Volume 50 Number 2 May 1996 ISSN 1171-0195

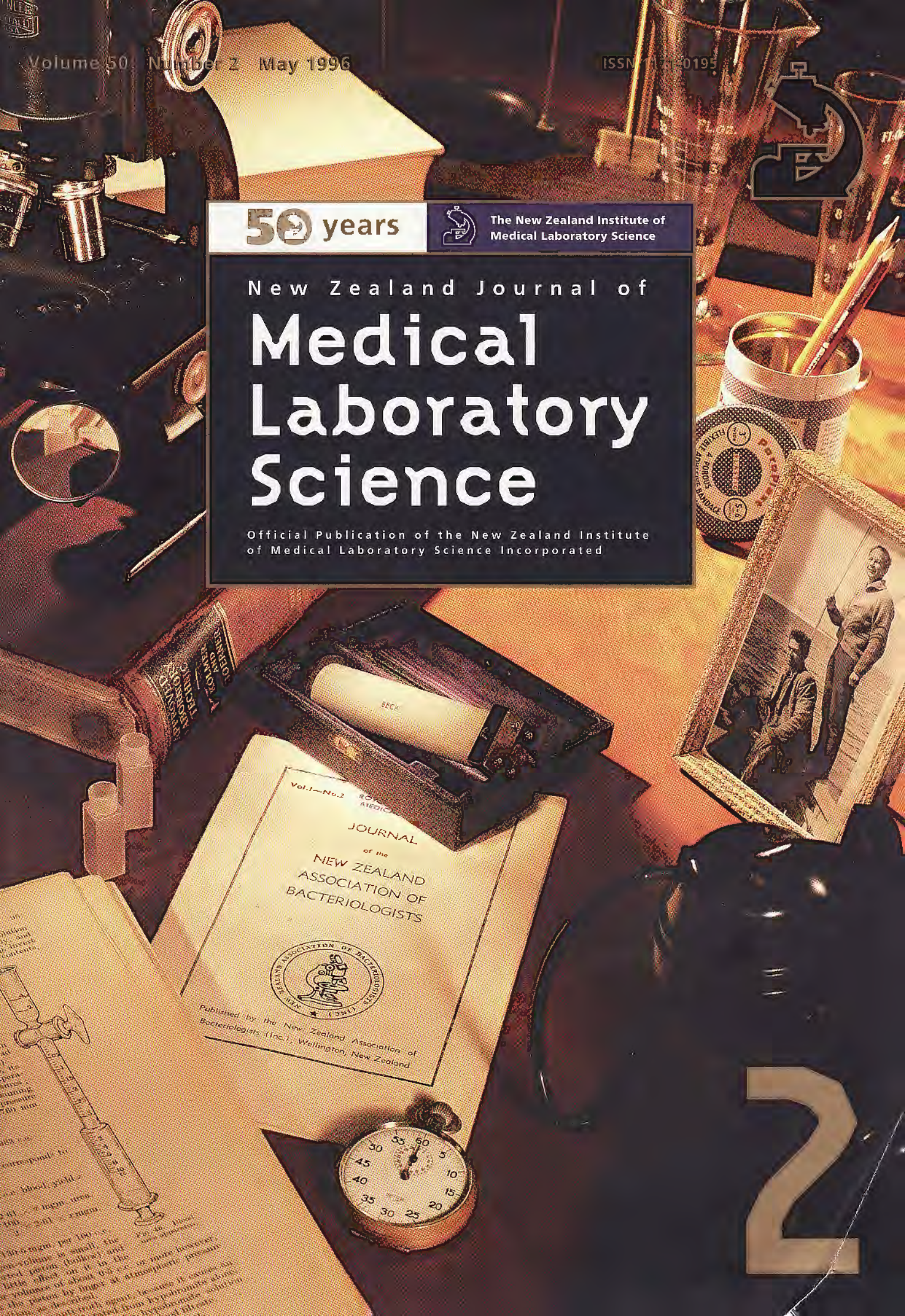
50 years

The New Zealand Institute of Medical Laboratory Science

