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New Zealand Institute of Medical Laboratory Science

The Barrie Edwards and Rod Kennedy Scholarships

Applications are invited for these prestigious scholarships. These scholarships are some of the most significant awards from the New Zealand Institute of Medical Laboratory Science (NZIMLS). The two scholarships each year provides the winners support to attend an international or national scientific meeting to a maximum value of $7,500.

Applications are 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. Applicants must intend to present orally or a poster presentation as 1st author at their nominated scientific meeting.

Applications will be judged 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 active participation in the profession. Their decision is final and no correspondence will be entered into.

Application is by letter to be addressed to the Executive Officer of the NZIMLS, PO Box 505, Rangiora. There will be two scholarships awarded in each calendar year with **closing dates of June 30th and December 20th**.

In your application letter provide details of:

- Your full name, position and work address, email address and contact phone number
- How long you have been a financial member of the NZIMLS
- The conference you wish to attend complete with dates
- A budget comprising airfares, conference registration and accommodation costs
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- Your intentions to publish your results
- State briefly what your active participation in the profession is and has been in the last 5 years
- State the reasons why you wish to attend your nominated scientific meeting

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

In this issue

In this issue

In an Editorial, Lorraine Ferris and Robert Fletcher discuss the World Association of Medical Editors (WAME) new policy on conflicts of interest in peer-reviewed journals. As they state in their opening paragraph *"Conflict of interest in medical publishing exists when a participant's private interests compete with his or her responsibilities to the scientific community, readers, and society"*. As our Journal, through its Editors, are members of WAME, submitting authors are now required to disclose potential conflicts of interest, such as being funded by medical companies to perform the study or conference assistance. These disclosures will be published with the accepted article.

John Waldon from the Research Centre for Maori Health and Development at Massey University was a keynote speaker at the NZIMLS Annual Scientific Meeting in Blenheim in August 2009. His address is presented in the Journal as a Viewpoint Article where he reflect on how matters of handling of the Maori person, the processing of their bodily samples, and their correct disposal can be approached, with some history and illustrative experiences.

Pleural fluid adenosine deaminase (ADA) levels are used in the diagnosis of tuberculous pleural effusion (TPE). In their article, David Song and colleagues evaluated the Diazyme ADA assay on a commercial analyzer and present their clinical experiences of pleural fluid ADA over a 6 month period in a New Zealand population. They conclude that there are differences in ADA results when compared to an alternative method and advise caution when literature based cut-offs are applied to the Diazyme ADA method.

Robyn Barnett and Bronwyn Kendrick present an interesting case study of HELLP syndrome in a 40 year old post-partum woman who had evidence of pre-eclampsia in the weeks leading up to delivery. Within 24 hours of delivery the patient's blood pressure was rising significantly, she experienced upper epigastric pain and vomiting, her platelet count was dropping rapidly and blood tests revealed that her liver enzymes were becoming increasingly abnormal. All of these symptoms led to the rapid diagnosis of HELLP syndrome.

Barbara Mohn evaluated the Nova StatStrip blood glucose for precision, accuracy, and interferences from haematocrit and maltose and also compared it against three other meters and two reference methods. From her study results she concluded that the StatStrip glucose meter did not show clinically significant interference from maltose or varying haematocrit levels and demonstrated the best correlation with the reference glucose method.

An Educational Article describes how to write up a laboratory-based case study for the Journal. Case studies are educational for the reader as they bring together laboratory results with the patient's clinical diagnosis.

In this issue there are also four book reviews and a Letter to the Editor on a recently published article on malaria and HIV together with the corresponding author's reply. The Journal encourages readers to comment in this column on recently published studies or on any matters relating to the profession of medical laboratory science.

The Journal's questionnaire continues to be a popular avenue for obtaining CPD points with nearly 2,000 members submitting answers in 2009. Another questionnaire appears in this issue, remember to read the questions and the articles carefully as all questions require more than one answer.

Details of the Barry Edwards/Rod Kennedy International Travel Award are in this issue. These scholarships are some of the most significant awards from the NZIMLS and provides the winners support to attend an international or national scientific meeting to a maximum value of $7,500. There will be two scholarships awarded in each calendar year with closing dates of June 30th and December 20th .

Editorial
Conflict of interest in peer-reviewed medical journals: the World Association of Medical Editors (WAME) position on a challenging problem

Lorraine E Ferris[1], Chair, WAME Ethics Committee
Robert H Fletcher[2], Chair, WAME Policy Committee

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Conflict of interest in medical publishing exists when a participant's private interests compete with his or her responsibilities to the scientific community, readers, and society. While conflict of interest is common, it reaches the level of concern when "a reasonable observer might wonder if the individual's behavior or judgment was motivated by his or her competing interests" (1). Having a competing interest does not, in itself, imply wrongdoing. But it can undermine the credibility of research results and damage public trust in medical journals.

In recent years, the extent of conflict of interest in medical journal articles has been increasingly recognized. Medical journals and the popular media have published numerous examples of competing interests that seemed to have biased published reports (2-4). Organizations have expressed concern for the effects of conflicts of interest on research (5), publication (1,6,7), teaching (8) and continuing medical and nursing education (9).

The World Association of Medical Editors (WAME) is one of the institutions engaged in this discussion. WAME was established in 1995 (10,11) to facilitate worldwide cooperation and communication among editors of peer-reviewed journals, improve editorial standards, and promote professionalism in medical editing (12). Membership in WAME is open to all editors of peer-reviewed biomedical journals worldwide; small journals in resource-poor countries are well represented. As of December 2009, WAME had 1595 individual members representing 965 journals in 92 countries. WAME has broad participation as there are no dues and WAME activities are largely carried out through the member list serve and the member password protected website.

In March 2009, WAME released an updated policy statement, "Conflict of Interest in Peer-Reviewed Medical Journals" (1). It details the issues WAME believes journals should address when establishing their own policies for conflict of interest. The editors of this journal thought that the issues were important enough to share with its readers. A summary of statement is presented in the Table and the full statement (1) can be found on WAME's website (12).

How does this statement differ from earlier conflict–of-interest statements?

First, WAME expands the scope of competing interests. Other statements have been concerned almost exclusively with conflicts of interest related to financial ties to industry – companies that sell healthcare products. The assumption is that financial incentives are especially powerful and are not readily recognized without special efforts to make them apparent. WAME has extended the concept of financial conflict of interest to include the effects of clinical income. For example, physicians who earn their livelihood by reading mammograms or performing colonoscopies may be biased in favor of these technologies. WAME has also included non-financial conflicts of interest (or the appearance of one) related to scholarly commitment: "intellectual passion," (the tendency to favor positions that one has already espoused or perhaps even established); personal relationships (the tendency to judge the works of friends/colleagues or competitors/foes differently because of the relationship); political or religious beliefs (the tendency to favor or reject positions because it affirms or challenges one's political or religious beliefs); and institutional affiliations (the tendency to favor or reject results of research because of one's institutional affiliations).

Second, WAME did not prescribe a universal standard for when meaningful conflict of interest exists. Rather, it defined and recommended elements of conflict of interest policies and encouraged journals to establish their own standards. WAME left operational definitions and standards on the basic issues to member journals, recognizing that journals exist in very different contexts across the globe, standards for conflict of interest are evolving, and some journals already have well-established policies and standards. WAME does not presume to judge which conflicts require action and what the appropriate action may be, although its policy does offer factors to consider (1). Obviously, excessive concern for these and more comprehensive lists of possible competing interests could paralyze the peer review and publication process and is not feasible. Editors must make judgments as to the strength of the conflict, but to do so must have uncensored information. Similarly, readers need transparency about conflicts, and therefore editors should publish with every article all relevant author disclosures (1).

Third, WAME confirms the seriousness of failure to disclose conflict of interest by indicating that editors have a responsibility for investigating, and if relevant acting, if competing interests surface after a manuscript is submitted or published. The intent is that allegations of failure to declare conflicts of interest must be taken seriously by journals.

Finally, WAME has addressed in a single statement the conflicts of interests threatening all participants in the research and publication continuum, including authors, peer reviewers, and editors. Conflicts between editors and journal owners, which might affect both the accuracy of articles and the credibility of journals, have been addressed in another WAME policy statement (13).

What can be done about conflict of interest in medical journals?

Conflicts of interest cannot be eliminated altogether but it can be managed so that it has the smallest possible effects on journal content and credibility. The backbone of managing conflicts of interest is full written disclosure; without it, nothing else is possible. Currently, authors may not reveal all of their competing interests and even if they do, journals too often do not publish them (14), so there is plenty of room for improvement. Even so, disclosure alone is an imperfect remedy; editors still must determine whether a conflict has sufficient potential to impair an individual's objectivity such that the article should not be published. Even more work may be needed on reviewers' and editors competing interests, given their critical role as gatekeepers for the medical literature.

No statement will solve the conflict of interest problem, nor will it ever be solved altogether. As understanding of the problem and its management evolves, journals should be given latitude to establish their own standards, matching their policies to the best standards of their discipline and culture. WAME believes journals should make these policies readily accessible to everyone. All of us—editors, authors, reviewers, and readers--should be paying more attention to conflict of interest than we have been. We hope this statement serves that purpose.

Acknowledgements

The authors wish to warmly thank the World Association of Medical Editors (WAME) Officers for their helpful comments on an earlier version of this editorial. Many thanks to President Margaret Winker (USA); Past President Michael Callaham (USA); Vice-President John Overbeke (Netherlands); Treasurer Tom Lang (USA); and Secretary Farrokh Habibzadeh (Iran).

The WAME Statement on Conflict of Interest in Peer-Reviewed Medical Journals was approved by the WAME Board in March 2009. Many thanks to the members of the WAME Ethics Committee and to the WAME Policy Committee for their insightful and helpful comments on an earlier version of the statement. Warm thanks to the WAME Board for their input and comments: Margaret Winker; Michael Callaham; John Overbeke; Tom Lang; Farrokh Habibzadeh; Adamson Muula (Malawi) and Rob Siebers (New Zealand).

Conflicts of interest

As a WAME Director, Lorraine Ferris did not participate in the WAME Board vote to approve the statement or the vote to endorse the editorial. The authors have no conflicts of interest to declare.

This Editorial may appear in other medical and biomedical journals whose editors are members of WAME.

Table 1. Summary of key elements for peer reviewed medical journal's conflict of interest policies

Element	Key aspects	Comments
1. Definition and scope	A clear definition the journal uses as to what is conflict of interest and who is captured in the definition.	Sample definition: Conflict of interest exists when a participant in the publication process (author, peer reviewer or editor) has a competing interest that could unduly influence (or be reasonably seen to do so) his or her responsibilities in the publication process (submission of manuscripts, peer review, editorial decisions, and communication between authors, reviewers and editors).
2. Types of competing interests	A clear statement of examples of the types of competing interests (and their definitions) the journal says must be declared. Should include the following as examples but there could be others: (a) Financial ties (b) Academic commitments (c) Personal relationships (d) Political or religious beliefs (e) Institutional affiliations	There is a need to consider a wide range of competing interests (and a recognition that they can coexist) which the individual assess as to whether they unduly influence (or be reasonably seen to do so) his or her responsibilities in the publication process. Examples and definitions of what competing interests should be declared needs to be articulated with Journals moving beyond just financial conflict of interest.
3. Declaring conflict of interests	Clear statements on (a) what is to be declared, when and to whom; (b) format for declaration; (c) a journal's role in asking additional questions or seeking clarification about disclosures; and, (d) consequences for failing to disclose before or after publication.	Journals rely on disclosure about the facts because *routine* monitoring or investigation is not possible. This creates a particular onus on the declarer to report carefully and comprehensively. It also means that journals should ask about conflict of interest in such a way that there will be a high likelihood of reporting relevant conflict of interest.
4. Managing conflict of interests	A clear statement on how conflict of interest will be managed by the journal, including the position that all relevant conflict of interest disclosures (or the declaration of no conflict of interest) will be published with the article and clarity about what conflict of interest situations will result in a manuscript not being considered.	Journals use various rules about how they will deal with conflict of interest and conflict of interest disclosures and these need to be made known to all those involved in the publication process.

References

1. WAME statement on conflict of interest in peer-reviewed medical journals. http://www.wame.org/conflict-of-interest-in-peer-reviewed-medical-journals
2. Bekelman JE, Li Y & Gross CP. Scope and impact of financial conflicts of interest in biomedical research: a systematic review. *JAMA* 2003; 289(4): 454-65.
3. Lexchin J, Bero LA, Djulbegovic B, Clark O. Pharmaceutical industry sponsorship and research outcome and quality: systematic review. *BMJ* 2003; 326(7400): 1167-70.
4. Altman LK. For science's gatekeepers, a credibility gap. The New York Times May 2 2006. http://www.nytimes.com/2006/05/02/health/02docs.html?scp=58&sq=conflict+of+interest+%26+medicine&st=nyt
5. Institute of Medicine. Conflict of interest in medical research, education, and practice. Washington, DC: National Academies Press, 2009 (April) http://www.iom.edu/Reports/2009/Conflict-of-Interest-in-Medical-Research-Education-and-Practice.aspx
6. International Committee of Medical Journal Editors (ICMJE). Uniform requirements for manuscripts submitted to biomedical journals; ethical considerations in the conduct and reporting

of research: conflicts of interest. http://www.icmje.org/ethical-4conflicts.html
7. International Committee of Medical Journal Editors (ICMJE). Uniform format for disclosure of competing interests in ICMJE journals. October 2009. http://www.icmje.org/format.pdf
8. American Association of Medical Colleges. Industry funding of medical education: report of an AAMC task force. June 2008.
9. Hager M, Russell S, Fletcher, SW (eds). Continuing education in the health professions: improving healthcare through lifelong learning. Josiah Macy J Foundation, November 2007. http://www.josiahmacyfoundation.org/documents/pub_ContEd_inHealthProf.pdf
10. Squires BP, Fletcher SW. The World Association of Medical Editors (WAME): thriving in its first decade. *Sci Edit* 2005, 28(1): 13-6.
11. Launching the World Association of Medical Editors: report of the conference to promote international cooperation among medical journal editors 1995. http://www.wame/org/Bellagio.htm
12. WAME Website. http://www.wame.org
13. WAME policy on the relationship between journal editors-in-chief and owners (formerly titled Editorial Independence). http://www.wame/org/resources/policies#independence
14. Bhargava N, Qureshi J, Vakil N. Funding source and conflict of interest disclosures by authors and editors in gastroenterology specialty journals. *Am J Gastroenterol* 2007; 102(6): 1146-50.

Editor's comments on conflict of interest editorial

Rob Siebers
University of Otago, Wellington

Authors potential conflicts of interest may lead to biased published articles. Potential conflicts of interest may be having received speaker's fees or conference attendance support from companies whose products are a main focus of the article. In most cases these would not bias authors in the interpretation of their studies. However, the potential remains and it is therefore important that authors indicate any potential conflicts of interest so that editors, reviewers and readers may form their own opinion of the article. Additionally, as stated in the above Editorial *"The backbone of managing conflicts of interest is full written disclosure; without it, nothing else is possible."*

The Editor of the New Zealand Journal of Medical Laboratory Science is a Board Director of WAME and has had an input in WAME's position of conflicts of interest, and thus fully supports the views above. As from now, authors submitting articles to the New Zealand Journal of Medical Laboratory Science will not only have to declare their exact involvement in the study, but also declare any potential conflicts of interest. If accepted for publication, the authors contributions to the study and potential conflicts of interest will be published with the article. If there are no conflicts of interest disclosed it will be stated that the authors have declared no conflicts of interest. Additionally, reviewers will also have to declare any potential conflicts of interest that may impact on their ability to impartially peer review submitted articles. Included are the Journal's Editors and Editorial Board Members. This puts the Journal in line with an increasing number of international peer reviewed biomedical journals for open transparency and for the benefit of authors, readers and anyone involved in the editorial process.

Conflicts of interest

The Editor is a Board Director of WAME and thus participated in approving the statement and endorsing the Editorial. The Editor declares no other conflicts of interest.

Cher bro': a thought that may cross your mind

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Research Centre for Māori Health & Development, Massey University, Palmerston North

Introduction

In 1976, my research career started in the laboratory of the paper-board mill at Whakatane. I worked for the Ministry of Agriculture and Fisheries for four years after I completed my degree in earth sciences. In 1988, I was offered a position at Whakatane Hospital in the Hepatitis Research Unit and my focus became health and the epidemiology of hepatitis B.

