the years.

10 year period	No of articles
1946 - 1955	90
1956 - 1965	115
1966 - 1975	262
1976 - 1985	264
1986 - 1995	181
1996 - 2005	147
2006 - 2015	121

As with the very first Editorial in the Journal by the founding Editor, Doug Whillans (1), and many others by succeeding Editors, this is yet another plea for material for the Journal. The Journal's Editors and Editorial Board Members are user friendly and can give you valued opinion and suggestions on any draft you may wish to send us. As our NZIMLS President, Ross Hewett, recently wrote in an Editorial, the Journal "*is still a tangible reminder of what we do, who we are professionally, and testament to our predecessors who believed and did something about it*" (2).

Yes, our Journal is still alive, but we need your contributions to keep the 'old codger' healthy and ongoing. Don't let it die! Thus deluge me with your scientific articles, technical notes, reviews, and especially case studies. I look forward to hearing from you.

AUTHOR INFORMATION

Rob Siebers, PGCertPH FNZIMLS FRSB, Editor¹ and Associate Professor²

¹NZIMLS, Rangiora

²School of Medicine and Health Sciences, University of Otago, Wellington

Correspondence: rob.siebers@otago.ac.nz

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The Journal questionnaire

Jillian Broadbent and Rob Siebers

NZIMLS

The Journal questionnaire was started in 2006 by the Editor as another aid to obtain CPD points. Questions were derived from articles published in each issue of the NZIMLS journal. Initially the questions were in a true/false format, this changed to a multiple answer-based format in 2007. At least seven out of ten correct answers were initially required to obtain five CPD points but this was increased to eight out of ten correct in 2007. Initially, submitted answers were marked by the Editor; however, this task has been undertaken by the CPD Co-ordinator since November 2011.

The Editor still sets the questions but provides the CPD Co-ordinator with the required model answers. Some of the questions are in two parts and most require multiple points to be covered in the perfect answer. Failure to cover all the required points/parts of the question is a common fault amongst those undertaking the questionnaire. Exact marks are not given for the questionnaire as there are often fractions of points involved. Participants receive an email either awarding them five CPD points (for at least eight out of ten correct answers), or informing them they did not reach the required standard. Failed participants are allowed to redo the questionnaire.

Only approximately 60% of submissions achieve ten out of ten answers correct, this is particularly disappointing as this is an 'open book' quiz. Careful reading of each article is necessary to ensure every answer is correct. Submitted answers are returned to the participant and each candidate should use these to compare against the model answers which are published in the next edition of the Journal. This should be seen as an important part of the learning or professional development process. Those who do not achieve at least eight out of ten questions correct are notified and are given further chances to submit their answers, provided they are received before the questionnaire is closed. Practitioners are not informed which questions are incorrect, as they are encouraged to re-read ALL articles to ensure they fully understand them.

Submissions must be solely the work of the person submitting, and a tick box signifying confirmation of this was added to the format of the questionnaire in 2014. Any persons suspected of colluding with others are brought to the attention of the Editor and the NZIMLS Executive Council. Council takes this matter very seriously and the Team Leader for that person is informed. If collusion is proven or admitted then the CPD points for that questionnaire are removed and a comment is placed permanently onto the practitioners CPD records. Collusion with others in answering the questionnaire has been detected on a number of occasions over the years.

The journal questionnaire has been a popular and successful method for members to use as part of their CPD points' activities with between 900 and 1000 submissions received for each edition of the journal. It is currently the fourth most popular means of earning CPD points, after the compulsory competency category, attending scientific meetings, and attending case studies/in-house meetings. Between 5.4% and 6.1% of scientists' CPD points are earned by answering the questionnaire and this has been stable since the introduction of the questionnaire. Without the journal questionnaire, many part-time and night time workers would struggle to obtain sufficient CPD points, due to limited opportunities to attend scientific or other meetings.

The sustainability of the journal questionnaire is solely dependent on there being enough suitable articles published in the journal to base the questions around. A constant fear of the Editor is that there may not be enough articles in any particular issue to derive questions from. This was nearly the case for this current issue as we originally only had one case study. However, with an original article lately being accepted, the Editor's fear was alleviated and enough questions were able to be derived from these two articles.

All members are encouraged to submit papers/articles for publication and anyone presenting case studies or scientific papers at seminars, Special Interest Group meetings or other scientific meetings are welcome to contact the Editor to discuss publication. Currently, a template for journal articles is being derived to help potential authors in submitting their scientific meetings presentations. Remember, it is your journal, they are your publications and they are your CPD points that are at stake here!

AUTHOR INFORMATION

Jillian Broadbent, FNZIMLS, CPD Co-ordinator¹ and Medical Laboratory Scientist²

Rob Siebers, FNZIMLS FRSB, Journal Editor¹ and Associate Professor³

¹NZIMLS, Rangiora

²Canterbury Health Laboratories, Christchurch

³School of Medicine and Health Sciences, University of Otago, Wellington

Correspondence: Jillian Broadbent. Email: cpd@nzimls.org.nz

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Evaluation of a single dimension gel electrophoresis with immunofixation, with and without heparin, for heparin binding site defect in antithrombin deficiency

Janko Legner¹, Julia K Phillips² and Christopher J Kendrick³

¹Wellington Southern Community Laboratories, ²Pathlab Waikato and ³Massey University

ABSTRACT

Objectives: Congenital antithrombin (AT) deficiency is associated with a high risk of deep vein thrombosis and pulmonary embolism. The AT heparin binding site (HBS) defect subtype is generally a mild form of AT deficiency, carrying a lower risk of thrombosis than other AT deficiencies. Patients with this condition are rarely identified in New Zealand and this form of AT deficiency may, therefore, be inappropriately treated with anticoagulant therapy. A two dimensional crossed electrophoresis method to detect the HBS defect is used in some countries, however, this method is expensive and time consuming. This work developed a single dimensional gel electrophoresis method with immunofixation, with and without heparin, to detect the HBS defect in patients with AT deficiency.

Methods: The method is mostly based on the Helena QuickGel Immunofixation Electrophoresis and some parts of the two dimensional electrophoresis of antithrombin method supplied by Sheffield Teaching Hospitals NHS Trust – Coagulation Department (courtesy of Peter Cooper). The method was evaluated using plasma from normal volunteers, known HBS defect patients, patients with type I defects, and purified AT.

Results: The purified AT reference protein was visualized on all gels as a single band that moved faster in the presence of heparin (enoxaparin) than in the absence of heparin. Samples from the two patients who were known to be heterozygous for AT HBS defect produced a single band when run in the absence of heparin, but 2 bands of similar staining intensity when run in the presence of heparin. A single band with the same electrophoretic characteristics as the AT reference protein was also seen in plasma samples from all five healthy volunteers and all six samples from type I AT deficient patients.

Conclusions: The results indicated that it was possible to distinguish AT HBS defect from normal AT and from AT Type I defects using this method.

Keywords: Antithrombin (AT) deficiency, heparin binding site (HBS) defect, heparin, enoxaparin, single dimension gel electrophoresis, Helena quick gel chamber, immunofixation.

N Z J Med Lab Sci 2015; 71: 07-10

INTRODUCTION

Antithrombin (AT) is a single-chain serpin glycoprotein that acts as a key regulator of clotting through its inhibitory effect on thrombin and factor Xa (1). An important feature of AT is its high affinity for binding to negatively charged glycosaminoglycans such as heparin or heparan sulfate increasing the protein's action on clotting factors 500 fold (1).

AT deficiency can be inherited or acquired with acquired deficiency seen in liver cirrhosis, nephrotic syndrome, and drug induced AT deficiency. Inherited AT deficiency is an autosomal dominant disorder with prevalence rates of 1:500-5000 in the population and more than 130 mutations in the AT gene have been reported (2). AT deficient patients can have an increased risk of venous thromboembolism (VTE), pulmonary embolism (PE), and fetal loss (2).

Two types of inherited AT deficiency exist with Type I a quantitative deficiency and Type II a qualitative defect. Type II is further subdivided into Type IIa, a mutation that affects the AT reactive domain; Type IIb, a mutation producing a defect in the heparin-binding site (HBS) of AT; and Type IIc, in which a range of mutations produce pleiotropic effects (2).

Type IIb mutations alter the heparin-binding ability of AT, resulting in less than normal augmentation of AT activity in the presence of heparin and normal AT activity in the absence of heparin (3). Individuals with heterozygous Type IIb deficiency usually have a lower risk for thrombosis while homozygous Type IIb deficiency may be associated with thrombosis at an earlier age than individuals with heterozygous Type I or other Type II deficiencies (1).

Differentiation of the types of AT deficiency is of clinical relevance, as the type will influence clinical decisions about anticoagulant therapy (1). If the heterozygous Type IIb HBS defect is not identified then this may result in the inappropriate use of anticoagulant therapy in patients with low thrombotic risk, with an associated unnecessary risk of morbidity and mortality due to bleeding.

The current diagnostic approach for AT deficiency testing in New Zealand is to perform a single functional assay with a repeat assay for confirmation. Functional assays rely on AT inhibition of either thrombin or Xa. Neither methodology reliably detects all HBS defects (2,4). Specific tests to detect Type IIb

HBS defect are not readily available (2). Genetic testing is possible, however, this is expensive and not definitive for Type IIb HBS defect with the capacity only to infer rather than confirm the functional defect. Internationally, a few expert labs offer a functional method for AT HBS defect diagnosis that requires complex and time-consuming two dimensional crossed electrophoresis (Personal communication, Peter Cooper).

A commercially available QuickGel Chamber with immunofixation offers a rapid, semi-automated method for separating proteins of differential electrophoretic mobility. This project employed the QuickGel technology and exploited the altered electrophoretic mobility that results from the heparin-binding characteristic of AT to develop and evaluate a single dimensional gel electrophoresis method for detecting Type IIb AT-HBS defect in plasma.

MATERIALS AND METHODS

Patient and volunteer samples

Plasma for testing was collected by venipuncture into sodium citrate tubes (0.105M). Specimens were collected from five healthy volunteers and six patients with genetically confirmed type I AT deficiency. Fresh blood was separated by centrifugation (2800rpm/8mins) and plasma was stored frozen at -25°C prior to testing. In addition, frozen plasma from two patients (a mother and daughter) with known AT Type IIb HBS defect samples were provided courtesy of Dr Campbell Tait, Haematologist, at the Royal Infirmary, Glasgow, UK. Frozen samples were rapidly thawed at 37°C and briefly vortexed before testing. All plasma donors provided written, informed, consent.