A requisite skill in our business is phlebotomy. My introduction was patiently supervised with the wisdom that comes with experience, a high level of skill as well as the steely resolve to account for the novice student. During a morning clinic I was introduced to and observed most of the patients that morning. The process was carefully explained. Before breaking for morning tea my tutor strapped on a tourniquet above her elbow. Pointing unflinchingly to the vein she asked me to draw blood. I was all thumbs as blood was drawn into the syringe. As the syringe filled I became nervous - unsure when to release the tourniquet. The needle was withdrawn and blood dispensed into the evacuated tube once its red rubber lid was pierced. No blood was spilt.

In my mind, medical technology is the application of science to the pathology of the human person, their fluids and tissues. An important aspect is the training and credentialing of a highly skilled workforce - pathologists, scientists and technicians. This training and unique skills set are practiced using protocols and a language which to the outsider appears to be a culture unto itself. To many outside the profession, medicine appears this way. For those who remember drawing their first blood sample, you may have felt as apprehensive or anxious as I was. The last thing I wanted was to do any harm. Your patient was probably nervous too. Health professionals and patients have important and complementary roles, each drawing on diverse life histories, cultural values and language.

Māori, who have a culture and rich language unique to New Zealand, have norms that are unfamiliar to most health professionals. Māori have well established protocols for the handling of the person, the processing of bodily samples, and their correct disposal. These protocols are a matter of respecting the integrity of a person, their mauri. In this paper I will reflect on how these matters can be approached, some history and illustrative experiences.

Background

The experience of Māori using laboratories I imagine is much same as non-Māori when dealing with you as a professional and highly skilled workforce. At the Hepatitis Research Unit I worked with Sandy Milne who taught me a lot about the health industry and research. During those six years we built on the work he had done with Māori. The two indelible memories from this time are meeting a man who had recently been diagnosed with liver cancer and Dr Eru Pomare. By the end of this talk those two Māori men will help illustrate ways of helping Māori through the uncertainty that happens when people are not well.

You may want to know the 'go to message' before you read further. Technical competence is important for safety and integration across the spectrum of the health sciences. Complementary to safety and integrating multiple disciplines across the health sector is the interaction with the patient. Interacting well with the patient is a dance - the best outcome happens when the right thing is done at the right time. To address the question, "how might you better deal with Māori when they come to laboratory?" the go to message is to treat people with respect and understanding by first acknowledging them in a manner that naturally puts them at ease. I hope that with a greater knowledge of Māori health, that you will better understand what is important for Māori and how to negotiate the meeting their needs. Respect will flow both ways and we all will become better people for that.

Understanding Māori health statistics

In order to provide some evidence as to why you may wish to acknowledge a person who is Māori, back it up if you like, I will outline the health status of Māori as reflected by official statistics, provide a view of health illustrated by a couple of Māori perspectives and then present an analysis of health with respect to the Treaty of Waitangi. Prof Pomare led the research published first by the New Zealand Medical Council (1-3). Pomare's leadership in Māori health was prematurely ended when he died in 1995. Prior to his death he established a leading Māori health research team at the Wellington School of Medicine in 1992 where he was Dean of Medicine after post graduate study in Bristol. At about the same time a family stricken by liver cancer presented for assistance at the Hepatitis Research Unit at Whakatane Hospital. While neither met, the future for this family along with most Māori living in the Eastern Bay of Plenty was improved with the case Pomare brought to bear with the Ministerial review of the Hepatitis B vaccination program.

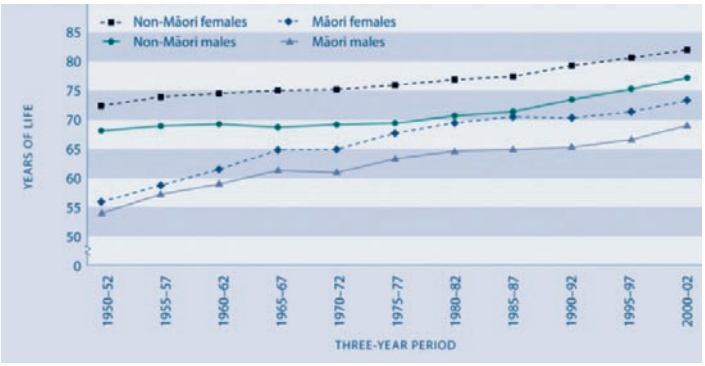
While pathology is the focus of this presentation, my view of Māorihealth may help you better understand the needs of Māori in a broader sense, ranging from hospital to health survey data. And to this end provide you with additional insight that will complement your chosen profession. In New Zealand Māori are well most of the time — most have a high quality of life, are safe, fed and loved. When anyone is not well, society accepts some obligation to meet their needs as is does for all of its citizens. In most cases care is provided and medicine intervenes to mitigate any further harm. Most of this care is provided by the family, however most of the funding is raised by taxes and the Government directs the funding for medicine through Vote:Health.

The broad scope of Māori health and wellbeing is linked to a number of domains that include the environment while measuring the effect of disease and counting numbers who die along side quality of life. The association between environment and health is well rehearsed and articulated within New Zealand (1-9) and by international comparison (10). Other health related data is collected from surveys commissioned by the Ministry of Health. In these surveys on a wide range of health issues, Māori are identified by ethnicity classification (11,12). The standard now accepted is the census ethnicity standard established by Statistics New Zealand (13).

Health indicators and a good life

Life expectancy for example: Life expectancy continues to vary by ethnic group. For non-Māori, life expectancy steady increased in at birth between 1985 and 2002. Boys born during this period are likely

to live 5.8 years than their older brothers. The same is expected for girls who can expect to live an additional 4.5 years than their older sisters. During the same period Māori, life expectancy increased a great deal for those children born between 2000–2002 after many year of smaller increases in life expectancy from the 1980s and well into the 1990s.



Source: Statistics New Zealand, 2007 (14)

While the gain in life expectancy for Māori over the whole period is 4.1 years for a boy, 2.7 years for girls, this was less than that for non-Māori, Māori gained more than non-Māori in the latter five-year period. Perhaps there has been some catch up as the difference in life expectancy has appeared to reduce. The gap in life expectancy at birth between non-Māori and Māori, which widened by 2.4 years between 1985–1987 and 1995–1997, reduced by 0.6 years in the five years to 2000–2002 (14).

The reduction of life expectancy for Māori children was coincident with the reforms of the 4th Labour Government and the later National Governments of the 1990s. It is unfair to blame governments for differentials in life expectancy and trends that may exacerbate this, however it was a time of relatively greater poverty for Māori as parts of the economy they populated were those most severely affected.

Will a widening of life expectancy be observed for this current recession begun in 2008?

In short the difference remains. Māori death rates are higher than non-Māori death rates at all ages. As a result, life expectancy at birth for females of Māori ethnicity was 75.1 years in 2005–07, compared with 83.0 years for non-Māori females. For males, life expectancy at birth was 70.4 years for Māori and 79.0 years for non-Māori. This is an average difference between Māori and non-Māori of **8.2** years in 2005–07, slightly less than the 8.5 years in 2000–02 and 9.1 years in 1995–97 (Statistics New Zealand, 2009). The impact of shorter life expectancy happens along the life course.

Health data is also collected from hospital discharges, death registration and cancer registration. There is much published in this area. The health status of New Zealand compares unfavorably with many other OECD countries (14). Within the population there are major health inequalities and disparities between Māori and Pacific, and people from low-income families compared to other ethnic groups. The Māori Health Strategy, *He Korowai Oranga* (15) was set in place in 2000 because New Zealand had relatively high infant death and youth suicide rates, child immunisation coverage statistics were static or decreasing, levels of hospitalisation for asthma and respiratory problems were unacceptably high, and unintentional injury and poisoning rates were high (16). The criteria that set the Māori Health Strategy in place have remained durable and so have the inequalities.

You may have noticed that most of the health data for Māori is contrasted with non-Māori. Comparison with non-Māori is useful when assessing need and has been used to establish a case for changing priorities to shift health resources. However, the evidence for Māori health priorities is often inadequately supported by health data because some issues fall outside the health sector (education and te reo Māori) and the data is not collected. The more serious and systemic Māori health issue for the health sector is the lack of fine grained detail in the statistics because of a "lack of numbers".

Once we dig below population level data we encounter multiple analytical challenges. Most of these challenges arise from a lack of numbers to populate a comprehensive statistical analysis. Further insight has been difficult to find because of the low numbers of Māori identified. Lack of numbers has precluded sufficient evidence to change priorities for comparatively rare causes of illness or death, or where Māori are disproportionally affected but low rates in the general population rank the issue as a low priority. This has been compounded by incomplete collection of ethnicity data. Recent reforms to data collection and incentives offered to fund health providers using capitation has led to more complete recording of ethnicity. The Government offered the incentive of providing higher fees for more complete patient registration that included ethnicity status.

To improve the quality of data collected in surveys and related health research, the 'lack of numbers' the recruitment of Māori for such research has had to under go a major rethink. A policy based on the notion of 'explanatory power' (17) was used to ensure sufficient numbers of Māori were enrolled as part of the survey in order to have the statistical power to provide summary data with the same level of confidence as non-Māori .

While challenging the value of medical intervention, the health of Māori, like any New Zealander, is dependent on many factors outside the domain of medicine. Medicine's value lies in its power to resolve illness **after** preventative and early intervention strategies have failed. Associated with many strategies for preventing illness are the actions of Government and publically funded services. Most recent strategies for improving Māori have been the Whanau Ora policy initiative driven by the Associate Minister of Health, Tariana Turia (18).

Government assistance

Community and government concern about the well being of Māori has been a feature of policy for some time (16). Widespread concern in social, health and education policy had extended in some cases to questioning the ability of the Government to respond to the needs of Māori (19). In terms of priorities for Māori, the health and well being of Māori children was a concern to both Government and Māori. The non-responsiveness of the Department of Social Welfare (DSW) was termed "cultural racism" in the findings of *Puao Te Ata Tu.* The findings were illustrative of the capacity of Government to assess the status of Māori children and intervene in their best interests of Māori (20).

During a series of radio interviews soon after the release of the report of the Ministerial Advisory Committee, John Rangihau predicted the likely life course of his whanau if the response of Government did not change:
"I have thirty grandchildren, with ages from twenty; twenty years to twenty days. If we're, if I am to expect what has been happening over the, over the last few years to my, to Māori *children, then I can reasonably expect to have six of those children, somewhere along the line, being absorbed by the institutions of the Social Welfare Department and eventually into the penal institutions of the country. That is the reality for us and that is what we are trying to address when we are talking about* Māori *people being responsible for looking after their own children which you must admit and people must admit have not been the case up to now and hence, our real need, to have the courts acknowledge the fact that we need to do things in a different way"* (21).

If government services continued to plot the same course, Rangihau's forecast and the prospects for Māori youth were poor and lent support for better consultation with Māori. Prior to the radio interview, a strong recommendation was made to undertake consultation with Māori. The Royal Commission on Social Policy suggested that consultation be

undertaken with Māori to further social policy objectives:
"Debate need not dwell on whether Māori values or delivery systems are appropriate to a particular policy area; more [sic] fruitfully, objectives should examine the methods by which Māori participation can be maximised and effect given to the Treaty of Waitangi" (22).

In the 1980s, the relative newness of the consultative role for Māori in informing social policy was illustrated by the consultation process that resulted in the recommendations of the report, Puao-Te-Ata-Tu –The Report of the Ministerial Advisory Committee on a Māori Perspective For The Department of Social Welfare (Puao-Te-Ata-Tu) and the April Report (20, 22-24).

Puao Te Ata Tu was the report of an inquiry to inform the then Minister of Social Welfare, Hon Ann Hercus on her department's responsiveness to Māori. The report was presented in July 1986, published in 1988. Within two years Dr Mason Durie was appointed as one of the three commissioners on the Royal Commission into Social Policy and in turn as part of the commission inquiry heard much about the social circumstances of Māori (22). The responsiveness of government to Māori has been poor at times, and the report by retired Family Court Judge Michael Brown (25) provided evidence of the inaction and policy-led neglect by Government through its Ministries.This inquiry was informed by interviews undertaken by Judge Brown.

There has been, and still is, a lack of detailed information to describe the health and wellbeing (including mental health) of Māori in terms that would inform the comprehensive delivery of health and related social services (26,27). The scarcity of such information limits the effective planning, development, funding, delivery and evaluation of prevention and treatment services for Māori, but also the assessment of the determinants of health. The fragmented approach governments have taken to addressing the needs of Māori remained a theme consistent for some time. The aim of Whanau Ora will be to reduce some of this fragmentation and have more Māori providers funded to address unmet need (18).

Māori health in government and non-government

The Government's influence on the needs of Māori is influenced by the structure of government, external pressures and changes made by government in the health sector. Many organisations take responsibility for maintaining the health of New Zealand and for promoting Māori health priorities. The health sector is complex with the Ministry of Health (MOH) primarily responsible for health policy and health funding for Māori health. In addition the office of the Health and Disability Commissioner and a number of other ministries now have an explicit interest in the health and wellbeing of Māori, including the Ministry of Social Development (MSD), the Ministry of Youth Affairs (MYA) and The Ministry of Māori Development Te Puni Kōkiri (TPK). Outside government a number of non-government organisations (NGO) have specific interests' in Māori health, many are Māori organizations including Māori health providers, Waananga and tribal executives.

Influencing health are social, cultural and economic factors (28) or health determinants as described by the National Health Committee (NHC) in the first section of their 1998 report (29). The NHC observed there was "now good evidence that social, cultural and economic factors are the most important determinants of good health" (29) — a relationship exists between these determinants and health. A point of view shared by the health determinants and their association with health have their genesis in the disciplines of economics and social sciences (30). The use of social epidemiology and economic theory provided new perspectives and tools with which researchers and policy makers explored economic and social data and where appropriate, undertake an analysis of indicators relevant to the health of the population. While economic theory may provide a choice of models on which to understand the behaviour of populations and inform policy development, how Māori health is understood crosses disciplinary boundaries that may ascribe health domains in a manner not sensible to Māori. An understanding of how health can be described from several Māori perspectives will provide new insight into how cultural, social and economic factors may influence Māori health.

Health concepts and being well

The introduction of models to describe health and wellbeing for Māori enabled the provision of more appropriate delivery mechanisms for health services in a health sector where Māori views were quite different to the paradigm of western medicine. The development of health services for Māori is not only prefaced on equity of health status for Māori, and the availability of choice as to provider but also in cultural relevance and cultural congruity.

Three models will be analysed and discussed in relation to the notion of health determinants for Māori. The models are Te Whare Tapa Whā (reference 31, pp. 69-73, 76) and Te Wheke (reference 31, pp.74,76; reference 32). They are presented in their order of appeared in the literature, however, this does not reflect on their origins, nor their importance, as they are views of health which accord with contemporary Māori thinking.

Te Whare Tapa Whā

Dr Mason Durie (reference 31, pp. 68-73) concluded a health hui for Māori Women's Welfare League workers undertaking training for the Rapuora research project (33). Durie drew together themes identified by speakers to create an image of a house, a representation of the relationship between four principles of health. The house (te whare) is a metaphor for health where the house's four sides (tapa whā) represent spiritual (taha wairua), mental (taha hinengaro), physical (taha tinana) and family (taha whānau) health. Together all four are necessary to ensure strength and symmetry, and in balance, represent good health.

Te Whare Tapa Whā is an influential model for describing concepts of health and wellbeing from a Māori perspective. The durability of this model and it's wide application in health policy indicate a successful "bridge" between two worldviews as non-Māori begin to deliver services, referencing this model to meet Māori need and also featured in the 'April Report" (22). While physical and mental health had close parallels with medicine, there appears to be no place for family (whanau) and spiritual (wairua) aspects of Māori health. It may be helpful from the perspective of medicine to consider wairua and whanau as health determinants.

Te Wheke

Dr. Rangimarie (Rose) Turuki Pere, a well-respected indigenous educator, described Te Wheke (the octopus), a model of health, at the Hui Whakaoranga (reference 31, pp. 75) in 1984. Dr. Pere described eight principles that intertwined like the tentacles of the octopus. The close relationship between the principles (tentacles) and health from a Māori perspective enabled Māori and non-Māori to understand the inter-relationship between these principles and wellbeing (32).

Te Wheke, developed for an education setting is based on the principles of ako, integrating the dimensions of wairuatanga, tinana, hinengaro, whanaungatanga, mana ake, hā a koro mā, hā a kui mā, and whatumanawa. This model illustrates the features of health from a whānau perspective. The head and body represent the whānau, and the tentacles represent each of the eight dimensions of health, "The suckers on each tentacle represent the many facets that exist in each dimension." (reference 32, pp. 3). The intertwining of the tentacles represents the manner in which each of the dimensions is interrelated. Durie observed that "waiora, total wellbeing for the individual and family, represented by the eyes of the octopus" (reference 31, pp. 75). The additional aspects of health as described by Pere suggests that there are aspects of Māori health that may provide finer detail to understanding health determinants that may fit outside the narrow scope of health indicators we use in medicine.

These three models of health described Māori approaches to conveying an understanding of how Māori concepts of health related to the environment and could be conveyed in English to a wider audience. Each was developed for a specific purpose. Te Whare Tapa Whā was used to teach how four domains related to each other in a manner that illustrated that their collective value relied on maintaining a balance (33). In a similar manner Te Wheke provided greater detail of how inter-personal relationships shaped health and well being and the transmission of knowledge. The dynamism of the inter-relationships between domains reflected the subtle and important role of the whanau. In each model emphasised importance of the relationship Māori had with their whanau. A recurring theme in these three models was the recognition and maintenance of inter-personal relationships and the balancing of well being with that of the whanau, the environment. Being Māori is healthy for Māori.

Health, safety and culture

The science on which medicine was established is added to by the second. An important scientist was Professor Eru Pomare. He established a sound footing for Māori health research at the Wellington School of Medicine and championed the establishment of Māori health within the new Health Research Council in 1991. For Professor Pomare, Māori health was an essential part of the health and well being of New Zealand. Many people were influenced by Professor Pomare and I am one of them. He not only added to the value of my work at the Hepatitis Research Unit easier as patron, he enabled the application of the skilled researchers working at Wellington Hospital to bring a new understanding to the epidemiology of hepatitis B in a family who was stricken by liver cancer. This new knowledge improved the health and well being of the family by bringing into their lives the option of diagnosing liver cancer at an earlier stage by regular review of those family member identified as hepatitis B carriers and preventing chronic infection with hepatitis B vaccine for those who had not been exposed to the virus. While liver cancer had brought this family in contact with the Hepatitis Research Unit and the skills of the Wellington School of Medicine, their willing participation in research gave new understanding to their health needs and Māori health. Being Māori and sharing similar cultural values made the building of a relationship much easier and sharing similar cultural values was important.

Culture is often how the behaviour and values of ethnic minorities are described to others. In the context of culture, the issue of health is shared between the patient and health professional but viewed from their perspective which may not share important factors. Understanding how safety is threatened by known scientifically proven hazards is something we are trained for as scientist. Differences in cultural norms play an important role in the relationship between the patient and the health professional. Mitigating the risk of the unknown may in part be a matter of understanding what health issues are important to your patient and strategies that can be used to avoid risk or mitigate negative outcomes while maintaining high scientific standards. Understanding not only the situation in which your patient may present but also the balancing their expectations with what can be explained is where professional skill meets art. Considering what may guide you these situations is what understanding more about Māori health may contribute to the better application of science to meet the health needs of your patients.

The skill of the phlebotomist is reflected in the willingness of the patient to be a partner to the successful extraction of blood. The tasks of the phlebotomist include the location of a suitable vein and extracting the blood while putting the patient at ease. Ensuring that the patient is relaxed as possible is in part how quickly a comfortable relationship is established. Knowing the person's name and pronouncing it in a manner in which they are familiar is a good start.

Phlebotomy is also an example of multi tasking, an artful application of science and social skills. However, for the learner the art of putting the patient at ease comes some time after the anatomy of the vein is intuitive and the insertion of the needle a reflex. For many Māori tena koe is a greeting said by a host who wishes to acknowledge their visitor as a Māori. To the patient who is Māori, tena koe is the recognition of a human person by another who appreciates the value of being Māori. Cher bro is used by younger Māori as a greeting signifying warmth and familiarity, having the same positive recognition as tena koe and bringing minds closer together.

Māori health is different to that of non-Māori. Health statistics tells us a story of a greater burden of ill health and disease borne by Māori and that Māori die earlier than their non-Māori peers. The factors of life and environment that influence Māori health and wellbeing are know and can be treated as health determinants. Where people interact with medicine is an opportunity to get off to a good start and build a good relationship. When next you meet a new patient and think to yourself should I address this person with tena koe or cher bro, you will have undertaken a new way of thinking about your patients. Should you begin to think of new and old patients in this way, the next time you are face to face with a patient, you reward your self with a "cher bro".

References

1. Pomare EW. Hauora: Māori standards of health: A study of the 20-year period 1955-75. Medical Research Council of New Zealand, Auckland, 1980.
2. Pomare EW, de Boer GM. Hauora: Māori Standards Health; a study of the years 1970-1984. Wellington: Department of Health, Wellington and Medical Research Council, 1988.
3. Pomare E, Keefe-Ormsby V,Ormsby C, Pearce N, Reid P, Robson B, Watene-Haydon N. Hauora: Māori Standards of Health III, a study of the years 1970-1991. Wellington: Te Ropu Rangahau Hauora a Eru Pomare, Wellington School of Medicine, 1995.
4. Ajwani S, Blakely T, Robson B, Tobias M, Bonne M. Decades of Disparity: Ethnic Mortality Trends in New Zealand, 1981–1999. Ministry of Health and University of Otago, Wellington, 2003.
5. Blakely T, Fawcett J, Atkinson J, Tobias M, Cheung J. Decades of Disparity II: Socioeconomic Mortality Trends in New Zealand, 1981–1999. Ministry of Health and University of Otago, Wellington, 2005.
6. Harris R, Tobias M, Jeffreys M, Waldegrave K, Karlsen S, Nazroo J. Effects of self-reported racial discrimination and deprivation on Māori health and inequalities in New Zealand: cross-sectional study. *Lancet* 2006; *367*: 2005–9.
7. Jeffreys M, Stevanovic V, Tobias M, Lewis C, Ellison-Loschmann L, Pearce N, et al. (2005). Ethnic inequalities in cancer survival in New Zealand: linkage study. *Am J Publ Health* 2005; *95*: 834-7.
8. Weir RP, Brunton CR, Blakely TA. Chronic liver disease mortality attributable to hepatitis B and C in New Zealand. *J Gastr Hepatol* 2002; 17: 582-8.
9. Smylie J, Anderson I, Ratima M, Crengle S, Anderson M. Indigenous health performance measurement systems in Canada, Australia, and New Zealand. *Lancet* 2006; 367: 2029-31.
10. Parnell W, Scragg R., Wilson N, Schaaf D, Fitzgerald E. NZ Food NZ Children: Key results of the 2002 National Children's Nutrition Survey. Wellington: Ministry of Health, Wellington, 2003.
11. Watson PD, Denny SJ, Adair V, Ameratunga, SN, Clark TC, Crengle SM, et al. Adolescents' perceptions of a health survey using multimedia computer-assisted self-administered interview. *Austr N Z J Publ Health* 2001; 25: 520-4.
12. Statistics New Zealand. Measuring ethnicity in The New Zealand population census, 2002. Retrieved March 29, 2007 from http://www.stats.govt.nz/NR/rdonlyres/964F9590-89EF-442B-A5E2-CEC248380989/0/Ethnicityquestion.pdf
13. Statistics New Zealand. QuickStats About Māori, 2007. Retrieved March 28, 2007, from http://www.stats.govt.nz/census/2006-census-data/default.htm

14. Hodgson HP. The challenge for public health. Public Health Association National Conference, Palmerston North, 2007
15. Minister of Health. He korowai oranga Māori health strategy discussion document. Office of The Minister of Health, Hon Annette King, Office of the Associate Minister of Health, Hon Tariana Turia, Wellington, 2001
16. Ministry of Health. Child Health Strategy. Ministry of Health, Wellington, 1998
17. Te Röpü Rangahau Hauora a Eru Pömare. Mana Whakamärama - Equal explanatory power: Mäori and non-Mäori sample size in national health surveys. Paper commissioned by Public Health Intelligence, Ministry of Health, Wellington, 2002
18. Ministry of Social Development. Whānau Ora: A whānau-centred approach to Māori wellbeing. A discussion paper by the Whānau Ora Taskforce, 2009. Retrieved from http://www.msd.govt.nz/documents/about-msd-and-our-work/work-programmes/initiatives/whanau-ora/whanau-ora-discussion-document.doc
19. Dow DA. Safeguarding the Public Health -a History of the New Zealand Department of Health. Victoria University Press, Wellington, 1995.
20. Ministerial Advisory Committee on a Māori Perspective for the Department of Social Welfare. Puao-te-ata-tu : Day Break - The Report of The Ministerial Advisory Committee on a Māori Perspective for the Department of Social Welfare. Department of Social Welfare, Wellington, 1986.
21. Robinson G, Rangihau JT. The Children and Young Person's Bill. Morning Report, Radio New Zealand Sound Archive, New Zealand, 1987.
22. Royal Commission on Social Policy. The April Report. Royal Commission on Social Policy, Wellington, 1988.
23. Burns B, Hill K, Rangihau JT. Ministerial Advisory Committee on a Māori Perspective of the Social Welfare Department. Morning Report, Radio New Zealand Sound Archive, New Zealand, 1986.
24. Rangihau JTR (Ed.). Puao-Te-Ata-Tu (Day Break) -The report of the Ministerial Advisory Committee on a Māori perspective for the Department of Social Welfare. Social Welfare Department, Wellington, 1988.
25. Brown MJ. Care and Protection is About Adult Behaviour: The Ministerial Review of the Department of Child, Youth and Family Services. Report to the Minister of Social Services and Employment Hon Steve Maharey. Wellington: Office of The Minister of Social Services and Employment, Wellington, 2000.
26. Ministry of Health. Progress on health outcome targets : the state of the public health in New Zealand = Te haere whakamua ki nga whainga hua mo te hauora (No. 1173-9169). Ministry of Health, Wellington, 1995.
27. Te Puni Kokiri Ministry of Māori Development. Progress towards closing social and economic gaps between Māori and non-Māori, Report to the Minister of Māori Affairs (No. 0-478-09133-8). Te Puni Kokiri -Ministry of Māori Development, Wellington, 1998. www.beehive.govt.nz/Print/PrintDocument.aspx?DocumentID=26389
28. Sen A. Mortality as an indicator of economic success and failure. *The Economic Journal* 1998; 108(446): 1-25.
29. National Advisory Committee on Health and Disability. The Social, Economic and Cultural Determinants of Health: Action to Improve Health -a report to the National Advisory Committee on Health and Disability (National Health Committee). Wellington: Ministry of Health, NHC, Wellington, 1998.
30. Berkman L F, Kawachi I (Eds.). Social Epidemiology. Oxford University Press, New York, 2000.
31. Durie MH. Whaiora Māori Health Development (2 ed.). Auckland: Oxford University Press, Auckland, 1998.
32. Pere R. Te oranga o te whanau (The health of the family). Paper presented at the Hui Whakaoranga -Māori health planning workshop, Hoani Waititi Marae, Auckland, March1984
33. Murchie E. *Rapuora:* Health and Māori Women. Wellington: The Māori Women's Welfare League/Te Ropu Wahine Māori Toko i Te Ora, 1984.

Diazyme adenosine deaminase in the diagnosis of tuberculous pleural effusion: method evaluation and clinical experiences in a New Zealand population

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Abstract
Objectives: We evaluated the Diazyme adenosine deaminase assay (ADA) on a commercial auto analyzer (Abbott CI8000; Abbott Laboratories) and the clinical experiences of pleural fluid ADA over a 6 month period in a New Zealand population.

Methods: The ADA assay was evaluated for linearity, precision, interferences, sample stability and accuracy. A retrospective audit of ADA levels in non-transduative pleural fluid from 120 patients over a six month period was conducted.

Results: In a New Zealand population, pleural fluid ADA has shown a sensitivity of 100% and specificity of 86% for tuberculous pleural effusions with a positive predictive value of 24% and a negative predictive value of 100% at a cut-off of 30 U/L.

Conclusions: The Diazyme ADA assay performs in accordance with manufacturer's claims. There are difference in ADA results when compared to an alternative method and caution is advised when literature based cut-offs are applied to the Diazyme ADA method.

Key words: adenosine deaminase, pleural effusion, tuberculosis

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Introduction
The analysis of pleural fluid adenosine deaminase (ADA) levels in the diagnosis of tuberculous pleural effusion (TPE) was established in 1978 by Piras et al (1). Adenosine deaminase assay is available on routine biochemistry analysers and meta-analyses of ADA in pleural fluid has shown ADA to have high sensitivity (90-92%) and specificity (90-92%) for tuberculosis (2). Despite its utility as an adjunctive test in the diagnosis of TPE, ADA is infrequently tested in Australasia. This may be due the low prevalence of tuberculosis and the availability of an automated assay.

Clinical cut-offs for ADA levels are often based on published literature, however the various ADA assays are not standardised and results may not be comparable. A meta-analysis of 40 studies published from 1966 to 1999 showed discrepancies in results among the reported studies (2). This can be attributed to the use of different methods of ADA analysis. Thus it is important that new ADA methods are validated by comparison with clinical outcomes.

We report the results of the method evaluation of the Diazyme ADA assay on the Abbott CI8000 auto-analyzer and the clinical experiences of its use over a 6 month period in a New Zealand population.

Materials and methods
Reagents
An ADA kitset (catalogue number DZ117A-K) manufactured and supplied by Diazyme Laboratories (General Atomics, Ponway, California, US) was evaluated in August 2007 on an Abbott CI8200 automated clinical chemistry and immunoassay analyzer (3).

The reagent kit contains one R1 and one R2 and is ready to use. Parameters for this kitset were obtained after correspondence from the headquarters of Abbott Diagnostics, Brazil. A single level calibrator (catalogue number DZ117A-CAL) and two control levels (catalogue number DZ117A-CON) were also supplied separately by Diazyme. These are lyophilized and stored at -20°C.

Validation study
A sample comparison with Queensland Central Laboratories, Royal Brisbane Hospital, Australia was conducted on six samples to externally verify our results. Their ADA method is an in-house non-Giusti method.

Interference studies
Interference studies were investigated at 3 levels of ADA concentrations; low level ADA 6.4 IU/L, medium level 28.4 IU/L and a high level 189.7 IU/L. Samples were spiked with haemolysate for haemolysis studies. Bilirubin standard dissolved in dimethyl sulphoxide (DMSO) representing bilirubin were spiked for icterus studies and 10% intralipid for lipaemia studies. Each sample was matched with a control spiked with equal volumes of 0.9% saline and measured in duplicates. For icterus studies, DMSO was used instead of 0.9% saline.

Precision studies
For intra-batch precision, 15 patient samples were analyzed in duplicate. For inter-batch precision the control levels at 31.9 U/L and 152.9 U/L were analyzed in 10 consecutive batches. Results are presented as coefficient of variation (CV).

Retrospective audit
The Middlemore Hospital Laboratory analyzes pleural fluid for both Middlemore Hospital and North Shore Hospital which are located in Auckland, New Zealand. They are secondary-level hospitals servicing a population of approximately 475,000 and 500,000 people respectively.

Pleural fluid was analyzed for ADA only if it was classified as a non-transudate. Modified Light's criteria were used to classify pleural fluid as either exudate or transudate (4). Where there was insufficient information to classify a pleural fluid as an exudate or transudate, it was treated as a non-transudate.

Clinical records were used to search for the diagnosis for each pleural fluid ADA tested between 1st of March 2008 to the 8th of September 2008. A diagnosis of tuberculosis required a confirmed culture of *Mycobacterium tuberculosis* in either sputum, pleural fluid or pleural biopsy. The clinical course was followed for a further 3 to 6 months.

Results

Method evaluation

Intra-batch precision was 1.75% while inter-batch precision were 3.4% and 2.7% at the control levels of 31.9 U/L and 152.9 U/L respectively

Good correlation was obtained between our method and that of the Queensland Central Laboratory. However, our results were about 30% lower (Figure 1).

ADA was minimally affected by haemolysis up to 1.8 g/L while ADA results demonstrated a 17% reduction by bilirubin concentrations >300µmol/L. Ten percent intralipid reduced ADA levels by 31%. The package insert claims that there is no interference from ascorbic acid up to 4mg/dL However, we did not verify this claim. No other interferences were tested for nor were stated in the package insert.

Retrospective audit

In the period from 1 March 2008 to 8 September 2008 there were 21 out of 120 patients with ADA results above the cut-off of 30 U/L (note: some patients had more than one pleural fluid where ADA was measured, only the first result was included in the analysis). Of these, 5 were confirmed TB positive, all with ADA results greater than 64 U/L (Figure 3).