Materials

Purified human AT was obtained from Sigma-Aldrich®. Unfractionated heparin sodium was obtained from DBL and enoxaparin sodium (Clexane®) from Sanofi-Aventis. All materials for the electrophoresis stage were provided in the Helena QuickGel IFE Kits – Helena Laboratories Cat. No. 3551T, and were run using the Helena QuickGel Chamber Instrument. Power for the electrophoresis was supplied by an Owl™ EC-1000XL (Thermo Scientific) that was able to provide 400 V.

Method

The majority of the method was based on the Helena QuickGel Chamber with immunofixation method (5) and some parts of the two dimensional (2-D) electrophoresis of the antithrombin method supplied by Sheffield Teaching Hospitals NHS Trust – Coagulation Department (courtesy of Peter Cooper). The main reason of referring to the 2-D method was to have a starting point to test various sample and saline mixes and heparin (enoxaparin) concentrations, so that an appropriate concentration could be used.

Electrophoresis

Patient and control samples were prepared by diluting 0.1mL plasma with 0.2mL saline. Purified normal human AT was dissolved in tris-buffered saline (TBS) to a concentration of 0.1mg/mL and frozen in aliquots of 0.2mL. Frozen purified AT was thawed at 37°C and diluted one in three with TBS to provide a final concentration of 0.033mg/mL prior to each assay.

High resolution, agarose gel electrophoresis was performed using a QuickGel® Chamber (Helena Laboratories) in the presence and absence of a heparin preparation. Initially unfractionated heparin was used and subsequently a low molecular weight heparin, enoxaparin (Clexane[®], Sanofi-Aventis) was substituted, as we found that low molecular weight heparin produced less background staining and clearer bands than unfractionated heparin.

The QuickGel chamber was allowed to come up to room temperature and the power supply set to deliver 400 volts for 8 minutes. The chamber was prepared by pipetting 1mL of REP prep to the chamber floor and the gel positioned in the chamber. Any bubbles trapped under the gel were removed and the gel edges were gently wiped with lint free cloth to remove excess moisture. The surface of the gel was first dried with a blotter to remove any excess fluid and a QuickGel IFE template positioned on the gel. Two microliters of patient sample, controls and pure AT were applied to the gel template (Figure 1) and for samples that would incorporate heparin, 2uL of enoxaparin (25uL/mL) were pipetted to the template in the upper end of the gel. The loaded gel was left for 5 minutes to allow absorption of the samples into the gel. The chamber lid was positioned with the electrodes touching the ends of the gel and run at 400 V for 8 minutes.

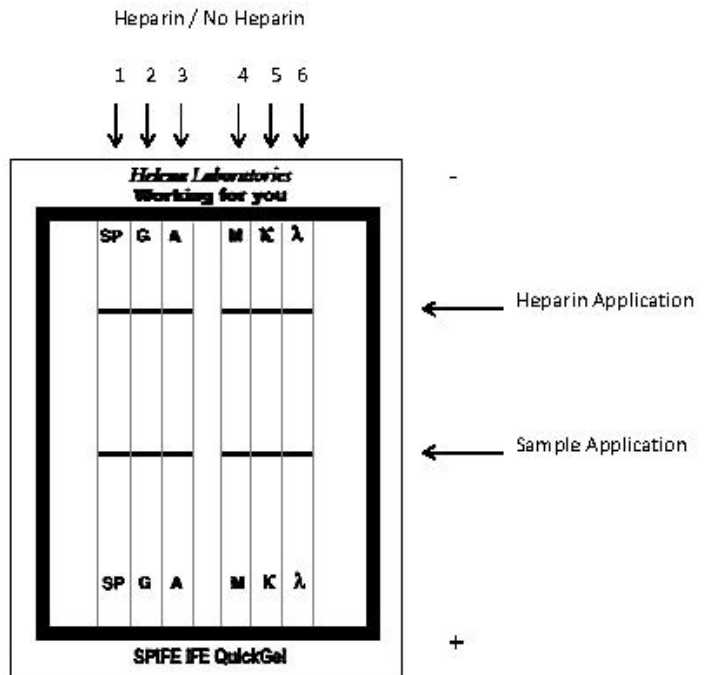


Figure 1. Representation of sample and heparin (enoxaparin) application on the Helena QuickGel.

Immunofixation

Following electrophoresis, gel blocks were removed from the gel. Undiluted AT-antibody was then pipetted directly onto the surface of the gel and spread evenly with a hollow glass roller over the right side (the bottom side) of the gel. The gel was left for four minutes to absorb the antibody and blotted to remove any excess antibody before further blotting for five minutes. A drying lid was placed on the chamber which activated the heating mode to dry the gel. The gel was then washed in TBS with gentle rotation for 10 minutes and stained using Acid Violet for four minutes followed by de-staining with citric acid wash until the gel was clear. After rinsing in deionized water, the gel was returned to the drying chamber and heated until dry.

The presence of AT was visualized as a stained precipitation of proteins produced by the interaction of AT and anti-AT in the gel. Band(s) produced by the control and AT reference samples were compared with the band(s) produced in the volunteer and patient samples. In addition, samples exposed to heparin were compared with the gels of samples tested without heparin.

Ethical approval

The research project received ethical approval from the Central Health and Disability Ethics Committee (Ethics Ref: CEN/12/06/016).

RESULTS

Figures 2-4 show the results of gels run in the presence and absence of enoxaparin for the healthy volunteer samples, patient samples, and the purified AT protein reference control. As expected, the purified AT reference protein was visualized on all gels as a single band that moved faster in the presence of heparin (enoxaparin) than in the absence of heparin. A single band with the same electrophoretic characteristics as the AT reference protein was also seen in plasma samples from all five healthy volunteers and all six samples from Type I AT deficient patients. Bands seen in AT Type I deficient patient samples were lighter in staining intensity than those from healthy volunteer samples, reflecting the low plasma AT protein concentration in these samples.

In contrast, samples from the two patients who were known to be heterozygous for AT HBS defect produced a single band when run in the absence of heparin, but two bands of similar staining intensity when run in the presence of heparin. One of these corresponded to the AT band seen in the reference AT protein, normal plasma, and type I AT deficient samples and the other was more slow moving.

On gels 1a and 3a (Figures 2 and 4 respectively) an unexpected faint second slower moving band was seen in addition to the AT band for the purified AT control sample. This was not seen on other gels.

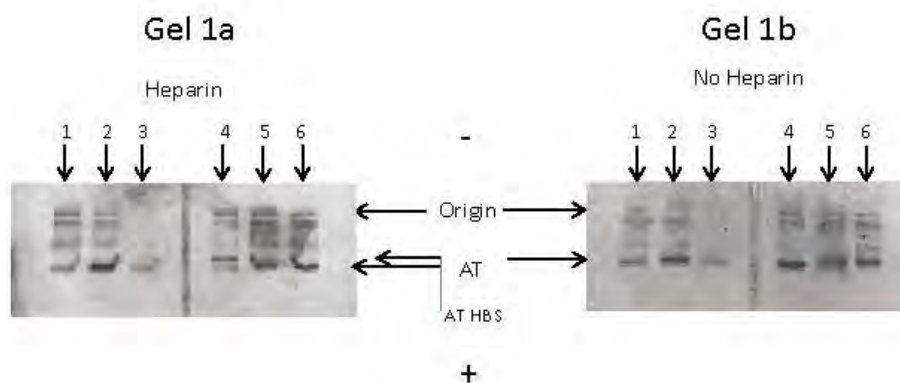


Figure 2. Gels 1a - with heparin and 1b - without heparin. Lanes: 1 - type I AT deficient #2; 2 - Normal #4; 3 - Pure AT; 4 - HBS defect positive Control #1; 5 - type I AT deficient #6; 6 - Normal #5.

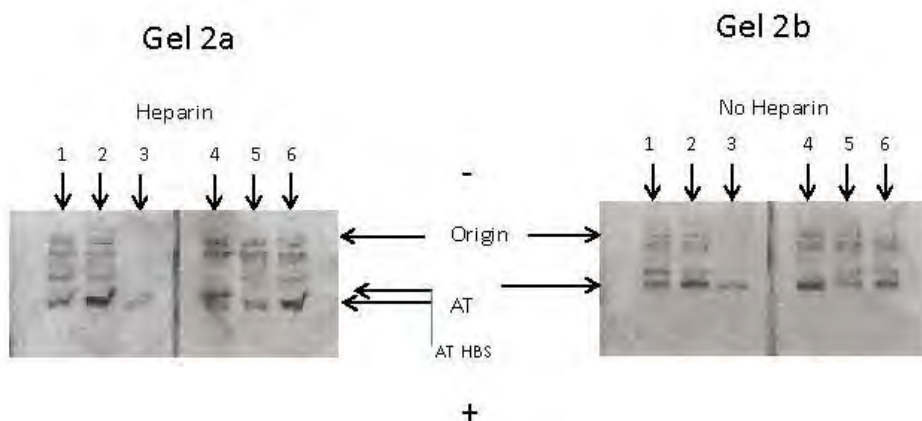


Figure 3. Gels 2a - with heparin and 2b - without heparin. Lanes: 1 - type I AT deficient #1; 2 - Normal #1; 3 - Pure AT; 4 - HBS defect positive Control #1; 5 - type I AT deficient #3; 6 - Normal #2.

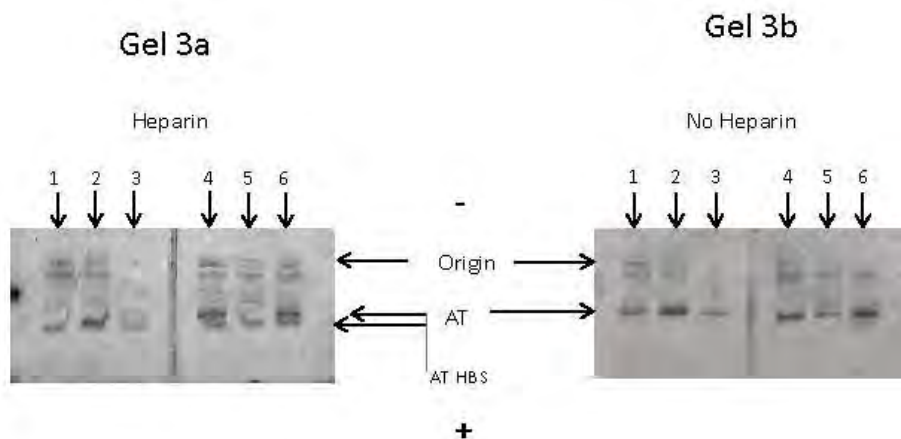


Figure 4. Gels 3a - with heparin and 3b - without heparin. Lanes: 1 - type I AT deficient #4; 2 - Normal #3; 3 - Pure AT; 4 - HBS defect positive Control #1; 5 - type I AT deficient #5; 6 - HBS defect positive control #2.