Table 1 shows the ADA results >30 U/L and the diagnosis. Table 2 compares ADA data from Middlemore Hospital with published studies and shows that our sensitivity (100%) and specificity (86%) compared well with published studies. Positive predictive value (PPV) was 24% and the negative predictive value (NPV) was 100% for an ADA cut-off of >30 U/L. The PPV increased to 45% and NPV was 99% at an ADA cut-off of >70 U/L. However, the sample size of tuberculous pleural effusions were small and calculations based on this maybe subject to variation.

Discussion

The diagnosis of TPE is usually determined on a combination of clinical, radiological and laboratory findings. In most series of patients with TPE, stains for acid fast bacilli are detected in less than 10% of cases (5). Pleural fluid cultures for TB are more sensitive with a yield ranging from 12% to 70% (5), however, turnaround time for cultures is usually 3 to 4 weeks. Polymerase chain reaction (PCR) has excellent specificity (78% - 100%) but there are significant differences in sensitivity amongst methods used (20% - 90%) owing to the presence of amplification inhibitors, type of primer used, genomic sequence amplified and the number of mycobacteria (2). Pleural biopsy is the gold standard for diagnosis of TPE but this is an invasive procedure and there are risks of complications. A biopsy involves a stain, culture and histology. In patients with TPE, pleural biopsy culture is positive in 39% - 80% of cases and histological examination reveals granulomatous inflammation in 50% - 97% of cases (2).

Adenosine deaminase (ADA) is an enzyme in the purine salvage pathway that catalyses the conversion of adenosine and deoxyadenosine to inosine and deoxyinosine with the release of ammonia. ADA is found in most cells but plays an important role in the differentiation of lymphoid cells (in particular, active T-lymphocytes). Elevated pleural fluid ADA levels are not specific for TPE and it may be seen in diseases such as lymphoma, empyema, malignancy, pneumonia and rheumatoid-associated pleural effusions (rheumatoid arthritis or systemic lupus erythrematosus).

The methods for measuring ADA can be grouped into broadly two groups: Giusti and non-Giusti. One of the first methods for ADA was described by Giusti and Galanti (5). In this reaction the first step, where adenosine plus water is converted to inosine and ammonia by ADA, is the same. Ammonia then reacts with sodium hypochlorite and phenol in an alkaline solution (with sodium nitroprusside as the catalyst) to form an intensely blue indophenol compound. Non-Giusti methods includes the reduced nicotinamide adenine dinucleotide-linked kinetic method, ADA deaminases adenosine to inosine. The ammonium released is then used by glutamate dehydrogenase to convert 2-oxoglutarate to L-glutamate, with the concomitant oxidation of NADH to NAD^+, allowing the reaction to be monitored by following the decrease in absorbance at 340 nm (6). The Diazyme ADA is a non-Giusti method, based on the enzymatic deamination of adenosine to inosine. Inosine is converted to hypoxanthine by purine nucleoside phosphorylase. Hypoxanthine is converted to uric acid and hydrogen peroxide by xanthine oxidase. Hydrogen peroxide is further reacted with two other compounds to generate a quinone dye. This is a rate up reaction measured at 548nm.

At Middlemore Hospital samples for ADA estimation are analysed three times per week. Our studies have shown that ADA in pleural aspirates are stable for up to six hours at room temperature and up to 1 month at 2°C - 8°C (unpublished studies). However, we advise transportation at 4°C. Miller et al looked at using preservatives, such as a mixture of glycerol and ethylene glycol or a mixture of glycerol and sodium sulphate, to enable samples to be shipped at ambient temperatures. However, this may be more beneficial in regions where refrigerated transport of samples is problematic (7).

We noted significant differences in ADA levels between different methods as illustrated in Figure 1. In meta-analyses optimum cut-off for ADA were different depending on the method utilised. There are no international standardisation programs for adenosine deaminase assays and published cut-off should therefore be viewed with caution. Determination of adenosine deaminase cut-offs should be method specific and use local populations where possible.

The sensitivity and specificity of ADA increases when the cut-off of >70 U/L is used, however, ADA cannot be used alone to diagnose TPE. Its value as an early marker of TPE was highlighted by one of our cases, where a high ADA result was obtained 55 days before a sample from this patient grew *Mycobacterium tuberculosis.* Cultures will still be required to identify mycobacterial drug resistance.

In the New Zealand population an elevated ADA level >70 U/L has a low positive predictive value (24%) for tuberculosis. However, in practice the causes of false positive results, such as empyema, are often identified promptly. The negative predictive value is very high (100%) and in cases where there is a low pre-test probability of TPE, a low ADA result can rule out the need for invasive procedures such as pleural biopsy. Most of the literature cites 40 U/L as the cut-off levels for ADA. The meta-analysis by Goto el al (including Giusti, non-Giusti and unknown methodologies) featured studies that used a cut-off levels between 30 U/L and 71 U/L.

At the initiation of the ADA assay we provided reference interval and interpretative comments based on the literature and the comparison of ADA results with an external laboratory. In view of the 30% lower ADA levels as compared to an external laboratory, we conservatively stated a low cut-off value of 30 U/L to maintain the assay's negative predictive value. A clinical grey-zone was disclosed, to acknowledge the uncertainty of the optimum cut-off for this assay in the New Zealand population.
Comments were as follows:

A. <30 U/L: Not strongly suggestive of tuberculous effusion. There is a high negative predictive value when the pre-test probability is low. However, if clinical details suggest a high pre-test probability, adenosine deaminase results should be interpreted with caution.

B. ≥30- 70 U/L: Borderline range, value of indeterminate significance. Specificity for tuberculosis is significantly improved if the effusion is predominately lymphocytic and empyema is excluded.
C. ≥70 U/L: Highly suggestive of tuberculosis. However, high levels can be seen in empyema, lymphoma and rheumatoid pleural effusions.

A limitation is that the study design was a retrospective audit and as such suffers from deficiencies inherent with this methodology; diagnoses other than tuberculosis were based on judgement from clinicians and did not have strict inclusion and exclusion criteria. There were only five confirmed cases of tuberculous pleural effusion in this cohort, therefore caution is advised when forming conclusions.

In conclusion, ADA is an economical enzymatic assay able to be performed on automated commercial analyzers. It is a useful adjunctive test in the diagnosis of tuberculous pleural effusions, especially in view of its short turnaround time. It identifies those patients who are high risk for tuberculosis and guiding investigations which are required to confirm the diagnosis. In low prevalence populations its excellent negative predictive value may save costs and unnecessary invasive procedures.

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Table 1. Diagnosis of pleural effusion ADA results >30 U/L

Diagnosis	Prevalence (n)
Tuberculosis	24% (5)
Neoplasm	29% (6)
Empyema	24% (5)
Para-pneumonic effusion	19% (4)
SLE pleuritis	5% (1)

Table 2. Middlemore Hospital ADA data compared with published data

Study	# Patients or studies	Cut-off (U/L)	Sensitivity	Specificity	PPV or LR*	NPV or LR*
MH	120 patients	30	100%	85%	23%	100%
Zaric et al[8]	121 patients	49	89.2%	70.4%	84.4%	78.4%
Liang et al[9]	63 studies		92%	90%	9.03*	0.1*
Goto et al[2]	40 studies	30-50	92.2%	92.2%		

MH = Middlemore Hospital *LR = likelihood ratio

Figure 1. Patient sample correlation with Queensland Central Pathology, Royal Brisbane Hospital.

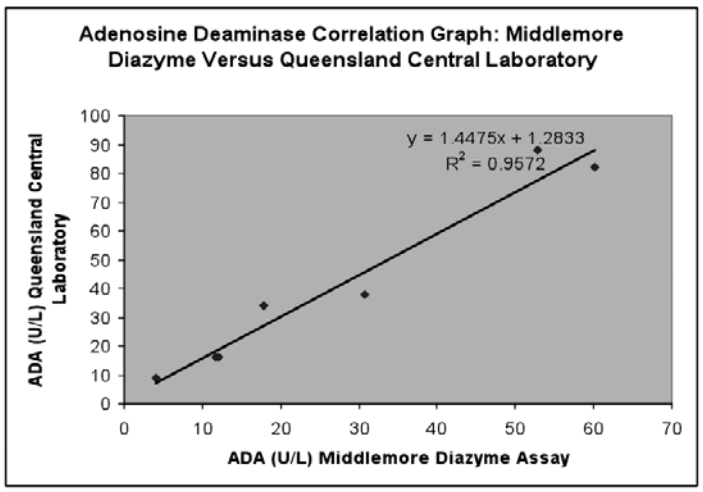
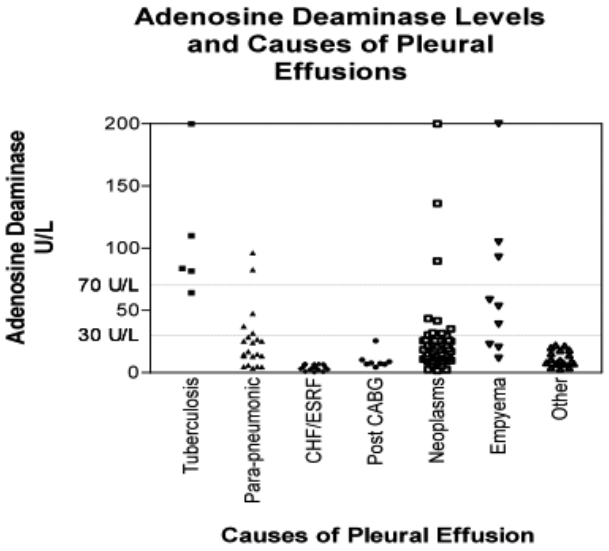


Figure 2. ADA results from March 2008 to September 2008
CHF: congestive heart failure; ESRF: end stage renal failure; CABG: coronary artery bypass graft



References

1. Piras MA, Gakis C, Budroni M, Andreoni G. Adenosine deaminase activity in pleural effusions: an aid to differential diagnosis. *Br Med J* 1978; 2 (6154): 1751-2.
2. Goto M, Noguchi Y, Koyama H, Hira K, Shimbo T, Fukui T. Diagnostic value of adenosine deaminase in tuberculous pleural effusions: a meta-analysis. *Ann Clin Biochem* 2003; 40: 374-81.
3. Guisti G, Galanti B. Methods in Enzymatic Analysis. Academic Press, New York, NY; 1974: 414-9.
4. Ellis G, Goldberg DM. A reduced nicotinamide adenine dinucleotide-linked kinetic assay for adenosine deaminase activity. *J Lab Clin Med* 1970; 76: 507-17.
5. Adenosine deaminase assay kit package insert. Diazyme Laboratories, catalogue number: DZ117A-K.
6. Light RW. Clinical practice. Pleural effusion. *N Engl J Med* 2002; 346: 1971-7.
7. Miller KD, Barnette R, Light RW. Stability of adenosine deaminase during transportation. *Chest* 2004; 126: 1933-7.
8. Zaric B, Kuruc V, Milovancev A, Markovic M, Sarcev T, Canak V, et al. Differential diagnosis of tuberculous and malignant pleural effusions: what is the role of adenosine deaminase? *Lung* 2008; 186: 233-40.
9. Jiang J, Shi H-Z, Liang Q-L, Qin S-M, Qin X-J. Diagnostic value of interferon-γ in tuberculous pleurisy: a metaanalysis. *Chest* 2007; 131: 1133–41.

Author contributions

David Song was the primary author, carried out the method evaluation and clinical correlations. Andrea Lund liaised with Diazyme Laboratories for the ADA assay and was involved with the method evaluation. Weldon Chiu advised on method evaluation, was involved with the reference intervals and contributed to writing of the article.

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HELLP syndrome – a case study

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Abstract

Background: HELLP syndrome is a life-threatening obstetric complication considered by many to be a severe form of pre-eclampsia involving haemolysis, thrombocytopenia and liver dysfunction. Both HELLP and pre-eclampsia occur during the later stages of pregnancy, and sometimes after childbirth. HELLP syndrome is a clinically progressive condition. Early diagnosis is critical to prevent liver distension, rupture and haemorrhage and the onset of Disseminated Intravascular Coagulation. If the condition presents antenatally, morbidity and mortality can affect both mother and baby.

Case study: We report a case study of HELLP syndrome in a 40 year old post-partum woman, who had evidence of pre-eclampsia in the weeks leading up to delivery. Within 24 hours of delivery the patient's blood pressure was rising significantly, she experienced upper epigastric pain and vomiting, her platelet count was dropping rapidly and blood tests revealed that her liver enzymes were becoming increasingly abnormal. All of these symptoms led to the rapid diagnosis of HELLP syndrome.

Conclusions: This case is of interest because if the diagnosis had been delayed, there could have been a significant risk of liver rupture, DIC or haemorrhage, and patient survival could have been compromised.

Key words: HELLP syndrome, haemolysis, liver enzymes, platelet count, hypertension, liver distension, pre-eclampsia

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Introduction

HELLP syndrome was first described by Pritchard et al in 1954 (1). However, the first published article naming the syndrome as HELLP appeared in the literature almost thirty years later. Weinstein reported his findings in a unique group of pre-eclamptic/eclamptic women who had a severe form of pre-eclampsia characterised by **h**aemolysis, **e**levated **l**iver enzymes, and a **l**ow **p**latelet count (2). He devised the acronym HELLP to name the syndrome. HELLP syndrome occurs in 4-12% of women with severe pre-eclampsia and can manifest at any stage during pregnancy. Approximately 30% of cases occur postpartum and in these cases, only 80% will have been diagnosed as pre-eclampsia antenatally (3).

HELLP syndrome can be associated with poor maternal and perinatal outcome. Maternal morbidity is mostly associated with disseminated intravascular coagulation (DIC), placental abruption, acute renal failure and ruptured liver haematoma. Most perinatal deaths are associated with placental abruption, intrauterine asphyxia and prematurity.

Case study

The patient had recently had a normal vaginal delivery of twins at $36^{+}6$ weeks gestation on the 9th January 2008 at 0900 and 0918 respectively. Her obstetric history includes two normal vaginal deliveries with no antenatal or postnatal complications. She was recovering in the postnatal ward and as she had exhibited mild symptoms of pre-eclampsia in the weeks leading up to delivery, her vital signs were being closely monitored. The majority of women with pre-eclampsia are expected to recover post delivery, but this patient's condition deteriorated rapidly over the next 24 hours of the postnatal period. Oral Augmentin was prescribed post delivery for 5 days. See tabulated results at the end of the case study.

9 January 2008

1500 Blood pressure had risen from 140/75 to 167/92.

1845 Blood pressure 161/95. Patient complained of chest pain radiating into her back, she vomited and had epigastric pain.

1930 Blood pressure 192/96. Labetalol prescribed 8 hourly. Patient was pale and continued to complain of epigastric tenderness. The clinical team queried whether she was having a reaction to oral Augmentin. Dexamethasone was added to patient's medications.

2020 A significant difference in haematology and biochemistry results was noted as follows:

Parameter (reference range)	**0507**	**1940**
Platelets 10^9 (150-400)	138	118
WBC 10^9 (4-10)	7.4	10.5
AST U/L (0-30)	26	216
ALT U/L (0-40)	15	105
LDH U/L (125-243)		
GGT U/L (0-36)		9

2050 Blood pressure 192/106.

2100 Marked haematuria was evident. Epigastric pain was worsening. Her consultant gynaecologist was called and due to her thrombocytopaenia, raised liver enzymes and epigastric pain, a diagnosis of HELLP syndrome was made. Her haematuria worsened and she had exaggerated reflexes. Blood pressure was now to be monitored every 15 minutes. ICU specialist consulted and a decision was made to repeat blood tests in 2 hours and if the results had deteriorated further, she was to be admitted to ICU. $MgSO_4$ infusion commenced as per ICU protocol. $MgSO_4$ minimises the risk of seizures in patients with marked hypertension.

2130 Indwelling catheter was draining frank haematuria. The patient described her chest pain on a 1-10 scale as 1/10. Chest x-ray was performed. Obstetrician was not convinced of the diagnosis of HELLP syndrome as 0_2 saturation was normal.

2155 Further blood tests taken. In this two hour period the platelet count had dropped even more to 74 $x10^9$; WBC had risen to 13.0 $x10^9$; AST had risen significantly to 1083 U/L; ALT had risen to 439 $x10^9$ and the LDH ws extremely high at 2,386 U/L; GGT was 11 U/L.

The patient now had severe HELLP syndrome. An acute admission from obstetrics to ICU was arranged.

2200 Epigastric pain was now described by the patient as 5/10. Patient declined analgesia.

2300 Patient admitted to ICU. There was a delay in admission to ICU because the obstetrician was not convinced of the diagnosis

of HELLP syndrome. Also the biochemistry specimens collected at 2155 were clotted and no biochemistry results were available until 2300.

10 January 2008

0015 Further blood tests taken. The platelet count had dropped even more to 40 $x10^9$; AST had risen higher to 1,482 U/L; ALT was essentially unchanged at 416 U/L; LDH had risen more to 2,585 U/L and GGT was 13 U/L.

0040 Haematologist consulted and it was decided to keep the platelet level at 50 $x10^9$ and authorisation to transfuse 1 unit of apheresis platelets was given. Obstetrician also discussed the benefits of plasmapheresis/exchange transfusion with the haematologist. The decision was made that at this stage there was not enough evidence to support this procedure and that this would be reviewed again later in the morning. Target blood pressure was 170/110 or less and controlled with medications.

0115 Blood pressure 192/101. $MgSO_4$ infusion increased as per ICU protocol. One off dose of hydrocortisone was administered.

0200 Further blood tests taken. Post platelet transfusion the platelet count had only risen slightly to 50 $x10^9$.

0600 Blood pressure 165/84. Further blood tests were taken to monitor the platelet count. Platelet count had now dropped back to 45 $x10^9$. Haematologist decided to keep the platelet level >20 $x10^9$.

0740 Further blood tests were taken to monitor the platelet count. Platelet count had now dropped to 40 $x10^9$. A GGT was performed which had now risen slightly to 14 U/L.

1000 Patient was improving clinically. Urine was clearer with excellent urine output. No epigastric pain.

1400 Further blood tests were taken to monitor the platelet count which now was 41 $x10^9$.

Further blood tests throughout the day showed no significant differences from the above results.

11 January 2008

0200 Blood pressure 130/70. Urine now clear with no signs of haematuria. Platelet count 44 $x10^9$.

0740 Further blood tests taken. Platelet count 45 $x10^9$; AST had fallen significantly to 208 U/L; ALT had dropped to 172 $x10^9$ and GGT still was 14 $x10^9$.

1030 Transferred from ICU back to obstetric ward.

1100 Blood pressure 131/74.

1400 Blood pressure 120/64.

12-14 January 2008

Final summary of blood tests taken 12-14 January are included in the tabulated results below. The only thing of note was that the GGT kept rising until discharge.

Table 1. Overview of physiological and pathology results

Date	Time	Blood Pressure	WCC	Platelet Count	AST	ALT	LDH	GGT	Haematuria	Medications	Blood Products
9.1.08	0507		7.4	138	26	15					
	0918	140/75								Augmentin	
	1500	167/92									
	1845	161/95									
	1930	192/96								Labetalol and Dexamethasone	
	1940		10.5	118	216	105		9			
	2050	192/106									
	2100								marked	$MgSO_4$	
	2130								frank		
	2155		13	74	1083	439	2386	11			
10.1.08	0015			40	1482	416	2585	13			
	0040										Platelet transfusion
	0115	192/101								$MgSO_4$ increased. Hydrocortisone	
	0200			50							
	0600	165/84		45							
	0740			40				14			
	1000								slight		
	1400			41							
11.1.08	0200	130/70		44					Clear		
	0740			45	208	172		14			
	1100	131/74									
	1400	120/64									
12.1.08	0800		11.6	82	83	116					
13.1.08	0945		8.2	121	72	99		100			
14.1.08	0630		8.0	138	61	91		121			
	2213		14.0	206	44	82		135			
16.1.08	1515		8.7	272	20	45					

Figure 1. Platelet count over time

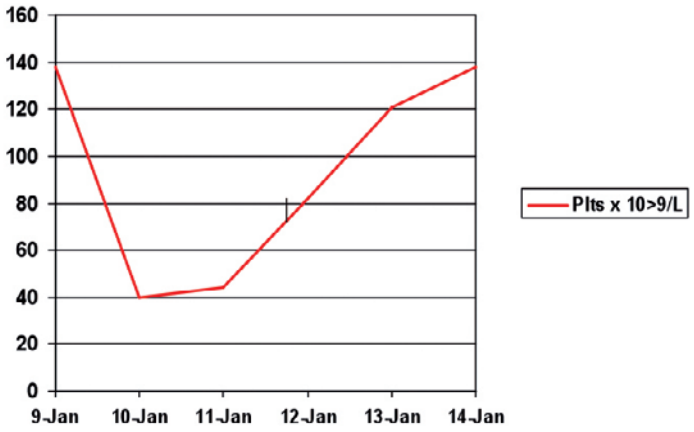
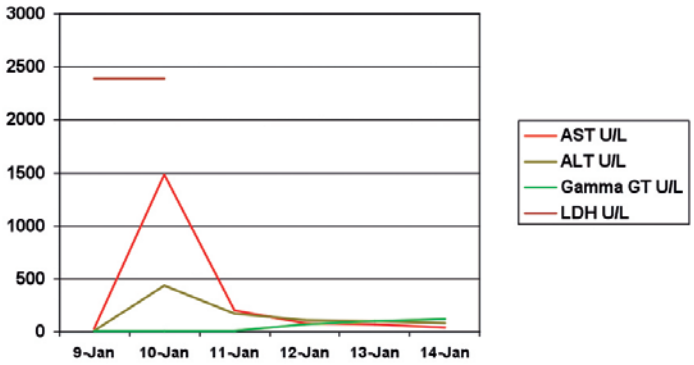


Figure 2. Liver enzyme levels over time



Discussion

The actual cause of HELLP syndrome is not well understood, however evidence now suggests endothelial cell damage during implantation in the uterine wall is an early stage of the disease (4). Tissue damage and insufficient blood supply due to blocked arteries can develop later in pregnancy in HELLP syndrome. This can cause the obstruction of blood flow and liver distension that can lead to rupture and haemorrhage. Liver distension causes the epigastric or right upper quadrant pain associated with HELLP syndrome.

The presentation of HELLP syndrome may be non-specific and misdiagnosis is more likely when the condition develops before term. Diagnosis may be delayed with consequent risk for mother and fetus (4). The symptoms of HELLP syndrome include epigastric pain, nausea and/or vomiting, non-specific viral illness type symptoms, visual disturbances, headache, bleeding from the gums, jaundice, and neck or shoulder pain. As the classic symptoms of pre-eclampsia such as hypertension and proteinuria are not always present in women with HELLP syndrome, a non-obstetric diagnosis, such as gall bladder disease, viral hepatitis, gastroenteritis, kidney stones, peptic ulcer, acute fatty liver of pregnancy, idiopathic thrombocytopenia purpura, thrombotic thrombocytopenia purpura, pyelonephritis and haemolytic uraemic syndrome, may be made. However, disseminated intravascular coagulation (DIC), placental abruption and fetal death contribute to the significant maternal and fetal morbidity and mortality involved with the condition (3).

The laboratory criteria for the diagnosis of HELLP syndrome most commonly used in clinical practice were defined by Sibai (4). However, there remains confusion regarding terminology and diagnosis and a lack of consensus regarding which tests and levels should be used to diagnose the syndrome. The following presents an overview of laboratory investigations most commonly used in the diagnosis of HELLP syndrome.

Haemolysis

Haemolysis, defined as the presence of microangiopathic haemolytic anaemia, is a hallmark of HELLP (5). It has been noted that only a small number of cases have overt haemolysis (5). Haemolysis is confirmed by an abnormal peripheral blood smear with the presence of burr cells, shistocytes and polychromasia. Activation of the coagulation cascade leads to fibrin forming cross-linked networks in the small blood vessels. The red blood cells become damaged when passing through these blood vessels leading to a microangiopathic haemolytic anaemia (4,5).

Elevated Liver enzymes and liver damage

Elevated levels of liver enzymes reflect damage within the liver. Hepatic damage results from micro-emboli in the hepatic vasculature. Jaundice may be present; serum bilirubin levels rise as a result of haemolysis (6). High levels of alanine transaminase (ALT) are specific for hepatic damage (6). Aspartate transaminase (AST) levels increase with liver damage but the enzyme is also found in other organs thus elevated levels of AST are not specific for hepatic damage (6). Gamma glutamyl transpeptidase (GGT) is found almost entirely in the liver and levels of this enzyme are elevated in HELLP syndrome. Lactate dehydrogenase (LDH) is another liver enzyme which is found in other parts of the body but may also be elevated in HELLP syndrome (6). In our case study the ALT, AST and GGT all continued to rise in the post-partum period. These liver enzymes remained raised during the acute phase of the patient's condition and rapidly returned to normal as she recovered.

Alkaline phosphatase (ALP) is another liver enzyme which may be raised in HELLP syndrome and is often still raised after other liver enzymes have returned to normal. Biochemistry results showed that the patient's ALP was 131 U/L two days post delivery, and 2 days later had further increased to 435 U/L. ALP may also be raised due to placental origin rather than liver damage, but the results indicate that in our case study the raised ALP was due to liver damage and not of placental origin.

Tissue damage and ischaemia within the liver leads to obstruction of blood flow and liver distension which potentiates liver rupture (2). The classic hepatic lesion associated with HELLP syndrome is periportal or focal parenchymal necrosis. Pain is usually localised to the right upper quadrant or mid-epigastric region and is caused by distension of the liver. A radiological finding from an abdominal CT scan on the patient showed tiny subcapsular bleeding in the liver. No bleeding point could be identified.

Low Platelets

Platelet consumption occurs in pre-eclampsia as arteriolar vasospasm damages the endothelial layer of small vasculature, forming lesions. The lesions allow platelet aggregation and the formation of a fibrin network (6). In HELLP syndrome the circulating volume of platelets reduces as consumption increases, resulting in thrombocytopenia. A platelet count of $<100 \times 10^9$/L is significant in the diagnosis of HELLP syndrome (6). Rapid diagnosis and treatment of this patient prevented DIC, a very severe complication of HELLP syndrome. Hydrocortisone and platelet transfusion maintained the platelet count close to 50×10^9. After this critical time, once the threat of DIC had lessened significantly and the patient stabilised further, the platelet count was kept at $>20 \times 10^9$, in keeping with best practice. Keeping the platelet count stable prevented ongoing coagulopathies culminating in DIC.

The syndrome can by typed into Type 1, Type 2 or Type 3 depending on the platelet count with the severe forms generally characterised as Type 1 (4). Women who manifest one or two but not all three of the components of HELLP syndrome have a better prognosis than women with complete HELLP syndrome (4,7). However, it must be realised that as with pre-eclampsia, the natural course of the

disease is to worsen over time (5).

The only definitive treatment for women who have HELLP syndrome is delivery. The condition is potentially fatal for mother and fetus with the most common cause of maternal death being liver rupture (8). Deteriorating liver function and/or progressive thrombocytopenia is an indication for delivery, regardless of gestation. It is important to be vigilant as HELLP may develop postpartum.

Several medications have been investigated for the treatment of HELLP syndrome, but with conflicting evidence. A low platelet count is treated with platelet transfusion, DIC with fresh frozen plasma, and anaemia may require transfusion of red cells. In mild cases, corticosteroids and anti-hypertensive medication may be sufficient. Intravenous fluids are generally required.

In conclusion, this case demonstrates the importance of rapid and early diagnosis of HELLP syndrome and treatment of the symptoms to ensure a favourable maternal and perinatal outcome.

References

1. Pritchard JA, Weisman R, Ratnoff OD. Intravascular hemolysis, thrombocytopenia and other hematologic abnormalities associated with severe toxaemia of pregnancy. *N Eng Med J* 1954; 250: 89-98.
2. Weinstein, L. Syndrome of hemolysis, elevated liver enzymes, and low platelet count: a severe consequence of hypertension in pregnancy. *Am J Obstet Gynecol* 1982; 142: 159-67.
3. Hayman R Myers J. Diagnosis and clinical presentation. In: PE Baker & JCP Kingdom (Eds.). Pre-eclampsia. Current Perspectives on Management. Parthenon Publishing Group, London, 2004: 133-43.
4. Sibai BM. The HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): much ado about nothing? *Am J Obstet Gynaecol* 1990; 162: 311-6.
5. Baxter JK Weinstein L. HELLP syndrome: the state of the art. *Obstet Gynecol Surv* 2004; 59: 838-45.
6. Cowan J. Women's Experience of Severe Early Onset Pre-eclampsia: A Hermeneutic Analysis. Masters Thesis, AUT, Auckland, New Zealand, 2005.
7. Audibert F, Friedman SA, Frangieh AY. Clinical utility of strict diagnostic criteria for the HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome. *Am J Obstet Gynecol* 1996; 175: 460-4.
8. Poole JH. Getting Perspective on HELLP Syndrome. Am J *Matern Child Nurs* 1998; 13: 432-7.

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Evaluation of the Nova Biomedical Statstrip glucose meter

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Abstract
Aims: The Nova StatStrip blood glucose monitoring system was evaluated for precision, accuracy, and interferences from haematocrit and maltose. It was also compared against three other meters and two reference methods.
Methods: Heparinised whole blood samples were analysed on the meters. These results were compared with whole blood samples analysed on the Radiometer ABL835 for the interference studies. Plasma samples, obtained from these whole blood samples, were measured on the Abbott CI8200 for accuracy studies.
Results: There were significant differences in the degree to which the meters correlated with the reference method. With the exception of the StatStrip, all meters were affected by variable haematocrit. Of the two glucose meters tested, the StatStrip did not show any maltose interference.
Conclusions: The StatStrip glucose meter did not show clinically significant interference from maltose or varying haematocrit levels. In addition, the StatStrip demonstrated the best correlation with the reference glucose method.

Key words: glucose, point-of-care, interferences, precision, bias

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Introduction
Point-of-care glucose meters are increasingly being used to make therapeutically important decisions. At Middlemore Hospital we are currently using approximately 160 glucose meters in the diabetes clinic, ICU, neonatal unit and other wards. It is essential that the results from these meters can be relied upon for clinical decisions, and therefore that they correlate well with those of the laboratory analysers.

A major concern of the use of point-of-care glucose meters is analytical interference. Patients are frequently taking numerous medications and critically ill patients and neonates often have an abnormal haematocrit. Previous studies have shown that both maltose(1-3) and haematocrit (3,4) have compromised performance of glucose meters.

Recently, companies have introduced improved glucose meters which are less affected by these interferences. The aim of this study was to assess the performance of the Nova Biomedical StatStrip blood glucose monitoring system evaluated for precision, accuracy, and interferences from haematocrit and maltose. It was also compared against three other meters and two reference methods.

Methods
Glucose meters tested
Nova Biomedical StatStrip, Arkray Glucocard, Roche Accu-Chek Advantage, Abbott Precision PCx Plus and the reference method was Radiometer ABL 835 (reference method was chosen to obtain haematocrit and glucose)

Interference study
Fresh heparinised venous blood drawn from healthy donors was used and allowed to sit at room temperature for 24 hours so that the glucose was almost completely depleted before concentrated solutions of glucose and interfering substances were added. Immediately prior to each interference study, the two blood tubes were spiked with a 1.1mmol/L glucose solution to obtain a low and high glucose range and mixed for at least 10 minutes by rocking.

Haematocrit interference
Each of these two blood tubes was further divided into 4 aliquots of 1ml. Centrifugation, using a micro-centrifuge, and plasma adjustments resulted in three aliquots with different haematocrit levels (22%, 45% and 62%) for each concentration of glucose. All six aliquots were rocked at least 10 minutes before analysis.

For each glucose meter there were 5 replicate measurements at 3 haematocrit levels and 2 different glucose levels, giving a total of 30 data points per meter. The haematocrit interference graph was generated for each tested glucose level using the average recovered value of the 5 replicates obtained for each haematocrit level on each manufacturer's meter.

Maltose interference
Each of these two blood tubes was further divided into 3 aliquots. Two levels of concentration of the maltose interference compound, D(+) maltose monohydrate 278 mmol/L, were spiked to two of the aliquots. The first concentration covered the upper end of the therapeutic or normal range (target 0.28 mmol/L) while the second concentration was in toxic range (target 5.6 mmol/L).

For each glucose meter there were 5 replicate measurements at each of 3 maltose levels and 2 different glucose levels, giving a total of 30 data points per meter. The maltose interference graph was generated for each tested glucose level using the average recovered value of the 5 replicates obtained for each investigated interference level on each manufacturer's meter.
Only two glucose meters (Nova Biomedical StatStrip and Roche Accu-Chek Advantage) were used for the maltose interference study as the other two meters are not subject to maltose interference (4-5). The reference glucose meter was the Radiometer ABL835.