DISCUSSION AND CONCLUSIONS

This project developed a single dimension gel electrophoresis method using the commercially available QuickGel® Chamber (Helena Laboratories) and immunofixation for the detection of AT HBS defects. We have demonstrated that this assay can detect AT HBS defects and distinguish them from normal AT and from Type I AT deficient patients.

The method was technically easy to perform. The procedure was not time consuming, being completed within approximately 1.5 to 2 hours. It did not require expensive or specialized technology, utilizing a commercially available kit that is already in use in many diagnostic laboratories for immunoglobulin electrophoresis. Our data show that the test results were reproducible.

A number of AT HBS testing methods have been used and include progressive AT assays (functional assays performed in the absence of heparin) (1), molecular genetic testing, two dimensional gel electrophoresis (reviewed in references 6 and 7). These are not considered suitable for the routine diagnostic laboratory use because of concerns about the technical complexities of the methods, time, cost, and accuracy of results. Unlike the existing two dimensional gel electrophoresis method, our method required only a single dimension gel electrophoresis step and was therefore technically less complex to perform.

There were still limitations to the method such as the presence of unexplained additional bands especially on row 3 that represent pure AT on gels 1a and 3a (Figures 2 and 4 respectively). The bands may represent AT that was not bound to heparin. It is not clear why we saw these and a possible explanation is the presence of some AT HBS defects in the plasma donors that were included in the purified AT batch used at the time. Other explanations for this might be a relative excess of AT protein compared to heparin, or incomplete mixing of AT protein and heparin in the gel resulting in residual unbound AT protein. Another possible explanation would be the presence of a non-AT protein that cross-reacts with the anti-AT antibody used.

The AT Type II HBS defect is generally a milder form of AT deficiency with a lower risk of thrombosis than other forms of AT deficiency. However, some mutations resulting in HBS defects have been associated with a high thrombotic risk, such as AT Basel (Pro73Leu) (8). In order to fully assess the thrombotic risk of a HBS defect, referral of samples to a reference laboratory for genetic studies would likely still be required. The use of an assay such as the one evaluated in this project would restrict the need for genetic testing to patients with HBS defects, where with currently used functional antigenic assay it would be necessary to perform genetic testing on all AT deficient patients to obtain this information.

Our study included only two patients with HBS defects. Both were heterozygotes and were relatives from the same kindred. A larger sample size to include more examples of AT HBS defect patients would be valuable to confirm that our findings are generalizable to HBS defects produced by such mutations. It would also be valuable to determine whether the assay could detect homozygosity for HBS defects. Homozygotes for this condition are rare and sourcing samples is likely to be challenging.

Patients with this type of AT defect are rarely identified in New Zealand because of the technical difficulties and costs of existing laboratory methods. Patients with AT Type II HBS defects are at an increased risk of morbidity and mortality due to bleeding caused by the inappropriate use of anticoagulant therapy. A straightforward laboratory test that could be used to identify the heterozygous AT HBS subtype from other AT subtypes would enable clinicians to provide better care for patients with antithrombin deficiency.

This study has shown that it is possible to distinguish AT HBS defects from normal AT and from Type I AT defects in plasma samples using a rapid and simple one dimensional electrophoretic method utilizing a commercially available kit. We believe that the assay has potential for routine use in diagnostic medical laboratories.

ACKNOWLEDGEMENTS

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AUTHOR INFORMATION

Janko Legner, BMLSc MSc(MLSc), Medical Laboratory Scientist¹

Julia K Phillips, MBChB MRCP MRCPATH FRACP, Consultant Haematologist²

Chris J Kendrick, DipSci MSc MNZIMLS, Senior Lecturer³

¹Haematology, Wellington Hospital Laboratory

²Pathlab Waikato

³College of Health, Massey University, Palmerston North

Correspondence: Janko Legner, Haematology, Wellington SCL, Level 5, CSB, Wellington Hospital, Riddiford Street, Newtown, Wellington 6021.

Email: Janko.Legner@wellingtonscl.co.nz

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Priapism: a case study and review

Christian Christian

Southern Community Laboratories, Dunedin

ABSTRACT

Priapism is a rare disorder, defined as a persistent erection of the penis that continues more than four hours beyond, or is unrelated to, sexual stimulation. It is categorised to three different subtypes which are ischaemic priapism, non-ischaemic priapism, and stuttering priapism. A forty year old Caucasian male presented with priapism. The initial physical examination suggested ischaemic priapism. The diagnosis was later confirmed by the cavernous blood gases result. Detumescence was achieved by cavernosal irrigation, followed by insertion of cavernospongious shunt under general anaesthesia. The aetiology of priapism is mainly divided to primary (idiopathic) and secondary to underlying diseases. A correct diagnosis is vital in priapism because it may determine the most effective treatment. If treatment is absent or delayed, it can develop to permanent damage of the penis and/or erectile dysfunction.

Key words: priapism, ischaemic, non-ischaemic, stuttering.

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INTRODUCTION

Priapism is defined as a persistent erection of the penis that continues more than four hours beyond, or is unrelated to, sexual stimulation (1). The term is derived from Priapus, the Greek God of fertility. It typically involves only the corpora cavernosa and resulting in dorsal penile erection with the corpus spongiosum and glans penis remain soft and uninvolved (2). However, cases of priapism with involvement of the corpus spongiosum and sparing of the cavernosal spaces have been reported. It is relatively uncommon disorder with an incident rate of 0.5-0.9 cases per 100,000 person-years (1). Even though priapism may occur at all ages, priapism in children is extremely rare and is most commonly related to underlying conditions such as malignancy and haematological diseases (1).

Priapism is categorised to three different subtypes which are ischaemic (veno-occlusive, low flow) priapism, non-ischaemic (arterial, high flow) priapism, and stuttering (intermittent) priapism. Ischaemic priapism is the most common form of priapism which accounts for 95% of all priapism cases. It is usually painful and rigid erection, characterised by little or no cavernous blood flow resulting in abnormal (dark red) cavernous blood gases (hypoxia, hypercapnia, and acidosis) (3). Therefore, ischaemic priapism is considered a medical emergency. On the other hand, non-ischaemic priapism is caused by unregulated cavernous arterial inflow. This unregulated flow results in a persistent erection, usually neither fully rigid nor painful, and has been proposed to occur via a mechanism that involves stimulation of endothelial nitric oxide synthase by the turbulent blood flow (1). Unlike ischaemic priapism, the cavernous blood gases in non-ischaemic priapism are not hypoxic, hypercapnic, or acidotic. Lastly, stuttering priapism is described as repeated form of ischaemic priapism in which painful erections occur frequently with intervening periods of detumescence (diminution of the erection) (3). This type of priapism is uncommon and poorly understood.

CASE PRESENTATION

A forty year old Caucasian male presented to the Emergency Department (ED) at Dunedin Public Hospital with priapism. He suffers from complete paraplegia at T12 level from a motor vehicle accident in 1994. After the accident, he develops difficulty passing urine due to urethral false passage or stricture. Initially, a suprapubic catheter was installed as an ongoing treatment together with urethral dilation. The suprapubic catheter was later removed after successful self-catheterisations. He also has a history of bladder stones.

Initial physical examination suggested ischaemic priapism. There was no sign of pelvic, penile, or perineal trauma. Prior to the episode, the patient took neither any drugs nor any alcoholic drinks. Blood samples were sent to the laboratory for cavernous blood gases, full blood count, and renal function tests.

The cavernous blood gases (Table 1) showed hypoxia, hypercapnia, and acidosis which confirmed the diagnosis of ischaemic priapism. All routine haematology results were within the respective reference ranges with the exception of the total white blood cell count ($12.2 \times 10^9/L$ [$4.0 - 11.0 \times 10^9/L$]) and neutrophils ($9.8 \times 10^9/L$ [$1.9 - 7.5 \times 10^9/L$]), which suggested the present of inflammation or infection. There was no sign of haematological abnormalities such as a haemoglobinopathy or leukaemia. In addition, renal function tests were also within the reference range limits.

Table 1. Cavernous blood gases. Specimen type: cavernous blood. Oxygen therapy (L/min): on air.

	Result	Reference range
Blood pH	6.99	7.35 – 7.45
pCO ₂ (mmHg)	85	35 - 45
pO ₂ (mmHg)	12	30 - 40
HCO ₃ (mmol/L)	19.2	22.0 – 32.0
Base excess (mmol/L)	-19.2	-2.0 – 2.0
O ₂ Saturation (%)	12	-
Lactate (mmol/L)	13.7	0.5 – 2.2

Detumescence was achieved by cavernosal irrigation with 0.90% w/v saline, followed by insertion of cavernospongious shunt under general anaesthesia. The patient was discharged the following day with 1000 mg paracetamol, 500 mg amoxicillin and 125 mg clavulanic acid.

DISCUSSION

In general, priapism can be considered as an imbalance between arterial inflow and outflow which disturbs regulatory control in erectile function. The imbalance of arterial inflow and outflow may be caused by disturbances in the mechanism controlling penile detumescence and the maintenance of penile

flaccidity due to excess release of contractile neurotransmitter, malfunction of the intrinsic detumescence mechanism, obstruction of draining venules, or prolonged relaxation of intracavernosal smooth muscle (4). The aetiology of priapism is mainly categorised to primary (idiopathic) and secondary to other conditions or diseases. However, it is more appropriate to discuss the aetiology of priapism according to its specific subtypes.