Correlation and bias
This was performed by analysing 120 heparinised whole blood specimens on the four glucose meters, compared to plasma obtained from those specimens and analysed on the CI8200 as reference method._The range of glucose values was 2.3-20.2 mmol/L.

The precision study on the StatStrip was performed by analysing five random heparinised whole blood samples at 20 replicates with a glucose range of 1.0-34.0 mmol/L.

Correlation was by least square linear regression and bias by Altman-Bland plots.

Results
With the exception of the StatStrip, all meters were affected by variable haematocrit (Figures 1 and 2). There was a clear trend for negative bias associated with increasing haematocrit for the PCx, Advantage and Glucocard. Of the two glucose meters tested for maltose interference the StatStrip did not show any significant changes (Figures 3 and 4).

There were significant differences in the degree to which the meters correlated with the reference method. The StatStrip, with a R_2 of 0.226 was the one which correlated best with the hexokinase plasma reference method., followed by the Advantage with a R_2 of 1.092, while the Glucocard with a R_2 of 2.998 and the PCx with a R_2 of 4.213 correlated badly with the reference method (Figures 5-8).
All four meters demonstrated a negative bias. The StatStrip, with a bias from -0.476, was the lowest, followed by the Advantage with a bias of -0.747, PCx with a bias of -1.045 and Glucocard with a bias of -1.731 (Figures 5-8).

Precision was assessed for a glucose range between 1.0 and 33.0 mmol/L The StatStrip showed coefficients of variation (CV) of less than 6% with the exception of a critical low glucose level(<1 mmol/L), where it was more than 13% (Table 1).

Table 1. Within run precision of the Statstrip (n=20)

Glucose	0.7-1.3 mmol/L	1.1-1.6 mmol/L	1.9-2.4 mmol/L	6.3-6.5 mmol/L	30.1-33.7 mmol/L
CV	13.4 %	4.8 %	5.1 %	1.9 %	2.5 %

Discussion

This study has shown that precision for the StatStrip glucose meter was acceptable as well as the correlation study. The StatStrip and the Advantage demonstrated the closest correlation with the plasma hexokinase reference method and demonstrated the lowest absolute bias. The glucose meter that is currently in use at Middlemore hospital, the Glucocard glucose meter demonstrated the second worst correlation with the reference method from the four glucose meters tested. We found similar results for haematocrit and maltose effect on glucose meter that have been published previously.(1-5).

In conclusion, the StatStrip glucose meter did not show clinically significant interference from maltose or varying haematocrit levels. In addition, it demonstrated the best correlation with the reference glucose method.

Acknowledgements

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References

1. Chen ET, Nichols JH, Duh SH, Hortin G. Performance evaluation of blood glucose monitoring devices. *Diabetes Technol Ther* 2003; 5: 749-68.
2. Chance JJ, Li DJ, Jones KA, Dyer KL, Nichols JH. Technical evaluation of five glucose meters with data management capabilities. *Am J Clin Pathol* 1999; 111: 547-56.
3. Karon BS, Griesmann L, Scott R, Bryant SC, DuBois JA, Shirey TL, et al. Evaluation of the impact of hematocrit and other interference on the accuracy of hospital-based glucose meters. *Diabetes Technol Ther* 2008; 10: 111-20.
4. FDA Reminders For Falsely Elevated Glucose Readings From Use of Inappropriate Test Method. http://www.fda.gov/cdrh/oivd/news/glucosefalse.html
5. Holtziger C, Szelag E, DuBois JA, Shirey TL, Presti S. Evaluation of a new POCT bedside glucose meter and strip with hematocrit and interference corrections. *Point of Care* 2008; 7: 1-6

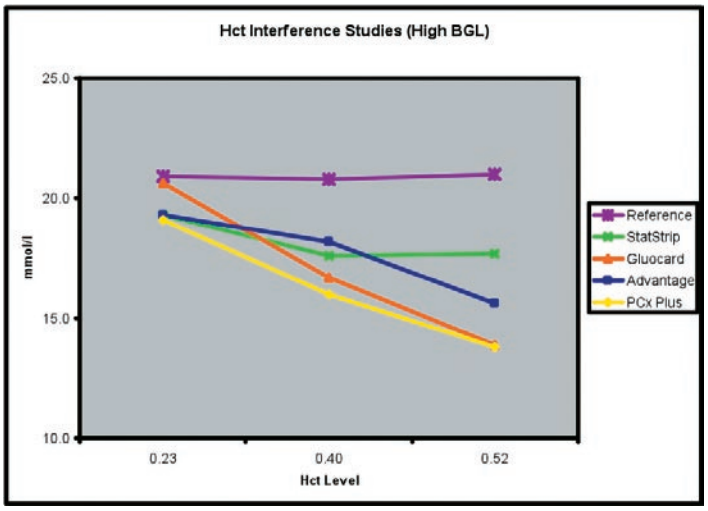

Figure 2. Effect of haematocrit on high glucose levelsevels

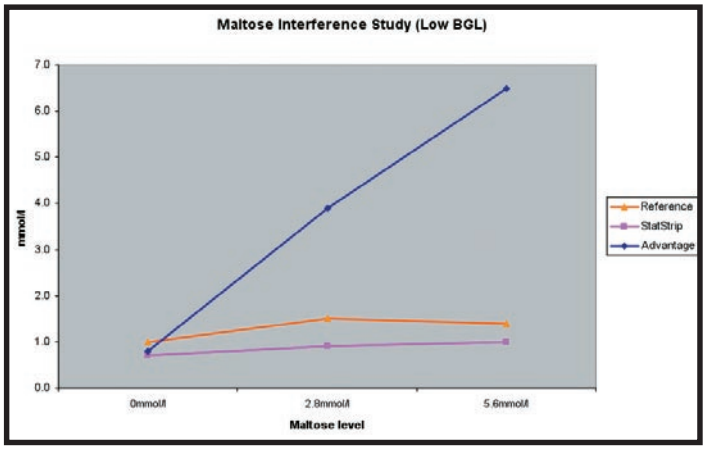

Figure 3. Effect of maltose on low glucose levels

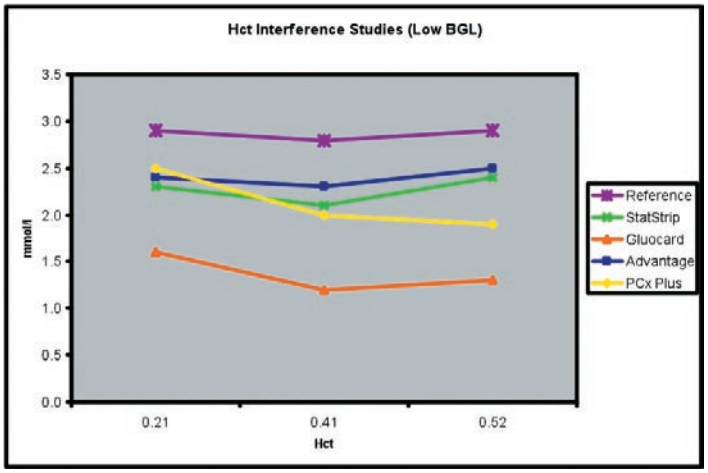

Figure 1. Effect of haematocrit on low glucose levels

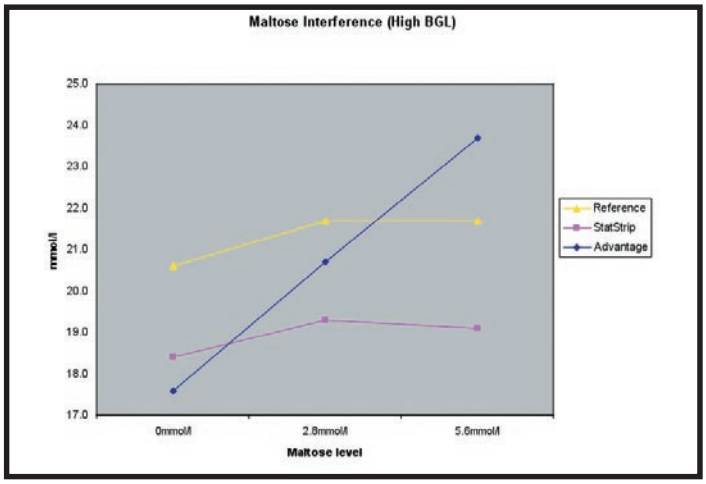

Figure 4. Effect of maltose on high glucose levels

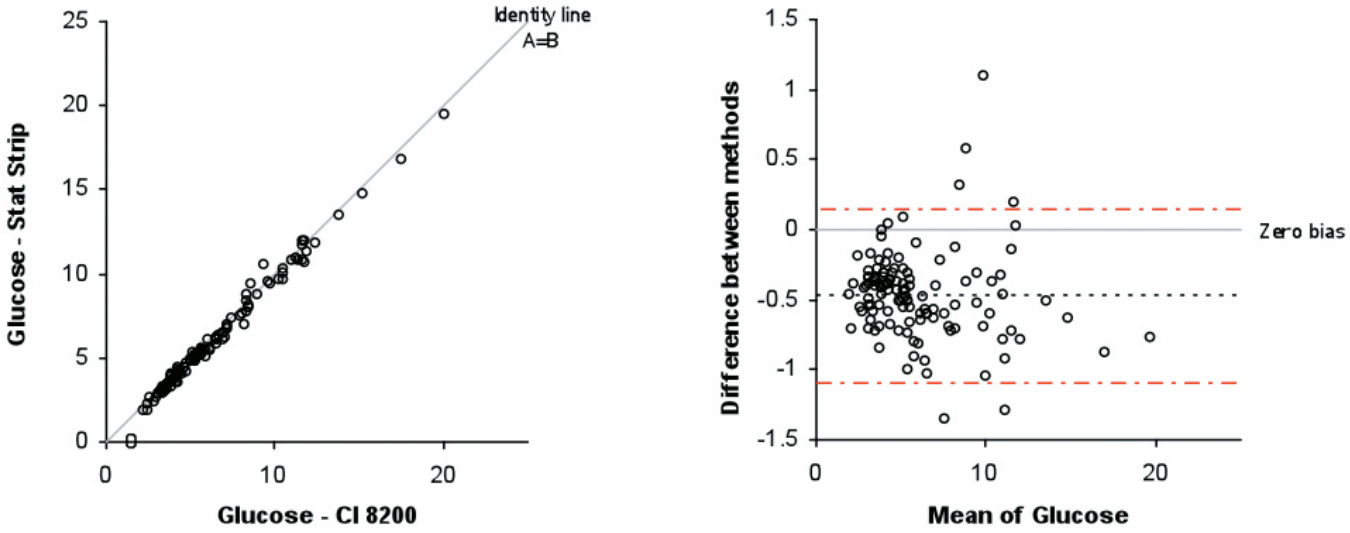

Figure 5. Correlation and bias of Statsstrip versus reference (CI 8200)

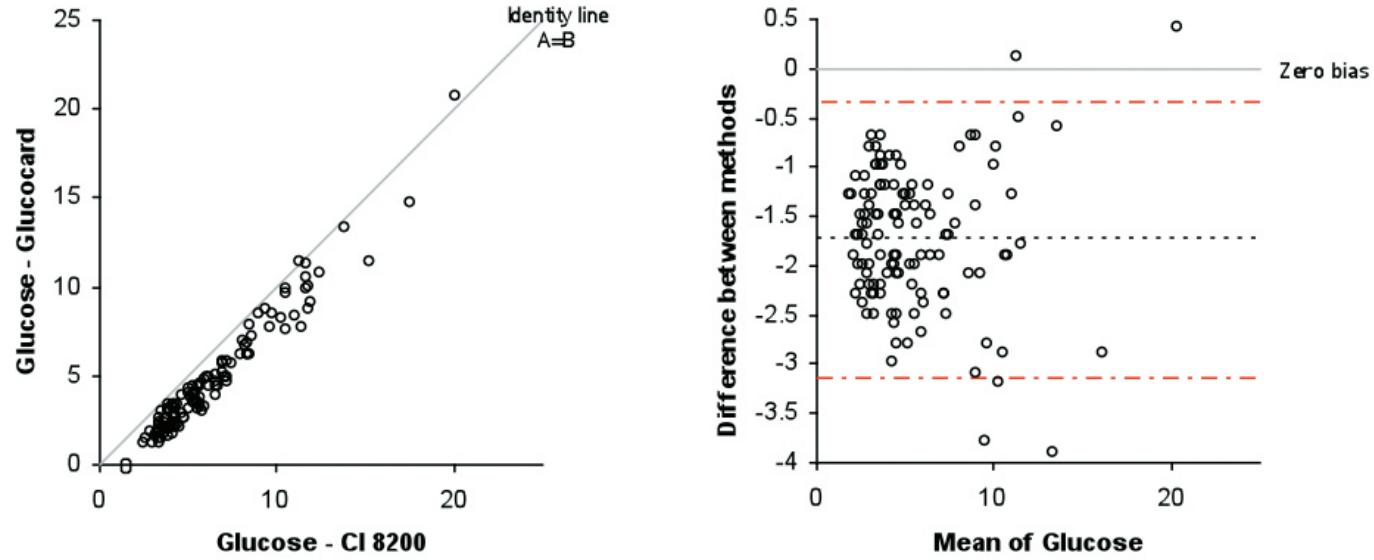

Figure 6. Correlation and bias of Glucocard versus reference (CI 8200)

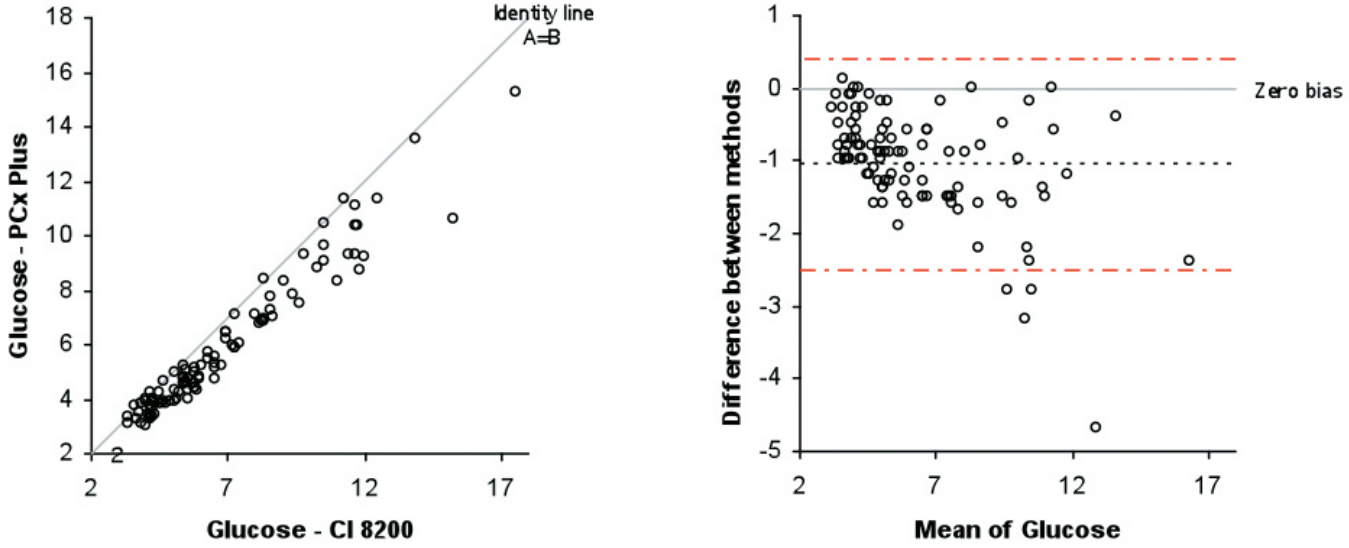

Figure 7. Correlation and bias of PCx versus reference (CI 8200)

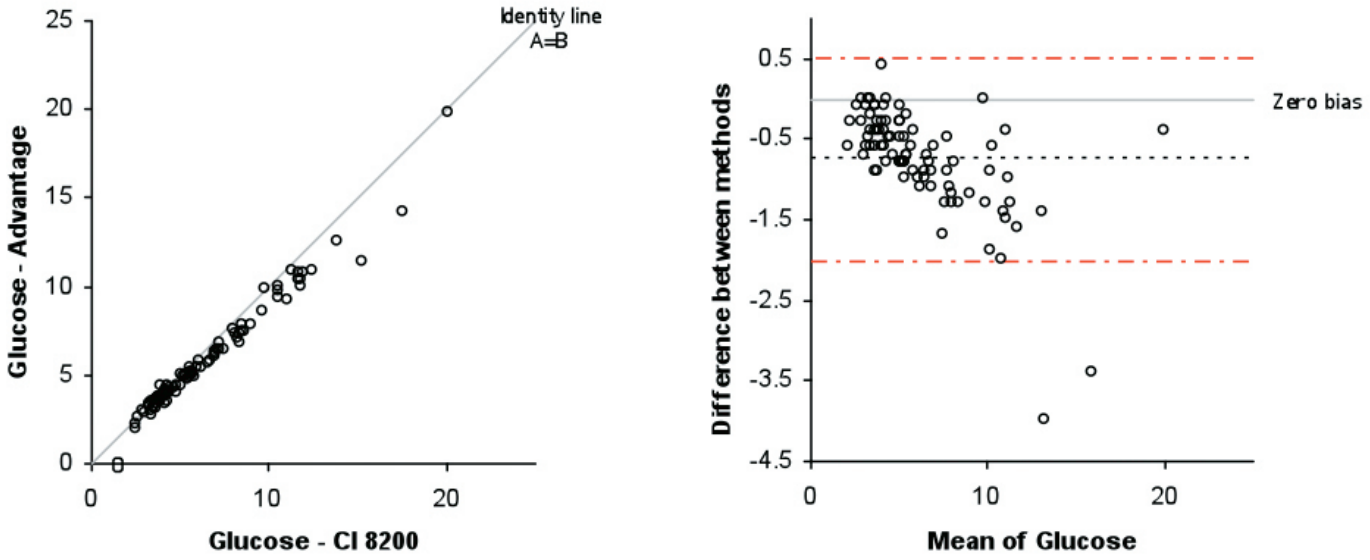

Figure 8. Correlation and bias of Advantage versus reference (CI 8200)

How to write a laboratory-based case study for the Journal

Rob Siebers, FNZIC, FNZIMLS, CBiol FBS; Journal Editor

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

Abstract
Case studies are educational for the reader. They bring together laboratory results with the patient's clinical diagnosis. Case studies include description of an unusual disorder or aetiology, support for or disconfirmation of a clinical hypothesis, new insight into disease pathogenesis, unusual case presentation, and description of adverse drug or food-induced reactions. Case studies show what medical laboratory science is all about - as a diagnostic aid for the clinician. In this article I will briefly describe the characteristics of a case study for submission to the Journal.