Ischaemic priapism can be caused by many factors as shown in Table 2. Sickle cell disease is the most common cause of ischaemic priapism. It accounts for 23% of adult cases and 63% of childhood cases (1). In patient with sickle cell disease, the prevalence of priapism is up to 3.6% in patients < 18 years old, increasing up to 42% in patients ≥ 18 years old (1). The dysfunctional nitric oxide synthase and Rho-associated protein kinase (ROCK) signalling, and increased oxidative stress associated with NADPH oxidase mediated signalling are thought to be the mechanisms of sickle cell disease associated priapism (1). The second most common causes of ischaemic priapism are the intracavernosal injection of a vasoactive erectile agent and alcohol or drugs abuse. In contrast, non-ischaemic priapism is mostly associated with antecedent trauma such as blunt perineal, pelvic, or penile trauma. In a rare case, Fabry's disease can also cause non-ischaemic priapism due to unregulated high arterial inflow (5). The causes of stuttering priapism are usually idiopathic and in rare cases, a neurological disorder. In addition, a very rare condition called partial priapism or idiopathic partial thrombosis of penis can be caused by bicycle riding, trauma, drug usage, sexual intercourse, haematological diseases and α-blockers (1). It is a subtype of priapism limited to the crura of the penis.

Table 2. Aetiology of ischaemic priapism (references 1 and 3)

• Idiopathic
• Haematological abnormalities (sickle cell disease, thalassemia, leukaemia)
• Drugs (vasoactive agents, antidepressants, anticoagulant, recreational drugs, alcohol)
• Toxin-mediated infections (scorpion sting, spider bite, rabies)
• Metabolic disorder (Fabry's disease, amyloidosis)
• Neurogenic disorders (syphilis, spinal cord injury, brain tumour)
• Neoplasms (metastatic or regional infiltration)

The differential diagnosis of priapism can be done by physical examination, laboratory testing, and/or penile imaging. Ischaemic priapism is a compartment syndrome. The corpora cavernosa are rigid and tender, but the glans penis is soft. The patient usually complains of pain. Conversely, in non-ischaemic priapism, the corpora cavernosa are tumescent (beginning to swell or erect) but not fully rigid. The physical examination usually reveals pelvic, penile, or perineal trauma. In stuttering priapism, the erection is painful and the penis is rigid as in ischaemic priapism. The duration however, is usually shorter. Between erections, the penis is usually normal, but fibrosis can be found in some cases (1). The key findings in priapism is summarised in Table 3. During physical examination, it is important to take notes on important points such as duration of erection, degree of pain, previous episodes of priapism and treatments, current erectile function, medication including recreational drugs, history of pelvic, genital, or perineal trauma, and history of haematological abnormalities (e.g. sickle cell disease, leukaemia) (2,3).

Table 3. Key findings in the evaluation of priapism (references 1 and 3)

	Ischaemic priapism	Non-ischaemic priapism
Corpora cavernosa fully rigid	Usually	Seldom
Corpora cavernosa tumescence without full rigidity	Seldom	Usually
Penile pain	Usually	Seldom
Abnormal cavernous blood gases	Usually	Seldom
Haematological abnormalities	Usually	Seldom
Recent intracavernosal injection	Sometimes	Sometimes
Perineal trauma	Seldom	Usually

Laboratory testing should include full blood count (followed by haemoglobin electrophoresis if a haemoglobinopathy is suspected), coagulation profile, cavernous blood gases, and urine toxicology (1,3). Cavernous blood gases are essential tests to distinguish between ischaemic and non-ischaemic priapism. Typical cavernous blood gas values in priapism are shown in Table 4. In addition, colour Doppler ultrasonography of the penis and perineum can be performed as an alternative to cavernous blood gases. Penile arteriography can also be done to show the lack of blood flowing the cavernous artery in ischaemic priapism. It can also identify the presence and site of cavernous artery fistula (ruptured helicine artery) in non-ischaemic priapism (3).

Table 4. Typical blood gas values in priapism (references 1 and 3)

Source	pO ₂ (mmHg)	pCO ₂ (mmHg)	pH
Normal mixed venous blood (room air) – similar to normal flaccid penis	40	50	7.35
Ischaemic priapism (cavernous blood)	<30	>60	<7.25
Normal arterial blood (room air) – similar to non-ischaemic priapism	>90	<40	7.40

In general, the aim of priapism treatment is to return the penis to a flaccid and non-painful state, and therefore to avoid permanent damage to the corpora cavernosa (corporal fibrosis) and/or erectile dysfunction. However, conditions such as persistent penile oedema, ecchymosis, and partial erections can occur and mimic unresolved priapism (3). Acute ischaemic priapism is considered a medical emergency and therefore requires rapid intervention. In ischaemic priapism beyond 12 hours, corporal specimens show interstitial oedema, progressing to destruction of sinusoidal endothelium, exposure of the basement membrane, and thrombocyte adherence at 24 hours (1). At 48 hours, there is evidence of thrombi found in the sinusoidal spaces and smooth muscle necrosis with fibroblast-like cell transformation (1). Studies show that treatment beyond 48-72 hours may help to relieve erection and pain but have minimum benefit in preventing erectile dysfunction. The ischaemic priapism treatment is sequential. It should move on to the next stage if the last treatment fails (Table 5). In patients with underlying condition, it is vital that the treatment of ischaemic priapism is provided concurrently with appropriate treatment for the underlying disease.

Table 5. Treatment of ischaemic priapism (references 1 and 3)

1. Preliminary treatment <ul style="list-style-type: none">Local anaesthesia of the penisInsertion of wide bore butterfly (16-18G)Aspiration cavernosal blood until bright red arterial blood obtained
2. Cavernosal irrigation <ul style="list-style-type: none">Irrigation with 0.90% w/v saline solution
3. Intracavernosal treatment <ul style="list-style-type: none">Injection of sympathomimetic drugs (phenylephrine)
4. Surgical treatment <ul style="list-style-type: none">Surgical shunting<ul style="list-style-type: none">Distal shunts (Winter, Ebbehøj, and Al-Ghorab procedures)Cavernospongiosal (corporospongiosal) shuntCavernosaphenous (corporosaphenous) shuntPrimary penile implantation for priapism >36 hours

On the other hand, treatment of non-ischaemic priapism is not an emergency. It should start with conventional treatment such as applying ice to the perineum or site-specific perineal compression. It is followed by selective arterial embolisation, preferably using absorbable materials such as autologous blood clot and gelatin sponges, which are non-permanent (3). Surgical therapy is rarely performed unless there are contraindications for selective embolisation or embolisation failure.

Finally, the main goal in the management of stuttering priapism is to prevent the future episodes. It includes alpha-adrenergic agonists (pseudoephedrine) and hormonal therapy (GnRH, digoxin, and baclofen) (1,3). It is important to remember that hormonal agents interfere with normal sexual maturation and therefore should not be used in children who have not achieved full sexual maturation (3). Intracavernosal injection of phenylephrine should be considered in patients who either fail or reject the systemic treatment.

CONCLUSIONS

Priapism is a rare condition which can potentially develop to a medical emergency depending on its subtypes. While some aspects of priapism are still lacking, a better understanding of the pathophysiology, diagnosis, and treatment will benefit the patient's outcome. Therefore, guidelines on the assessment and management of priapism need to be followed consistently.

AUTHOR INFORMATION

Christian Christian, BMLSc MNZIMLS, Medical Laboratory Scientist

Biochemistry Department, Southern Community Laboratories, PO Box 6064, Dunedin, 9016.

Email: christian.christian@sclabs.co.nz.

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A forensic dilemma

Michael Legge

Department of Biochemistry, University of Otago, Dunedin

BACKGROUND

In 1993 a bizarre sequence of events unfolded in Europe when a serial killer was identified as being 'on the loose'. This vignette traces the events as they unfolded and the final forensic solution to the 'serial killer'.

A serial killer was believed to be responsible for at least six murders in Germany, which started in 1993. In addition, on the basis of DNA evidence, other murders in France were attributed to the serial killer. DNA from the serial killer was obtained from each scene of crime and was identified as a woman, which was considered unusual for a serial killer. In addition a discarded syringe and needle were found and the DNA matched that of the killer giving police to consider that the killer was also on drugs and that the killings were a result of drug taking. The independent profiler provided the police with information that the killer was compelled to kill to feed her drug habit and probably had a damaged home life and was abused.

Over a few years an identical DNA profile was associated with a series of minor offences but no match was achieved and the person remained unknown to the police. In 2007 two German police officers were shot while sitting in their car and traces of the identical DNA profile were identified from the car. Further killings occurred with the same DNA profile being found at the scene of crime.

In 2008 the charred body of a male illegal immigrant was found by the French police, and DNA that was used to identify the body matched the DNA profile of the alleged female serial killer. A second sample from the body gave a male DNA profile.

All the DNA samples were collected on cotton swabs that were manufactured by one German medical company and the source of the DNA was eventually identified as a female employee who had contaminated the swabs during their manufacture. The employee was never identified and the German and French police finally concluded that there was no mysterious female serial killer.

NEWS FROM THE UNIVERSITIES

AUCKLAND UNIVERSITY OF TECHNOLOGY



Bailey McIntosh a third year BMLS student specialising in Immunology and Biochemistry has been offered a summer scholarship from the Heart Research Institute (HRI). Thanks to her perseverance and hard work, Bailey brilliantly went through the interview process.

This is an outstanding opportunity for talented undergraduate students to develop their skills and gain valuable experience at state-of-the-art research facilities. High calibre scientists, new technologies and cutting edge equipment are just a few features that make this an exciting opportunity for promising AUT Undergraduate students.

The 8-week scholarship provides high-achieving and promising students the opportunity to work on a medical research project directly related to cardiovascular disease, expand on their skills and knowledge and be mentored in a world-class research institute. Scholarship recipients are required to write a report on their work experience and have the opportunity to make a presentation to their peers and supervisors at the end of the scholarship period. Scholarships take place over the Australian summer holidays for 8 weeks. Placements are full-time and typically begin in early December 2016 and finish end of January 2017.

Bailey worked at the Charles Perkins Centre at the University of Sydney with Dr John O'Sullivan whose focus is cardio metabolic diseases. His current work is centred around setting up metabolomics platforms that will give a broad coverage of all metabolic pathways. His projects range from young people with heart attacks to mouse models of obesity, diabetes and fatty liver.

During her summer scholarship, Bailey evidenced that metabolomic profiling can quickly demonstrate underlying risk in a cohort of young MI (myocardial infarction) patients that did not have cardiovascular risk established by traditional clinical chemistry. This deeper probing of cardiovascular risk could potentially re-classify patients whose risk was determined by traditional methods, and highlight a risk parameter that needs further attention. Clearly, an underlying diabetic risk in a young MI patient needs to be investigated further (fasting glucose, glycosylated haemoglobin, glucose tolerance testing), and appropriate measures taken to reduce this risk. Diabetes is also known as a "risk equivalent", and may well have been a primary driver of atherosclerosis in this young patient.

These findings highlight the clinical utility of plasma metabolomic profiling to provide a deeper investigation into a patients' underlying risk cardiovascular risk. Well-validated markers of diabetic risk such as branched-chain and aromatic amino acids provide additional information on potential underlying pre-diabetes, which can subsequently be addressed to reduce future risk.

MASSEY UNIVERSITY



Nicole Screech was the top third year BMLSc student so she has won the Massey NZIMLS Student Prize.

2016 NZIMLS Prize Winner –

My name is Nicole Screech. I became interested in a Bachelor of Medical Laboratory Science due to the wide range of diversity it offered me; not only throughout my study, but also in future career opportunities. Although for my fourth year placements I have chosen to specialise in diagnostic Microbiology and Haematology, I hope to one day expand my knowledge into other fields throughout medical laboratory science.



While still a full time BMLSc student, I have been working as a laboratory technician with Fonterra Brands, Palmerston North. Within this job I have gained a year's laboratory

experience and I have recently gained 6 months experience working for the quality department; working on various projects aiming to enhance the proficiency of both laboratory and site operations. Although I have enjoyed working in a food laboratory, I look forward to challenging myself further in my fourth year BMLSc placements in pathology laboratories.

UNIVERSITY OF OTAGO



2016 was a busy year, with a highly successful accreditation visit from the Medical Sciences Council of New Zealand. We welcomed our first credit transfer student from Malaysia, as part of our MOU with the International Medical University. She had a great semester and we hopeful for future collaborations. There have been some changes on our Board of Studies, with Dr Sean MacPherson replacing Professor David Murdoch and Sian Horan replacing Terry Taylor as the representative for the profession. We are grateful to David and Terry for their years of service and wish them well in their future endeavours. Thank-you also to our clinical partner laboratories across NZ and Australia for taking our 4th year students on placement. We look forward to working with you all again in 2017.

The BMLSc class of 2016 graduated on Saturday 10th December with a record number of students graduating with distinction or credit. They were:

BMLSc with distinction

Katelyn Brook
Ria Castillo
Catherine Dalley
Kayleigh Hancock
Harriet Kingston-Smith
Rachel Lamont
Summer Parslow
Hannah Reid
Summer Sanson
Monique Sinton
Felicia Tjandra
Yuyi Zhang
Vinci Lok

BMLSc with Credit

James Ferguson
Alistair Fisher
Mary Hampton
Stella Hlavac
Yulia Hwang
Rita James
Samantha Kerruish
Ashwin Kumar
Wilma Magorimbo
Stuart Offen
Bryn Pettersen
Jenny Too
Andrew Ung

Prize winners

Roche Diagnostics Haematology Prize:	Yuyi Zhang
AACB Biochemistry Prize:	Katelyn Brook
Professor Sandy Smith Prize in Medical Microbiology:	Hannah Reid
Colin Watts Prize for top student overall (over 3 years):	Katelyn Brook
Prince of Wales Nomination:	Katelyn Brook
NZIMLS Prize Top 4 th year student:	Hannah Reid
NZIMLS Prize Top 3 rd year student:	Sharon Ward
NZIMLS Prize Top 2 nd year student:	Hanlin Chen

Top graduates' interviews

Yuyi Zhang

What made you decide to become a medical laboratory scientist?

My decision to become a medical laboratory scientist was very much inspired by my parents, as I am the third generation of medical laboratory scientist in my family.

You are originally from Shanghai, China. What attracted you to Otago University?

I chose University of Otago for its stellar reputation within the health science teaching and research field. The four-year medical laboratory science degree provided me a good academic background and clinical experiences.



Which aspects of the course did you like best? Which aspects did you find most challenging?

The 15-week clinical placement was the aspect of the course that I liked the best and also found most challenging. It allowed me to learn how to apply the academic knowledge to real life diagnostic laboratory testing.

Your clinical placements were in Auckland and Nelson. What were they like?

I had my first placement in the haematology department at LabPlus, Auckland. It is a large laboratory from which I gained lots of experience with automation. I had my second placement in the microbiology department at Medlab South, Nelson. Compared to Labplus, it is a relatively small laboratory with less automation. However, it allows me to learn the basic theory in diagnostic methods by doing them manually.

What are you up to now?

I am currently working as a medical laboratory scientist in microbiology and haematology department at Medlab South.

What are your plans for the future?

I plan to study a master of epidemiology qualification in the UK to gain more experience that will enable me to improve the efficiency of services that the diagnostic laboratory provides.

Hannah Reid

What made you decide to become a medical laboratory scientist?

I liked the idea of being able to aid patients from a diagnostic perspective, by using various testing methods to effectively solve a puzzle. Additionally, understanding human disease was very intriguing to me.



You are originally from England. What attracted you to Otago University?

The range of courses, including the medical laboratory science course, was appealing to me, as was the colder weather in Dunedin, and how the University was such an integral part of the city.

Which aspects of the course did you like best? Which aspects did you find most challenging?

I enjoyed the practical aspects of the course, where we often tested our own samples, which gave perspective on what a working laboratory would be like. I found balancing the workload initially challenging, especially with part of the course being self-directed learning, but easily adapted to it as the course went on.

What do you like about medical laboratory science?

I like that the work is hands-on and patient based, and cases often vary from day to day. Medical laboratory science is constantly evolving, with new testing methods and concepts being discovered, which is an exciting prospect for furthering understanding of diseases.

Your clinical placements were both in Dunedin. What were they like?

My first placement was in Molecular Diagnostics at SCL Dunedin, where I was part of a great team. I had an amazing time learning about a variety of molecular tests both practically and in theory. My second placement was in Microbiology, which linked well with some concepts from my first placement. It was a change going from an automated to quite a manual department but was enjoyable and I learned many additional skills.

What are you up to now?

I'm currently residing in Christchurch and actively seeking employment in a medical laboratory.

What are your plans for the future?

To be employed in a medical laboratory or do post graduate study, preferably in the molecular genetics field.

Katelyn Brook

What made you decide to become a medical laboratory scientist?

I went to a health sciences career evening during my first year of university and talked with the students at the Medical Laboratory Science stand. I loved the sound of diagnosing all sorts of diseases and disorders, as well as its practical aspects.



You are originally from Christchurch. What attracted you to Otago University?

I was really attracted to the variety of health science courses the University of Otago had to offer. Choosing to do first year health sciences meant that I didn't have to decide which degree I wanted to do before I started my tertiary study.

Which aspects of the course did you like best? Which aspects did you find most challenging?

My favourite part was the small class sizes which provided a more one on one experience with each of the lecturers. I also enjoyed the laboratory practicals where we would test our own samples and find out our blood type, liver functions or INR. But this was also the most challenging part, as we had to lancet ourselves or perform venipuncture on each other to collect the blood.

What do you like about medical laboratory science?

I like that, as medical laboratory scientists, we provide a huge amount of information for the requestor which helps shape a patient's diagnosis and treatment. Medical laboratory science is also a challenging area which is continually developing.

Your clinical placements were in Adelaide and Christchurch. What were they like?

Molecular diagnostics in Adelaide was amazing as I got to experience a huge variety of diagnostic techniques and tests which are not yet routinely used in New Zealand. It was good to be back in my home city for my placement in Biochemistry at Canterbury SCL. Everybody was really nice and welcoming.

What are you up to now?

Currently I am employed at Canterbury Health Laboratories in the Cytogenetics department, working towards my full scientist registration. Most of my work involves fluorescent in situ hybridization (FISH) of cultured and uncultured cells, and prenatal diagnosis of amniotic fluid and chorionic villus samples.

What are your plans for the future?

I plan to complete my provisional registration and continue to advance my skills in Cytogenetics and Molecular Pathology.

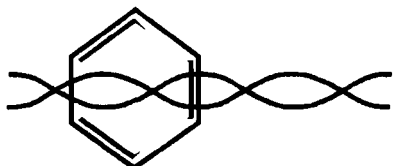
Biochemistry Special Interest Group

The 2016 Biochemistry SIG was held at the Ibis Tainui Hotel in Hamilton. The SIG was attended by approximately 45 delegates, with a diverse range of topics presented. Dr Stephen May, from Pathlab Waikato, presented an interesting topic on bone marrow examination, which provided insight to a technique that is seldom encountered by biochemistry staff.

Overall the day was very enjoyable and informative, with a mixture of case studies, informative topics and a dash of history to wrap things up. The NZIMLS best presenter was awarded to Claudia Sugrue, for her presentation on an IgE paraprotein and the runner up best presenter was awarded to Maria Carter for her presentation on Beer Potomania.

Thank you to all who attended and presented. I would also like to acknowledge the sponsors for the day; Roche, Biorad and Abbott- thank you for your support and also to the NZIMLS and AACB for providing the awards for the best presentations.

Kate McLaughlin
BSIG Convenor



NZIMLS Journal Prize



Council of the NZIMLS has approved an annual Journal prize to the value of NZ\$300 for the best article published in the Journal during the calendar year. The article can be a review article, original article, case study, research letter, or technical communication (excluded are Fellowship dissertations).

Many 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 presentation to the Journal. If accepted, you are in consideration for the NZIMLS Journal Prize and will also earn you valuable 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 the 1st author and a current financial member of the NZIMLS to be eligible. All articles accepted and published during the calendar year (April, August and November issues) will be considered. The Editor, Deputy Editor and the President of the NZIMLS will judge all eligible articles in December. Their decision will be final and no correspondence will be entered into.

The 2016 winner of the NZIMLS Journal Prize is Noah Sibanda from Middlemore Hospital, Auckland for his article: Helicobacter pylori infection and the platelet count.

Meet the NZIMLS Team

The NZIMLS Executive Office is staffed by Fran van Til (Executive Officer) and Sharon Tozer (Finance and Membership Administrator). Jillian Broadbent takes care of all your CPD requirements. The ladies contact details are:

fran@nzimls.org.nz	03 313 4761
sharon@nzimls.org.nz	03 313 4761
cpd@nzimls.org.nz	027 242 4712



(Left to right) Jillian, Sharon and Fran

Science Digest

Contributed by Michael Legge

Welcome to Science Digest, a look at some science and related news items in the last few weeks.