Key words: case study, clinical diagnosis, laboratory results

***N Z J Med Lab Sci* 2010; 64: 22-23**

Introduction
Case studies published in the Journal brings together laboratory results with the patient's medical conditions and clinical diagnosis. They are educational for the reader and shows what medical laboratory science is all about - as a diagnostic aid for the clinician. Reasons for submitting case studies include: presentation of an unusual disorder or unusual aetiology; to support or disconfirm a clinical hypothesis; offer new insight into disease pathogenesis; to describe an unusual case presentation; or to describe adverse drug or food-induced reactions.

For the last two years there has been an annual prize awarded by the NZIMLS for the best case study published in the Journal during the calendar year (1,2). In this educational article I will briefly describe how to write up a case study and hope this will spur some from our profession to submit case studies to the Journal. Over the years many good case studies have been presented at SIG or other scientific meetings yet only a very few have been published in the Journal, where a larger and an increasing international readership awaits. Case studies are important contributions to the medical laboratory science literature and evidence base, are educational for readers, and are often a way for authors to start their publication record.

Case studies typically are divided under the following headings: Title, Abstract, Introduction, Case Report, Results, Discussion, Conclusions, Acknowledgements, References.

Title
The title should be accurate description of the case study followed by the words – "a case study", or case studies if more than one patient is presented as in a retrospective analysis. Do not use a funny title, except maybe as a subtitle. The title should alert the reader to the main focus of the case study.

Abstract
Unlike a scientific article, the abstract for a case study is unstructured, i.e. no subheadings of background, methods, results and conclusions. The abstract should start with a very brief background generally outlining the clinical condition of the case study to be presented. This is followed by a paragraph briefly outlining the case study with only the relevant clinical details and the main laboratory results. In the final paragraph state the overall conclusion arising from the case study. At the end include up to five key words, preferably medical subheading terms from Index Medicus. An excellent example is the case study published in the August 2008 issue of the Journal (1).

Introduction
Describe clearly the purpose of the case study and provide a brief review of the published literature pertaining to the topic. Do not write an in-depth review of the topic. The best type of articles to reference here are previously published review articles and, if relevant, the first described case in the literature. Do not discuss your case study in relation to the published literature here. This is for the Discussion section later.

Case report
This is the part where you describe the patient's medical presentation, outcomes, treatments if applicable, and laboratory results. The case study has to be presented in a chronological order with enough but succinct details. For the laboratory results include your laboratory's reference ranges and units of measurement. Often, if there is a lot of laboratory data, especially if the patient is followed over many days, it is better to present the data in tables and/or figures. Do not repeat data in text that is presented in the tables or figures. Only present data that is pertinent to the case study, do not add other laboratory data from tests ordered that do not add value to the case study.

An important consideration is patient privacy. Nowhere in the case study must the patient be able to be identified. If a photo of the patient is critical for the case study report, written informed consent is essential. Contact your local institution's ethical authority for guidance and approval. Another consideration is the clinician or medical team caring for the patient. They may have plans to write up the case study for possible publication in a medical journal. If published elsewhere, duplicate publication of the case in the Journal is not allowed. Contact the primary care physician in the first instance and explain that you wish to publish the case, focussing primarily on the laboratory data. Offering the clinician co-authorship, often leads to agreement in allowing you to write up the case study.

Discussion
This is the part of the paper where the author discusses the case and the laboratory findings. Relate the case to what is already known from the published literature and if the results are different to what has been published, discuss possible reasons for this and what your opinion is. State any limitations to your case study. For instance, it may have been useful to have additional laboratory results for other tests, but these were not ordered at the time. You should also state, if so, what was unique about your case study.

Conclusion
The conclusion should briefly and succinctly be what was learned from the presented case study, it should not be a repeat of the case

history. Do not make any unsupported statements, conclusions or suggestions here.

Acknowledgements

Acknowledge anyone who has assisted you but does not justify authorship (3). For instance, you may have asked a colleague to read a draft of the article and asked for critical advise. Or one of your colleagues may have gathered the data or performed some of the laboratory analyses. Do ask their permission to acknowledge them.

References

Use only references from peer-reviewed articles that preferably are indexed in the major databases such as PubMed, EMBASE or CINAHL. Do not reference abstracts or personal observations. Reference only articles that are pertinent to the case study. In many cases only one authorative reference per point made is necessary. Make sure that you have read in full the referenced article and make sure that the whole reference (authors names, article title, journal abbreviation, volume number and page numbers) is accurate (4). Do not reference data from abstracts as research has shown that up to 40% of published abstracts may have data in them that is not consistent with what is reported in the full text of the article (5).

Tables and figures

Only put relevant data in tables. If there are just a few data points do not put them in a table. Rather, these should be clearly conveyed in a sentence or two. Make sure you have a short and succinct title for tables (or figures) and put in any footnotes for clarification. Figures or photographs are a way to make the case study visually interesting and self explanatory. Again, do not put data that is in the tables or figures in the text and only include data that is necessary for the case study. Colour photos, although expensive to print, will be allowed providing that they are necessary to make a point and that no superfluous colour figures are included, Colour photos are essential in cases where haematology or histology findings are necessary for the case study.

In conclusion, it is hoped that the above brief guidelines on writing case studies will encourage members of our profession to submit to the Journal. Next time you are about to present a case study at a SIG or other scientific meeting, think simultaneously about writing it up for the Journal. If published, your interesting case study will reach a much wider audience (readership $\pm$ 2,000) that at a scientific meeting. Additionally, you can earn valuable CPD points and, if a financial member of the Institute, be eligible for the best case study prize ($300). You will also be contributing to the world wide literature. Finally, READ THE INSTRUCTIONS FOR AUTHORS before submitting (www.nzimls.org.nz).

References

1. Naran S, Lallu S, Kenwright D. Pulmonary mucormycosis diagnosed by brushing cytology. A case report. *N Z J Med Lab Sci* 2008; 62: 32-4.
2. Hemmady S. Adrenal carcinoma: a case study. *N Z J Med Lab Sci* 2009; 63: 48-50.
3. Siebers R. Authorship (and non-authorship) criteria and new author requirements. *N Z J Med Lab Sci* 2008; 62: 3.
4. Siebers R, Holt S. Accuracy of references in five leading medical journals. *Lancet* 2000; 356: 1445.
5. Siebers R. Data inconsistencies in abstracts of articles in Clinical Chemistry. *Clin Chem* 2001; 47: 149.

Letters to the Editor

Malaria and HIV

It is always a pleasure to read the Journal but in the November 2009 issue the article on the prevalence of malaria and anaemia among HIV-infected patients is misleading and misinforming (1). The opening statement of the discussion section "*Malaria may be helping to spread the HIV virus that causes AIDS…*" is wrong and misinforming readers of the Journal.

It is possible that AIDS in HIV-positive persons can be exacerbated by a malaria infection but malaria itself has nothing to do with HIV. If the authors are sure that malaria carries the HIV virus and spreads as in their statements, then they need to state the species of *Plasmodium spp* and the vector mechanics that lead to the transmission.

Reference

1. Akinbo FO, Okaka CE, Omoregie R, Mordi R, Igbinuwen O. Prevalence of malaria and anaemia among HIV-infected patients in Benin City, Nigeria. *N Z J Med Lab Sci* 2009; 63: 78-80.

Owen Tafirenyika Mandisodza, Msc, MLSc
Section Head-Mycology
LabTests, Auckland

The corresponding author replies

I feel Mandisodza has misinterpreted the statement in question. The statement was referenced from a WHO technical report (1). The report states that malaria increases the risk of HIV transmission. The explanation given was that malaria results in anaemia which may lead to frequent blood transfusions, which is a potential risk for HIV infection.

The statement is not a consequence of our research, but a WHO technical report. I hope this explanation satisfies Mandisodza's queries.

Reference

1. WHO. Malaria and HIV/AIDS. Interactions and complications. Conclusions of a technical consultation convened by WHO, 23-25 June 2004; World Health Organisation. WHO/HIV/2004.08.

Frederick O Akinbo, PhD, FIMLS
Department of Pathology, University of Benin Teaching Hospital
Benin City, Nigeria

Book reviews

Diagnostic Surgical Pathology of the Head and Neck (2d ed.)

Edited by Douglas R. Gnepp. April 2009: 1205 p
ISBN-978-1-4160-2589-4
Price: A$590.00
Available from: Elsevier Australia (shop.elsevier.com.au) or your medical bookshop.

The purpose of this book is to provide a comprehensive book that covers the full range of surgical pathology with an emphasis on differential diagnosis and the more problematic areas in head and neck pathology. The text is aimed at Surgical or Oral Pathologists, ENT surgeons, Oral and Maxillofacial Surgeons, and anyone interested in the field of head and neck pathology.

My initial thought on looking at the table of contents was that there was a strong likelihood of repetition, particularly as 32 contributors were involved in writing the book. Excellent editing has brought the chapters together in an extremely cohesive way and where repetition has occurred it is due to regional or organ specific reasons. Incidentally a number of the internationally recognized contributors have been involved in other important texts in their field.

The first chapter on precancerous changes of the Upper Aerodigestive System (UADT) is an important starting point. The review of early neoplastic change in UADT recognizes the traditional site based lesions and presents a balanced representation of the current understanding of pathology and pathobiology of preinvasive neoplasia of UADT. It places emphasis on aspects intended to attempt standardization of terms, definitions and classifications of pathologic and molecular changes associated with early neoplastic transformation. It also balances this with clinical correlations and studies of progression of lesions. The end of the chapter summarizes molecular genetic studies of selected markers involved in carcinogenesis.

The second chapter on squamous cell carcinoma of UADT thoroughly examines epidemiology and each individual site in detail. Importantly the lip and intraoral sites are considered separately so the data on prevalence and survival rates is meaningful. In the context of addressing challenging lesions, the unusual variants are addressed in detail.

A shift to specific regional pathology takes place in chapter 3 addressing lesions of nasal cavity, paranasal sinuses and nasopharynx beginning with inflammatory and related conditions, hamatomas, cysts and pseudocysts and epithelail and mesenchymal benign and malignant neoplasms. As many of these require further extensive discussion in other chapters less detail is provided in this chapter. This is not a shortcoming, but a reflection on how one might approach reading about a specific entity in these sites.

Chapter 4 has a similar approach to lesions of the oral cavity. In the course of preparing this review I used it when unusual specimens were referred in. One minor shortcoming was on checking for the recently described gingival lesion called in this text "juvenile inflammatory papillary gingival hyperplasia" or juvenile spongiotic gingival hyperplasia no clinical image or photomicrograph was available and it was incorrectly referenced. Other relatively recently described entities e.g. Bisphosphanate-associated osteonecrosis have both clinical images and photomicrographs in addition to the text. The section on osteonecrosis also includes neuralgia-inducing cavitational osteonecrosis (NICO), which is a contentious issue. I am not surprised at the inclusion of this entity as one of the chapter's co-authors is a major advocate of NICO and it is included in other textbooks he has co-authored.

A similar approach follows in Chapter 5 (hypopharynx, larynx and trachea). Understandably, a number of diseases have also been discussed in previous chapters so much of the discussion is site specific in terms of differential diagnosis and management.

Chapter 6 extensively covers non-neoplastic and neoplastic salivary gland and to a lesser extent lacrimal gland disease. The differential diagnosis sections for several uncommon salivary gland tumours received in our laboratory recently was useful for arriving at a diagnosis. Immunoprofiles and genetics are included where appropriate.

Other chapters include other regions and organs of the head and neck i.e. thyroid and parathyroid glands, soft tissue tumours, lesions of bone, odontogenic cysts and neoplasms, cysts of the neck (and neck dissection), ear lesions, haemopoietic lesions, cutaneous lesions. Fine needle aspiration biopsy forms the final chapter.

All chapters have an extensive reference list divided into the topics as covered in each chapter. The chapters have been updated to include recent advances. The book is an, "Expert Consult" title enabling the owner to access the full text, download the illustrations (over 1700 photomicrographs, clinical photographs, tables and diagrams), follow links to the references and therefore abstracts via PubMed. Access is via the Web, so there is no need for a CD-ROM. The quality of the illustrations is excellent and serve to highlight the diagnostic features described in the text. Most are colour, which is just one of the improvements over the first edition. The book includes up to date ancillary tests including immunohistochemistry and molecular genetics to assist in diagnosis.

Two appendices are included. The first summarizes the UICC TNM classification and staging system, while the second provides guidelines for the dissection of head and neck specimens.

This book is useful for anyone involved with head and neck pathology. Trainees will gain an understanding on current knowledge, and also the background to that current knowledge. All pathologists receiving head and neck specimens and the surgeons providing the specimens will find this book invaluable.

Norman A. Firth, BDS, MDSc, FRACDS, FFOP(RCPA)
Senior Lecturer/Oral Pathologist
University of Otago, Dunedin

Lab Notes: Guide to Lab and Diagnostic Tests (2nd Ed.)

By Tracey B. Hopkins. February 2009
ISBN-13: 9780803621381
Price: A$46.00; NZ$55.00
Available from: Elsevier Australia (shop.elsevier.com.au) or your medical bookshop.

This is a very compact portable reference manual designed as an information resource for clinicians. The information, although brief in nature, is set out clearly and is systematic in format detailing over 400 lab tests with reference to abnormal findings, normal ranges, panic levels, potential complications, red flag alerts, pre and post test care, critical results, as well as ECG and ABG interpretation.

This manual details the procedures of patient identification, correct collection of samples for laboratory analysis, preparation

of equipment,venipuncture methodology (including the use of a syringe, IV infusion sets and skin puncture) the selection of blood collection site and the use of appropriate vacutainers.

Urine sample collection, faecal occult blood samples, sputum sample, nasopharyngeal swabs and wound swabs are dealt with independanly in an intelligent and concise approach. Diagnostic tests are discussed alphabetically in terms of appropriate sample, principle, and interpretation as well as normal range.

Besides laboratory investigation, this little book is very informative in the diagnostic procedures of X-ray, CT/MRI scans, nuclear scans, ultra sounds and other clinical diagnostic testing. Each section is clearly indexed allowing the reader to locate information both rapidly and with ease. The rear of the book is devoted to the presentation of a variety of diagnostic panels that simply help to direct the clinician towards a positive diagnostic outcome.