Three parent babies

The announcement that a Mexican IVF clinic has used donor mitochondria injected in to another woman's eggs has raised concerns about the ethics of the procedure and the eventual outcomes for the child born as a result of this procedure.

Gene editing

Recent research using the CRISPR-Cas9 gene editing technique in mice with sickle cell disease indicates that correcting the inherited defect in embryos raises the possibility for the use in humans in the future. An estimated 250,000 children are born every year with this inherited haemoglobin abnormality.

Genetic variant for high fat food

Researchers in the UK have identified that people with a variant in the melanocortin 4-receptor (MC4R) gene have an increased desire and do eat higher fat food than those without this gene variant.

Brain computer interface

A patient with late stage amyotrophic lateral sclerosis (ALS) had a fully implanted computer interface with electrodes to the motor cortex and a transmitter placed in the left thorax. By moving the left hand the patient controlled the computer typing programme at the equivalent of two letters per minute, which at times proved better than an eye tracking device.

Possible new antimalarial target

Plasmodium spp have complex life cycles, which limit the use of antimalarial strategies. Researchers have screened 100,000 compounds to identify novel drug interactions and have identified a series of compounds, bicyclic azetidines that target the enzyme phenylalanine-tRNA synthase. This group of compounds had activity against all *Plasmodium spp* parasite life stages in the mouse models investigated.

Can diet tailor cancer treatment?

Recent research using mice has demonstrated that high fat-low carbohydrate diets boosted blood acetoacetate concentrations, which enhanced tumour growth. Treatment whereby acetoacetate was reduced, slowed tumour growth, this raises the question of whether tailoring diets can slow cancer progression?

Zika virus conundrum

From a study in Brazil, 55% of first trimester pregnancies had adverse outcomes, 52% in the second trimester and 29% in the third trimester respectively following infection with Zika virus. Adverse pregnancy outcomes were miscarriages, calcification of areas of the babies' brain, small for dates, brain haemorrhage, and 3.4% had microcephaly. A second study of pregnant women in the USA who had been infected with Zika virus revealed that only 6% had Zika associated defects, but the rates of microcephaly remained the same as those in Brazil. Currently no explanation can be provided for the 46% and 6% differences between the two studies. As the Zika virus starts spreading through Asia, Australia, and the South Pacific, researchers urge pregnant women to protect themselves against Zika virus infections.

23andMe decide against next generation sequencing

The intention to launch the use of next generation sequencing (NGS) for public use has been halted by 23andMe.

The decision was based on the complexity of the information and results that may be too vague to understand. The intention was to launch saliva based testing using a NGS testing system, but the company became concerned about the possibility of the ambiguity of the results. Other commercial DNA sequencing companies, such as Color Genomics, Helix, and VeritasGenomics are intending to progress commercial NGS to the public.

Faulty heart genes put over 600,000 people at risk

The British Heart Foundation estimates that many of the unexplained deaths in the UK could be due to undiscovered faulty genes affecting heart function. On average 12 people under the age of 35 years per week die from sudden unexplained cardiac arrest. Although many of the genes are still not known an emerging pattern is that of the inherited heart conditions, a substantial proportion have a 50% chance of inheriting the condition (autosomal dominant), which results in sudden death by cardiac arrest. An example is that a gene variant of the muscle protein titin is associated with enlarged hearts and heart failure.

Periodontal infection linked to autoimmunity in rheumatoid arthritis

For some time there has been consideration that there is a link between rheumatoid arthritis and bacterial infections. A connection between *Aggregatibacter actinomycetemcomitans* (*Aa*) and rheumatoid arthritis has been identified. The periodontal pathogen *Aa* has been shown to induce changes in neutrophil function, which included hypercitrullination of host proteins, an observation identified in rheumatoid arthritis patients. Additional evidence indicates that HLA-DRB1 (associated with rheumatoid arthritis) was only found in patients exposed to *Aa*. The exposure to the periodontal pathogen *Aa* is considered to be a bacterial trigger of autoimmunity rheumatoid arthritis.

Safety of e-cigarettes

Researchers in the USA have identified that the use of e-cigarettes increased the risk of heart disease. Users of e-cigarette had elevated levels of epinephrine and increased oxidative stress, two factors associated with increased heart disease and arterial hardening. Additional research has linked e-cigarette use to lung inflammation, which has been linked to the presence of acrolein, acetaldehyde and formaldehyde in the vapours.

Diphtheria antitoxin in short supply

Two children are known to have died in Europe due to a shortage of the antitoxin to the toxin produced by *Corynebacterium diphtheriae*. Declining vaccination rates in certain countries raise the fear that the antitoxin may not be readily available for treatment. The antitoxin is produced using horses and the market is too small to make the production profitable. Although the antitoxin is produced in Russia, India, and Brazil, there are concerns relating to quality and safety. Researchers are currently investigating the production of the antitoxin in cell culture systems but financial backing is problematic.

Influence of Factor X1 in arterial hypertension

Interactions of platelets, leukocytes and the blood vessel wall support coagulation and precipitate arterial and venous thrombosis. Recent research has shown that vascular disease and hypertension can be driven by overactive thrombin-driven factor X1 feedback loop by platelets. This was associated with a coagulation- inflammation circuit promoting vascular dysfunction and hypertension. Inhibition of factor X1 reduced blood pressure and reversed vascular dysfunction.



A Weekend in Beautiful Bay of Islands



NZIMLS Presents the North Island Seminar 2017 “Medical Laboratory Science”

DATE: Saturday 6th May 2017

VENUE: Copthorne Hotel and Resort Paihia (Bay of Islands)



After the seminar, enjoy the weekend or few extra days exploring Bay of Islands by visiting Waitangi Treaty Grounds; take a boat trip to “Hole in the Rock” & Dolphin/Whale watch; visit Haruru Falls; Parasailing; Fishing; Kerikeri Sunday market; playing golf at Waitangi; or just exploring Paihia and relaxing on the beach.

Presentations Invited. Please Contact:

Sailesh Singh

Sailesh.Singh@northlanddhb.org.nz

Greg Elias

Greg.Elias@northlanddhb.org.nz

Presentations invited for:

Phlebotomy, Specimen Services, Molecular Diagnostics, Biochemistry, Haematology, Immunology, Transfusion Medicine, Anatomical Pathology, Cytology, Microbiology, Automation, Point of Care, Quality Management, Laboratory Management, LIS, Case Studies.

Prizes for Best Presentation and Runner-up Best Presentation

Registration for the North Island Seminar is only available online at www.nzimls.org.nz



INNOVATION!

Leading the way...

Pathlab in the Bay.

MSIG 2017

Saturday 10th June

Clinical School, Tauranga Hospital campus

Microbiology with

Space age technology

Is the new

Generation

Register online at: www.nzimls.org.nz

Drinks, Nibbles & Laboratory Tour

Friday evening 9th June

Pathlab, Tauranga Hospital Campus

Presentations Welcome! contact: Jo Madden
jo.madden@pathlab.co.nz



BIOCHEMISTRY SIG SEMINAR

Tauranga Yacht & Power Boat Club

Saturday 1 July 2017

Presentations Welcome

For further information and to register interest for presentations, please contact:

maria.carter@pathlab.co.nz

Registration will be available at www.nzimls.org.nz



Rutherford Hotel August 22 - 25

LIGHTING THE WAY



2017 Nelson

NZIMLS Annual Scientific Meeting



MOLECULAR DIAGNOSTICS SIG SEMINAR 2017

FRIDAY

13 OCTOBER

COMMODORE HOTEL
MEMORIAL AVENUE, CHRISTCHURCH

Molecular Diagnostics is the field of the future, hear what's happening now within the disciplines of Molecular Genetics, Molecular Virology and Microbiology, Molecular Haematology and Cytogenetics!

Closing date for Abstracts: Friday 8 September

Contact: kevin.barratt@cdhb.health.nz



Register online at www.nzimls.org.nz

THE Pacific WAY

Blood bank in 2016

A blood bank course was provided by the PPTC in November 2016 at its centre in Wellington, and the following three students attended: Michael Ala from Vanuatu, Catherine Gootinngin from Yap, and Ianeta Tewaaki from Kiribati.



The PPTC wishes to especially thank Chris Kendrick for making the transfusion course possible last year. Chris was fantastic in helping the PPTC deliver this course, and the students very much appreciated and respected the contribution he made as their lecturer and mentor. Thank you also to NZ Red Cross and the Norman Kirk Trust for awarding a scholarship to Michael Ala (Santo, Vanuatu) in order to attend this course.

Pacific Paramedical Training centre training courses 2017

Haematology and blood cell morphology	24/04/17 - 19/05/17
Biochemistry	19/06/17 - 14/07/17
Laboratory quality management systems	07/08/17 - 01/09/17
Microbiology	18/09/17 - 13/10/17
Blood transfusion science	30/10/17 - 24/11/17

For further information contact: Navin Karan, Programme Manager, PO Box 7013 Wellington, New Zealand.
Telephone: +64 4 389 6294.
Email: pptc@pptc.org.nz or navink@pptc.org.nz. Website: www.pptc.org.nz

Second Regional Forum of WHO collaborating centers in the Western Pacific, 28-29 November 2016, Manila, Philippines

In 2014, WHO hosted its 1st Collaborating Centre forum in Manila the objectives of which were:

- Provide an overview of WHO current priorities and future initiatives.
- Identify innovative opportunities and means for strengthening and enhancing partnerships between WHO collaborating centres and WHO.
- Share good practices and success stories of effective collaboration for scaling-up and wider adoption.

The 2nd WHO Collaborating Forum meeting which was held in Manila, Philippines between the 28th - 29th November 2016 built on the gains of the first forum by further developing inter-professional approaches to collaboration and partnerships to help countries meet the sustainable development goals. 260 delegates were invited to attend and Phil Wakem and Navin Karan who represented the PPTC were asked to provide a presentation on distance learning through POLHN (Pacific Open Learning Health Net) which was well received.

United Nations development programme

The Western Pacific Multi-Country Integrated HIV/TB Programme funded by the Global Fund to Fight AIDS, Tuberculosis and Malaria and implemented by the United Nations Development Programme (UNDP) provides support to improve and maintain quality of TB laboratory services in PICTs through external quality assessment (EQA). EQA is understood as a combination of three major elements:

- On-site (in-country) evaluations.
- Blinded smear rechecking.
- Panel tests.

A proposal submitted to UNDP by the PPTC that would provide TB surveillance through on site evaluations in eight countries was accepted as a contract for delivery in February – March of this year. The countries covered by this proposal include: Tonga, Kiribati, Nauru, Niue, Samoa, Cooks, Tuvalu, and Vanuatu.

As per the UNDP contract for external quality assessment in-country evaluation visits, the terms of reference (TOR's) were as follows:

- To review and assess existing laboratory facilities.
- To assess the current staffing situation, as well as knowledge and qualifications of laboratory personnel.
- To review specimen processing and use of safe working practices.
- To review each laboratory's consumables and reagents inventory, processes for ordering and receiving of supplies.
- To review preparation and storage of in-house prepared reagents.



World Health Organization
Western Pacific Region
Second Regional Forum of WHO Collaborating Centres in the Western Pacific
28-29 November 2016 • Manila, Philippines

- To review management of infectious and laboratory waste.
- To review present EQA principles and advise revisions as per the newer recommendations if necessary.
- To discuss ways and means of increasing sample numbers tested in the laboratory.
- To provide immediate advice and on-site coaching whenever possible in the areas where gaps were identified.
- To prepare the evaluation report and action plan to address any quality issues identified during each visit.
- To work with the National TB Control Program team to strengthen communication and laboratory liaisons.

This programme, which is one of process surveillance and assessment in terms of TB laboratory operation, is currently being carried out by Russell Cole and Navin Karan and is scheduled to be completed by the end of March.

Antimicrobial resistance

In 2016, WHO granted funding to the PPTC to deliver an antimicrobial resistance surveillance and control programme to four selected countries, the purpose of which was to:

- Build capacity for accurate and reliable antimicrobial testing and reporting amongst the Pacific region laboratories, monitored by the PPTC microbiology EQA results.
- Establish standardised antimicrobial testing methods, essential laboratory supplies, and standards of practice. i.e. CLSI guidelines/ EUCAST guidelines across the region.
- Facilitate the development of laboratory antibiotic testing regimes in accordance with recommended treatment guidelines and local pharmacy availability in Pacific Island countries where no national guidelines exist or antibiotic policies are in place.
- Strengthen laboratory-based antimicrobial resistance data collection and analysis for selected bacterial pathogens and the dissemination of that information to appropriate agencies.

Russell Cole, Navin Karan and John Elliot, as PPTC microbiology consultants, successfully provided:

- Four facilitating visits to the selected countries of Fiji, Cook Islands, Kiribati, and the Marshall Islands to teach and train laboratory staff in antimicrobial testing procedures and interpretation.
- Standardisation and verification of antibiotic susceptibility data for selected organisms of concern. i.e. ESBL's metallo-beta lactamases, MRSA, VRE Enterococci.
- Laboratory consumables required for quality control organisms, regular QC scheduled cycles, and microbiology antimicrobial testing items such as agar plates and antibiotic discs.
- A review of microbiology standard operating procedures and associated quality manual documentation relating to antibiotic testing procedures, quality control, reporting comments, and susceptibility data collection.

Thank you to the NZIMLS

The NZIMLS has generously contributed once again to the PPTC's regional external quality assurance programme. As the principal teaching and training institution in the medical laboratory sciences for the Pacific region, it is extremely

important that the PPTC maintains and supports Pacific Island laboratories through its continued physical presence. This grant will assist the PPTC in the monitoring and evaluation of a Pacific Island laboratory that is currently a registered participant in our REQA programme. This contribution will enable the PPTC to monitor REQA performance more effectively as well as troubleshoot on site any issues that this selected laboratory is challenged with. For this year, 2017, the PPTC will invest in Papua New Guinea for PPTC on-site performance evaluation. Papua New Guinea has never in previous years had PPTC on-site support with reference to REQA monitoring and evaluation, and so a special thank you goes to the NZIMLS for their support in creating this opportunity.

Welcome to our two new PPTC Board of Governance members



Angela Brounts is a registered medical laboratory scientist who started her training in Christchurch, specialising in haematology. She travelled extensively around NZ with her husband who was in the RNZAF and gained extra qualifications in biochemistry while working in laboratories in Auckland, Palmerston North, Christchurch, and more recently Wellington. Now a

Wellingtonian by choice she completed a BSc in chemistry and technology in 2003, and an MBA in 2009. From 2000 to 2009 she was the Core Laboratory Co-ordinator at Capital and Coast DHB; a role that introduced her to the PPTC and enabled her to provide support by facilitating her staff helping with training of Pacific students. She has continued to support the PPTC in her most recent position as the Laboratory Operations Manager at the Institute of Environmental Science and Research (ESR) and recently gave a series of lectures on management topics. She has also been part of Pacific PaceNet Plus meetings, and facilitates ESR providing arbovirus confirmation testing to the Pacific Island communities in collaboration with SPC and WHO.



Marion Clark has extensive governance, management, and public policy experience, in New Zealand and Australia and has worked effectively with a range of stakeholders to achieve effective and workable solutions to identified issues. She has worked in senior public policy roles in the Ministry of Health, Ministry of Foreign Affairs and Trade, Australian Health

Ministers Council, and Queensland Health, and with health officials in the Pacific and South East Asia. She is an experienced leader with excellent relationship management and communication skills. The PPTC is most fortunate to have Marion as a member of its Board of Governance.

Journal Questionnaire

Below are ten questions based on articles from the April 2017 issue. Read the articles carefully as 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. It is recommended that you write your answers in a word document and then cut and paste your answers on the web site.

The site has been developed for use with Microsoft's Internet Explorer web browser. If you are having problems submitting your questionnaire and you are using the Firefox web browser, try re-submitting using Microsoft's Internet Explorer.

You are reminded that to claim valid CPD points for successfully completing the journal questionnaire you must submit an individual entry. It must not be part of a consultative or group process. **In addition, members who have successfully completed the journal questionnaire cannot then claim additional CPD points for reading the articles from which the questions were derived.**

The site will remain open until Friday 7th July, 2017. You must get a minimum of eight questions right to obtain five CPD points.

The Editor sets the questions but the CPD Co-ordinator, Jillian Broadbent, marks the answers. Please direct any queries to her at cpd@nzimls.org.nz.

APRIL 2017 JOURNAL QUESTIONNAIRE

1. What is point 9 of the NZIMLS Code of Ethics.
2. What should members do with the returned answers to their journal questionnaire submissions, and why?
3. What is thought to be the mechanisms of sickle cell disease associated priapism?
4. What are the typical cavernous blood gas values in ischaemic priapism?
5. Define priapism, and where is the term derived from.
6. What is antithrombin, and how does it act?
7. What are antithrombin deficient patients at increased risk of?
8. Inherited antithrombin deficiency Type IIb alters what and results in what?
9. Name the antithrombin heparin binding site methods that have been used.
10. Recent research has shown that vascular disease and hypertension are driven by what, and what was this associated with.

NOVEMBER 2016 JOURNAL QUESTIONNAIRE ANSWERS

1. At Aotea Pathology, what was their initial protocol for the cultivation and isolation of *Salmonella* and *Shigella* from clinical faecal specimens?
Directly plating faeces onto Hektoen enteric agar (half plate) and sub-culturing onto Xylose Lysine Deoxychocolate agar following enrichment in Selenite F broth after overnight incubation.
2. Both chronic and acute immune thrombocytopenia are caused by what?
By increased platelet destruction in which anti-platelet antibodies sensitise circulating platelets marking them for removal by splenic macrophages bearing Fcγ receptors.
3. *Helicobacter pylori* is what type of bacteria?
Fastidious, microaerophilic, helical shaped Gram-negative bacteria.
4. Co-carriage of other resistant mechanisms in Enterobacteriaceae and non-glucose-fermenting Gram-negative bacilli isolates can result in what?
Compromised treatment options, and high mortality rates in patients with severe infections.
5. What was the current screening protocol at Canterbury Health Laboratories for the detection of carbapenemase producing organisms in routine faecal screening samples?
CHROMagar™ ESBL and MacConkey agar with a 10 µg meropenem disc.
6. Preparation of re-suspended red cells for transfusion involves what processes?
Leucocyte depletion by high-efficiency filtration in an adsorption depth filter, followed by a centrifugation step and separation of the red cells and plasma. The red cells are then resuspended in saline, adenine, glucose and mannitol additive solution with only a minimal volume of plasma.
7. What are the principal constituents of aggregated material in unfiltered units of blood?
Platelets, granulocytes and monocytes, together with small amounts of fibrin.
8. What concerns exist over the association of aggregated material with febrile transfusion reactions?
Transmission of cell-associated cytomegalovirus, immunisation by transfused leucocytes resulting in HLA antibodies and refractoriness to platelet transfusion, post-transfusion thrombocytopenia, immunomodulation and potentially other adverse effects.
9. Juvenile myelomonocytic leukemia is characterised by what?
Overproduction of granulocytic and monocytic cells that can infiltrate organs including the spleen, liver, gastrointestinal tract and lung.
10. What non-specific features on presentation of juvenile myelomonocytic leukemia are typically included?
Fever, splenomegaly, cough, rash and a failure to thrive.

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Questions we've been asked

NZIMLS CPD PROGRAMME

Is there a booklet about the CPD programme?

Yes, booklet downloads are available in the Scientist and Technician programme section under the CPD tab.

Can/should I claim more (or less) than 60 points for category 1 of the CPD programme?

This is a compulsory section in the Scientist CPD programme. The maximum (and minimum) points claimed under this section in any year is 60. In the audit process it was brought to our notice that some were confused about this section. Some practitioners claimed no points, some less than 60 and even some more than 60. Irrespective of the hours you work and the number of departments of the lab you work in, the maximum allowable claim/year is 60 points, and you are either competent or not.

How can I get my points if I am a part-timer or shift-worker?

The same target of 100 points per year applies to both full and part-time staff employed as scientists. Each person must undergo a yearly peer review of their laboratory competencies irrespective of the hours worked. This exercise is worth 60 points and the remaining 40 points can be gained from attendance at seminars, journal clubs, SIG meetings, or through one of the other options outlined in the Scientist's CPD programme booklet. Please refer to the "CPD programme for Technicians" booklet for more details on what is required for Technicians.