This book is an excellent pocket reference companion for all clinicians. It is informative and accurate as well as waterproof and robust in design. As with any science, and medical science is no exception, advances in diagnostic approach and procedure will continue to rapidly progress, and therefore this manual like all others will require regular updating. It is thoroughly recommended for all health professionals who contribute to the diagnosis of active disease and its effective management.

Philip Wakem, MMLSc
Pacific Paramedical Training Centre, Wellington

Mosby's Diagnostic and Laboratory Test Reference (9th Ed.)

By Kathleen D. Pagana and Timothy J. Pagana. October 2008
ISBN-13: 9780323053457
Price: A$87.00; NZ$105.00
Available from: Elsevier Australia (shop.elsevier.com.au) or your medical bookshop

This small but comprehensive reference manual is systematically presented in terms of laboratory and diagnostic testing, allowing for quick easy access to essential information. Each described test is presented in a consistent format and each test begins its description on a new page. The format is a logical sequence of informative material and is presented under the following headings where appropriate:

- Name of test
- Type of test
- Normal findings
- Possible critical values
- Test explanation
- Contraindications
- Potential complication
- Interfering factors
- Procedure and Patient care
- Before during and after testing
- Abnormal findings
- Blank note space

The first section of this reference is dedicated to sample collection and test preparation which is well documented and clearly written. The body of the manual is devoted to the desciption of an extensive range of well established laboratory and non-laboratory diagnostic testing presented alphabetically, with the inclusion of 41 new tests and 42 coloured illustrations .

The rear of the manual is dedicated to four apendices (A-D) .
"A" lists the tests by body system
"B" lists the tests by type, ranging from blood to X-Ray
"C" describes the tests appropriate for the diagnosis of a particular disease
"D" lists a range of symbols and units of measurement commonly used in test description.

Inside the front cover and continuing on the inside back cover is a list of test abbreviations commonly used in both laboratory, non laboratory and clinical settings.

This manual is an excellent information resource, suitable for use by all health professionals. It is formatted extremely well and information is easily accessible and logically presented .Thoroughly recommended.

Philip Wakem, MMLSc
Pacific Paramedical Training Centre, Wellington

A Guide to Laboratory Investigations (5th Ed.)

By Michael McGhee. March 2008.
ISBN-13: 9781846192104
Price: A$65.00; NZ$78.00
Available from: Elsevier Australia (shop.elsevier.com.au) or your medical bookshop

This manual discusses laboratory testing in a more interpretative style. It is written by a GP and is a valuable resource for doctors, nurses and other health professionals who require in- depth laboratory result interpretation to tests that have been requested.

The Guide is divided into 6 sections, headed by a comprehensive glossary of medical abreviations and quantitative units commonly used by the author throughout the sections themselves.

Section 1 is dedicated to Haematology and discusses Haematological testing and interpretation, covering FBC parameters both in health and disease, coagulation disorders and haemoglobinopathies.

Section 2 is dedicated to Microbiology and discusses organisms under gastrointestinal and urogenital headings with reference to isolation and treatment. Fungal infections, CSF, selected parasites, streptococcus, toxoplasmosis, rubella and chickenpox are also discussed in terms of result interpretation.

Section 3 is dedicated to fertility and pregnancy testing and discusses female hormone profiles, pregancy tests, semen analysis rhesus grouping and alpha fetoproteins.

Section 4 is dedicated to Rheumatology and immunological test interpretation, and Section 5 is dedicated to Biochemistry and associated interpretation

Section 6 covers such topics as drug monitoring, cervical smears and urinalysis and faecal occult blood.

Although this guide appears at first glance to be simplistic in content and presentation, it is actually a comprehensive, easily understandable diagnostic tool that clinicians should find particularly helpful in the diagnosis treatment and overall management of their patients.

Philip Wakem, MMLSc
Pacific Paramedical Training Centre, Wellington

Med-Bio Journal Award

Med-Bio, a division of Global Science & Technology Ltd. offers an award for the best article published during the calendar year in the *New Zealand Journal of Medical Laboratory Science* worth $300. All financial members of the NZIMLS are eligible. The article can be an Original, Review or Technical Article. Excluded are Editorials, Reports, Fellowship Treatises or Case Studies (Case Studies are judged under the NZIMLS Journal Prize)

No formal application is necessary but you must be a financial member of the NZIMLS to be eligible. The Editor and Deputy Editor will decide in December which article is deemed worthy of the award. Their decision will be final and no correspondence will be entered into.

NZIMLS Journal Prize

Council of the NZIMLS has approved an annual Journal prize ($300) for the best case study published in the Journal during the calendar year.

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

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

Winner of the NZIMLS Journal prize for 2009 was Sujata Hemmady, Department of Chemical Pathology, LabPlus, Auckland for her article "Adrenal carcinoma: a case study. N Z J Med Lab Sci 2009; 63 (2): 48-50

Massey University – NZIMLS Student Award

The recipient of the NZIMLS award for top student in the 3rd year of the Massey BMLSc in 2009 was Kayla Riley-Regan. Kayla is currently a fourth year student at Medlab Central in Palmerston North.

I was born and raised in Wanganui however, shortly after I moved to Palmerston North, my family moved to Tauranga. In 2006, I graduated from Wanganui Girls' College. During my time there, I developed a passion for science, rowing, cake decorating and photography. Like most high school students, I was unsure what I was going to do once I finished school. I had looked into a few courses offered by various Universities but made my final decision following a conversation with one of the girls from school. Her father had spent many years working in the Wanganui Laboratory and seemed to thoroughly enjoy the work.

Being the first of my family to attend University I initially found the task a little daunting, however, as I progressed through my years at Massey I began to enjoy the challenge presented by tertiary study. I was fortunate enough to be employed by Medlab Central during my second year starting out in specimen reception before moving into Microbiology. After working a few weeks, I was certain a career in this line of work was ideal for me. The team at Medlab Central have been (and are) great - they are always available to offer guidance or advice should you need it. Deciding what subjects to take in my fourth year was a difficult decision to make. After weighing the pros and cons of each major for many weeks (or possibly months) I decided to go with Microbiology and Transfusion Science.

I currently live in Palmerston North with my fiancé Ross and two cats (Dusty and Chester). We don't have a lot of family here however we have made some great friends since moving here. Following the completion of my course, I hope to gain some work experience in a NZ laboratory before traveling and working overseas. However, this may be easier said than done as my partner is dead set against moving overseas (but I figure I can talk him around!). It has been a great privilege to receive the NZIMLS scholarship and I look forward to an exciting and rewarding career.

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Life needs answers

Journal questionnaire

Below are 10 questions based on articles in the April 2010 issue of the Journal. Read the articles fully and carefully, most questions require more than one answer.

Answers are to be submitted through the NZIMLS web site. Make sure you supply your correct email address and membership number. It is recommend that you write your answers in a word doc and then cut & 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 resubmitting from a computer or system 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 can only claim 5 CPD points. You can not then claim additional CPD points (2/article) for reading the articles from which the questions were derived.

The site will remain open until Friday 2nd July 2010. You must get a minimum of 8 questions right to obtain 5 CPD points.

Journal questions

1. What are the reasons for the significant differences in sensitivity amongs PCR methods for TB bacilli.
2. What is the gold standard for diagnosis of tuberculous pleural effusion, but what are potential problems.
3. In patients with tuberculous pleural effusion what is the positivity of pleural biopsy culture and what does histological examination reveal.
4. Apart from tuberculous pleural effusion, in what other conditions can elevated levels of adenosine deaminase be found.
5. What do do the initials of HELLP syndrome stand for.
6. In HELLP syndrome, what is maternal morbidity mostly associated with.
7. What are the symptoms of HELLP syndrome.
8. What contributes to the significant maternal and fetal morbidity and mortality involved with HELLP syndrome.
9. What is the major concern of the use of point-of-care glucose meters and what has previously compromised their performance.
10. Which glucose meters showed the closest correlation with the plasma hexokinase reference method and what additionally did they demonstrate.

Questions and answers for the November 2009 Journal questionnaire

1. What is the possible reason for the high concentration reading for white cell count, red cell count and protein by the Urisys 1800.
   May be related to the principle of measurement. A testing pad with a colour intensity developed that is slightly darker than a reference scale is classified into the same concentration range as the reference scale by manual readings.
2. What can cause blood-stained and abnormally coloured urine samples.
   Blood-stained specimens can be caused by bladder and/or kidney conditions while abnormally coloured urine may be a result of liver disease, or intake of certain medications or food.
3. What are white cell counts measured by the Urisys 1800 directly proportional to, and what does this mean.
   They are directly proportional to the amount of esterase present in the urine, which means both intact and lysed white cells are counted.
4. What factors can potentially influence the measurement of white cells by the Urisys 1800.
   False negative white cell concentration may occur due to the presence of leukocyte esterase inhibitors. Glycosuria and/or ketonuria can result in falsely low measurements. High urine protein and usage of certain oxidizing drugs may lead to a falsely negative white cell count. False positive white cell counts are usually caused by contaminants, such as high numbers of epithelial cells.
5. How is the inhibitory effect of vitamin C in the measurement of red cells by the Urisys 1800 minimised.
   By the incorporation of iodate in the test region on the test strips.
6. What was the limitation of the Urisys 1800 study and how may this be overcome.
   The sample size needed to be larger and include samples from random community patients to ensure a better interpretation of the entire population with minimal bias.
7. Which age groups showed the highest and lowest prevalence of anaemia in HIV-infected patients.
   The 20-29 and 50-59 year age groups showed the highest prevalence of anaemia with the 40-49 year age group showing the lowest prevalence of anaemia.
8. What is the possible reason for HIV-infected adults to develop malaria.
   HIV-infected patients are at a higher risk for malaria because of their weakened immune systems.
9. What has been reported as a mechanism of anaemia among HIV-infected patients.
   Bone marrow suppression by the HIV virus.
10. What is associated with an increased risk of malaria among HIV-infected patients.
    A $CD4^+$ count of <200 cells/µl.

Chateau de Biochem

NZIMLS Biochemistry Special Interest Group Meeting

Saturday 12 June 2010
Chateau on the Park
189 Deans Avenue

CHRISTCHURCH NZ

Registration and coffee 9.30am
Finish 5pm approx
Dinner 7pm

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

Prizes for Best Presentation and Best First Time Presenter

Contact Sandy Woods
Specialist Biochemistry Dept
Canterbury Health Laboratories
PO Box 151, Christchurch
Sandy.Woods@cdhb.govt.nz

Online registration available at www.nzimls.org.nz

The Pacific Way

Greetings to you all and a very happy start to the new year. The PPTC had a very busy 2009, and 2010 will be no exception.

Courses held at our centre in late 2009

A course in Transfusion Science was held in November 2009 and five students from both the North and South Pacific regions attended. They were:

Victoria Wuatai (Cook Islands)
Iasko Ada (Pohnpei)
Kay Aliklik (Nauru)
Raymond Seule (Vanuatu)
Solomon Soakai (Solomon Islands)

The course was a great success and we are sincerely grateful to Jeanette Watson and her blood bank staff for the excellent tuition and practical training given to the students throughout the duration of the course.

Students and staff of the Transfusion Science Course

Courses to be held at our centre in 2010

This year, the PPTC has scheduled three courses to be held at its centre in Wellington New Zealand. The starting dates are as follows:

Haematology and blood cell identification: 1st – 26th March
Biochemistry: 14th June - 2nd July
Blood bank: 1st - 26th November

A special note to Charge Technicians or Heads of Departments!

If you have nominated members of your staff to attend one or more of the above courses, could you email their names to the PPTC as soon as you are able so as we can gain an idea of numbers who wish to attend as well as begin the appropriate arrangements. You will need to apply for funding from either NZAID, or other nominated sources, as soon as possible to ensure your own students do not miss this opportunity.

The POLHN distance learning programme

A final opportunity to complete outstanding POLHN distance learning modules for the PPTC's Diploma in Medical Laboratory Technology will be offered 2010. The five modules include Biochemistry, Haematology, Microbiology, Transfusion Science and Immunology.

Starting dates for each of the modules is scheduled as follows

POLHN 015 Haematology	- 01 March 2010
POLHN 026 Biochemistry	- 03 May 2010
POLHN 017 Blood Bank Technology	- 05 July 2010
POLHN 016 Microbiology	- 06 September 2010
POLHN 018 Immunology	- 01 November 2010

2011 will see a change in course requirements in terms of the practical element of the POLHN programme. As a result of on-going evaluation of the POLHN programme, it has been proposed by the PPTC that, because the current practical element attached to each module is known to be brief and because the final qualification is now a Diploma, log books will be introduced for each module. These will have to be completed before the Certificate of Attainment for that module can be awarded. The content of each log book is based on routine procedures currently employed within each discipline and as each log book task is completed, signoff by the Charge Technician or Head of Department will be required until the log book is fully completed. Instead of a six week completion time for each module as it has been in the past, each module will now take at least FOUR months and because of this, fewer modules will be offered over the academic year. In other words, instead of the Diploma being a one year course, it will now take up to two years to complete.

Each log book not only details the practical tasks to be carried out, but also contains a great deal of useful information associated with each procedure. The log books which are currently under design will act as an information resource once completed by the student. Each completed log book will be returned to the student once audited by the PPTC so as it can be used by the student for future reference.

In 2009, the Laboratory Management POLHN course was launched and 25 students registered to undertake this study. Students have received the learning material from the PPTC and we are slowly now receiving answers to module questions. This year, 2010, a newly developed STI course will be offered by the PPTC through POLHN Distance Learning.

Pacific travel

Manila, Palau and Yap: 2009

Phil travelled to Yap in the Federated States of Micronesia and Palau for two weeks in October. The reason for visiting was to assess the current programmes that the PPTC provides to these and the other Pacific nations, especially the EQA programme. The visit also gave Phil the opportunity to identify operational needs that each laboratory currently has in the pursuit of a Quality Management System. He firstly stopped in the Philippines to visit WHO in Manila and then travelled through Guam to Palau and Yap to meet with laboratory staff. It was fortunate he was able to teach during his stay which was very welcomed.

Samoa 2009

On the 1st November 2009, both John and Phil visited Samoa to meet with the laboratory staff. During their stay they were given the opportunity to discuss EQA as well as future directions of the PPTC's POLHN distance learning programmes in which large

numbers of students are currently registered. John was also able to introduce procedures for the detection of Extended Spectrum Beta Lactamase organisms (ESBL's) which up to that point had not routinely been tested for. Phil visited Haematology frequently to conduct Haematology morphology workshops as well as to cool off as it was perceived that the Haematology laboratory had the largest air conditioning unit in the entire lab. The educational contribution made by John and Phil was gratefully received during their weeklong stay.

They were also able to visit the area devastated by the Tsunami on the South Coast and meet with Red Cross staff who were making an enormous contribution towards the welfare of those who had suffered as a result this catastrophe.

Demolished buildings post tsunami

Red Cross staff involved in the distribution of aid

Fiji 2010
John and Phil travelled to Fiji to participate in a meeting which focused on the implications of current and future availability of point of care tests for sexually transmitted infections in Pacific Island Countries and Territories. Those present consisted of health professionals from many organisations such as WHO, SPC and UNICEF as well as experts and co-ordinators involved in the diagnosis and management of STI in the Pacific. The objective of this meeting was to develop a toolkit specifically designed for Health Ministries in the Pacific which could be used by each Ministry as an implementation tool or guide if there was movement towards rapid POC testing in outer clinics or remote locations. The meeting was very interesting and successful with a positive outcome in the framework development of the toolkit.

Proposed visits

In April John is travelling to Vanuatu as a UNFPA consultant to evaluate laboratory procedures used in the diagnosis of HIV and STI's.

In May Phil is scheduled to visit, Pohnpei, Chuuk, and Kosrae in the Federated States of Micronesia and Majuro in the Marshall Islands for nearly 3 weeks to perform quality assessments and discuss training programmes.

Advertisers in this issue

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New Zealand Institute of Medical Laboratory Science

NZIMLS Annual Scientific Meeting
Bay of Islands, Northland 2010

The Diversity of Pathology

NZIMLS / NZSC Annual Scientific Meeting
Copthorne Hotel & Resort Paihia, Bay of Islands, 23-27 August 2010

- Set in the historic Bay of Islands
- Free buses Monday and Tuesday from Auckland to Paihia via the magical Waipoua Forest Conservation Estate to see the famous "Tane Mahuta" (Lord of the Forest), New Zealand's largest known living kauri tree. Returning Friday.
- Workshops Tuesday 23rd August
- Plenary plus concurrent sessions Wednesday 24th – Friday 27th
- Proffered papers wanted!
- For further information contact Ross Hewett at LabPLUS, Auckland City Hospital or on rossh@adhb.govt.nz

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