What happens to my CPD points requirement if I am on maternity leave?

You should inform the Medical Sciences Council that you will be off work for part/all of the year and apply for an exemption next time you apply for an APC.

Do I need CPD points for the part year in which I will be away on maternity leave?

This depends on timing but if you work any part of a year for which you will require an APC then you will be required to have a minimum of 60 points for your Laboratory Competency. In addition you should also accumulate points for professional development for the part of the year in which you were working.

I am a laboratory or section manager within a laboratory. Do I need to join the CPD programme?

If you practice the profession or perform tasks that are classified as MLS then you must hold an APC. If you answer yes to any of these then you are required to participate in a competency programme such as the NZIMLS CPD programme.

How will the yearly audit process work and will I be audited?

Approx 10% of all participants will be audited yearly. This will occur at the end of each year and if selected you will be required to provide documentation to support your CPD points record. Failure to cooperate with the auditors or failure to provide adequate evidence to support your claimed CPD total could result in the MSC refusing to issue you an annual practising certificate (APC) for the upcoming year. The CPD booklet states the minimum requirements of the audit process for both Scientists & Technicians.

Are all scientific meetings approved for CPD points?

Most NZ scientific meetings are approved as CPD meetings. This process relies upon organisers seeking CPD approval for their meetings from the NZIMLS before the meeting. Where a meeting has been approved, the NZIMLS provides the Conference/Seminar organisers with a meeting code for inclusion on certificates of attendance issued to those present at the meeting. Notes or a report must also be kept as part of the audit process.

If I attend a meeting that has not been pre-approved as a CPD event, can I still claim CPD points?

If the meeting has relevance to your area of work, then points can be claimed. The claim allowed under these circumstances is 2 points per hour which is pro-rata to the length of time of the meeting. The maximum daily claim for attendance at a conference or seminar is 16 points. Check the CPD booklet for documentation that is to be kept.

Is the CPD programme available for Medical Laboratory Technicians?

The NZIMLS offers a CPD programme for Technicians. The annual APC application with the Medical Sciences Council requires Technicians to undertake a minimum of 8 hours (16 points) of professional development yearly in order to obtain an APC.

I have recently attended a course on occupational safety and health in the laboratory. Can I claim CPD points for this?

CPD points for courses such as OSH, people management (eg. train the trainer), dangerous goods handling, assertiveness training, etc. are not specified in the CPD programme booklet. While these may improve "people management" or "laboratory safety skills", they should not detract from the prime goal of attaining 40 CPD points per year, for MLS related activities. Claim 2 points per course (max. 10 points per year), with claims made under the "Other" section of the programme. This does not include Health and Safety meetings, First Aid, Fire Training, Hand-washing/Hygiene sessions, which are all part of your compulsory competence requirements.

Do I get CPD points for the provision of student training?

The supervision/teaching/training of either BMLSc students or staff training toward Technician qualifications, do not qualify for CPD points. This is because this activity is not considered development or ongoing learning for yourself, as you are passing on information used as part of your day to day job. For this you receive 60 points per annum i.e. annual competency. Person(s) receiving training (e.g. someone working towards a QMLT) may be eligible to claim CPD points if they are required to collect professional development points for their APC.

NZIMLS CPD COORDINATOR



Jillian Broadbent is available for any CPD queries. More information on the NZIMLS CPD Programme is available on the NZIMLS website at www.nzimls.org.nz

Jillian may be contacted at cpd@nzimls.org.nz

Fellowship of the NZIMLS

The NZIMLS encourages members to consider Fellowship as an option for advancing their knowledge and career prospects. Fellowship provides an attractive option to academic postgraduate qualifications at a fraction of the cost.

Recently, changes to the regulations have been made, the main one doing away with the examination route to Fellowship.

Fellowship of the NZIMLS may be gained by thesis, by peer reviewed publications; or by treatise in case of a member holding an appropriate postgraduate or professional qualification.

Fellows may use the nominals FNZIMLS if a current financial member of the Institute.

Thesis

The thesis must be based on the style of Master of Science by thesis requirement of New Zealand universities.

Publications

A minimum of ten peer reviewed publications in international or discipline acknowledged biomedical journals. The candidate must be the 1st or senior author of at least six of these publications. A comprehensive review of the submitted publications is also required.

Treatise

By submission of a treatise in the form of a dissertation of 3000 - 5000 words on a medical laboratory science subject. The dissertation may take the form of a review, a scientific study, development of a hypothesis, or any other presentation that meets with the approval of the Fellowship Committee.

Candidates considering Fellowship by this route must be holders of at least a Master's degree in medical laboratory science or a closely related subject; or have a professional qualification such as Fellowship of the following professional bodies: the Australian Institute of Medical Science (FAIMS); the Institute of Biomedical Science (FIBMS); the Faculty of Science of the RCPA, the Australian Association of Clinical Biochemists (FAACB), or the Royal Institute of Biology, London (FRSB).

For full Fellowship regulations and the application process visit the NZIMLS website at www.nzimls.org.nz

Current Financial Fellows

Jenny Bennett
Jillian Broadbent
Jennifer Castle
Marilyn Eales (Life Member)
Christine Hickton
Michael Legge (Life Member)
Christine Leaver
Ron Mackenzie (Life Member)
Howard Potter
Maxine Reed
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Robert Siebers (Life Member)
Andrew Stewart
Vanessa Thomson
Vasanthan Thuraisamy
Emil Wasef
Jacqueline Wright
Rubee Yee
Sheryl Young

Mark Bevan
Ailsa Bunker
Jan Deroles-Main
Susan Evans
Sheryl Khull



Barrie Edwards & Rod Kennedy Scholarship

The Barrie Edwards & Rod Kennedy scholarship is one of the most significant awards offered by the NZIMLS. The scholarship provides the winner with support to attend an international or national scientific meeting up to a maximum value of \$7,500.



Barrie Edwards

Application for this prestigious scholarship is invited from Fellows, Members and Associate Members of the NZIMLS. Applicants must be a current financial member of the NZIMLS and have been a financial member for at least two concurrent years prior to application. To be eligible applicants must make an oral presentation or present a poster as 1st author at their nominated scientific meeting.



Rod Kennedy

All applications will be considered by a panel consisting of the President and Vice-President of the NZIMLS and the Editor of the New Zealand Journal of Medical Laboratory Science (who are ineligible to apply for the scholarships). The applications will be judged on your professional and academic abilities together with your participation in the profession. The panel's decision is final and no correspondence will be entered into.

Application is by letter. Please address all correspondence to:
NZIMLS Executive Officer
PO Box 505
Rangiora 7440

There is one scholarship awarded in each calendar year. Closing date is December 20th in any given year.

In your application letter please provide the following details:

- Full name, position, work address, email address and contact phone number
- The length of time you have been a financial member of the NZIMLS
- The conference you wish to attend – please provide dates
- A budget comprising airfares, conference registration and accommodation costs
- The abstract of your intended oral or poster presentation and whether it has been accepted for presentation (proof required)
- Your intentions to publish your results
- State briefly your history of participation in the profession over the last 5 years
- State the reasons why you wish to attend your nominated scientific meeting

Successful applicants will be required to provide a full written report on return which will be published in the Journal. If not intended to publish elsewhere, successful applicants will be required to submit their study results for consideration by the New Zealand Journal of Medical Laboratory Science .

2017 NZIMLS CALENDAR

(Dates may be subject to change)

DATE	COUNCIL	CONTACT
4-5 May	Council Meeting, Bay of Islands	fran@nzimls.org.nz
21 & 22 August	Council Meeting, Nelson	fran@nzimls.org.nz
December 2017	Council Meeting	fran@nzimls.org.nz
DATE	SEMINARS	CONTACT
6 May	North Island Seminar, Copthorne Hotel, Bay of Islands	sailesh.singh@northlanddhb.org.nz
19-21 May	NICE Weekend, Wairakei	raewyn.cameron@lsr.net.nz
10 June	Microbiology SIG Seminar	murray.robinson@pathlab.co.nz
1 July	Biochemistry SIG Seminar, Tauranga Yacht & Power Board Club	maria.carter@pathlab.co.nz
September	Immunology SIG Seminar	
October	PreAnalytical SIG Seminar, Auckland	
13 October	Molecular Diagnostics SIG Seminar, Commodore Hotel, Christchurch	kevin.barratt@cdhb.health.nz
14 October	Haematology SIG Seminar, Sudima Hotel, Christchurch	
28 October	Anatomical Pathology SIG Seminar, Copthorne Hotel, Queenstown	
November	Mortuary SIG Seminar	
DATE	CONFERENCE	CONTACT
22-25 August	Annual Scientific Meeting, Rutherford Hotel, Nelson	tony.barnett @nmhs.govt.nz fran@nzimls.org.nz
DATE	MEMBERSHIP INFORMATION	CONTACT
28 January	Membership and CPD enrolment due for renewal by 28 February 2017	sharon@nzimls.org.nz
31 January	CPD points for 2016 to be entered before 31 January 2017	cpd@nzimls.org.nz
15 February	Material for the April issue of the Journal must be with the Editor	rob.siebers@otago.ac.nz
15 June	Material for the August Journal must be with the Editor	rob.siebers@otago.ac.nz
23 June	Nomination forms for election of Officers and Remits to be with the Membership (60 days prior to AGM)	fran@nzimls.org.nz
13 July	Nominations close for election of officers (40 days prior to AGM)	fran@nzimls.org.nz
2 August	Ballot papers to be with the membership (21 days prior to AGM)	fran@nzimls.org.nz
10 August	Annual Reports and Balance Sheet to be with the membership (14 days prior to AGM)	sharon@nzimls.org.nz
17 August	Ballot papers and proxies to be with the Executive Officer (7 days prior to AGM)	fran@nzimls.org.nz
15 September	Material for the November Journal must be with the Editor	rob.siebers@otago.ac.nz
DATE	NZIMLS EXAMINATIONS	CONTACT
04 November 2017	QMLT Examinations	fran@nzimls.org.nz

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CORPORATE HEADQUARTERS

904 Caribbean Drive
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PHONE +1.408.541.4191
FAX +1.408.541.4192

CEPHEID AUSTRALIA/NEW ZEALAND

PHONE (AUSTRALIA) 1800.107.884
PHONE (NEW ZEALAND) 0800.001.028
EMAIL CepheidANZ@cepheid.com

www.Cepheidinternational.com