New Zealand Journal of

Medical Laboratory Science

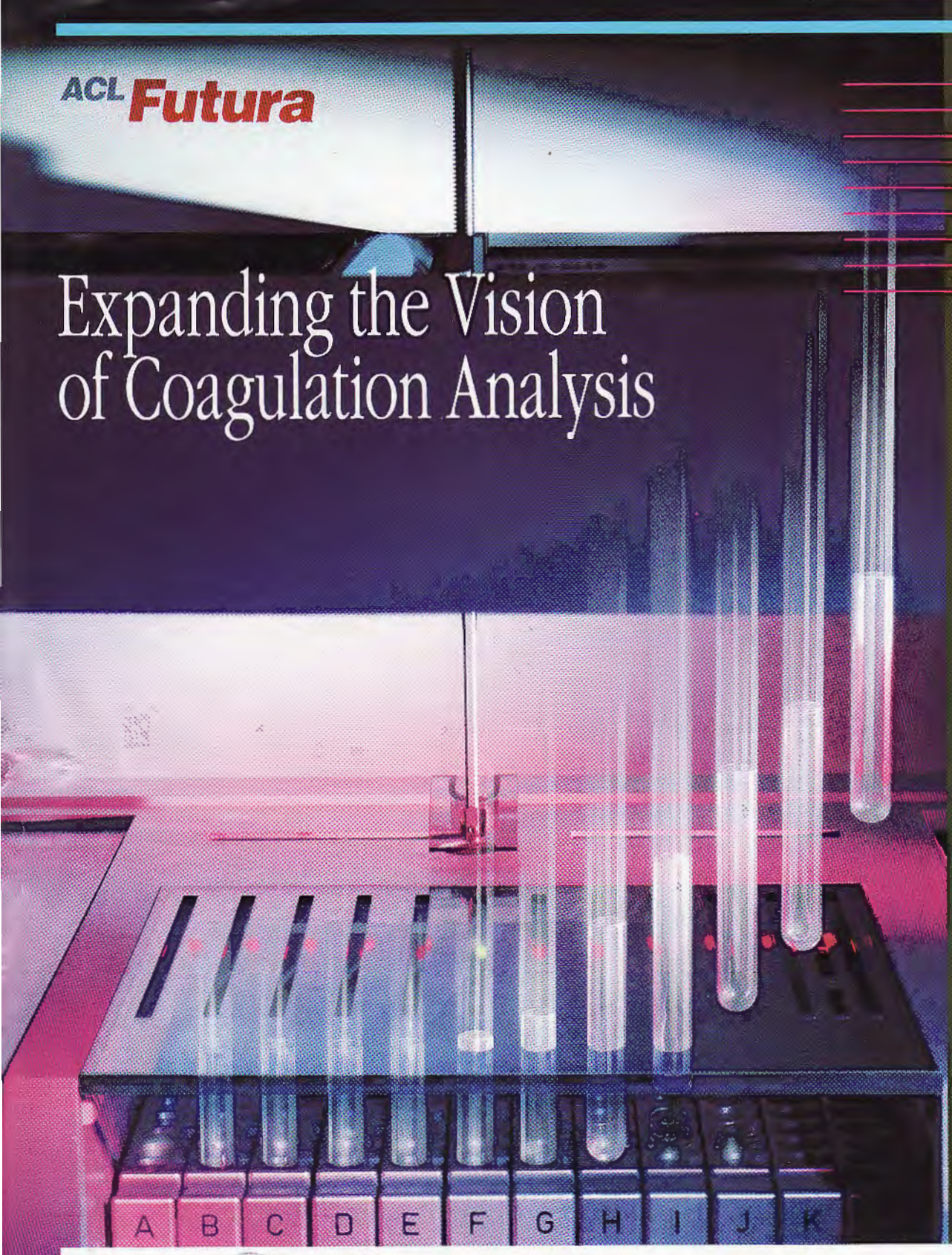
Official Publication of the New Zealand Institute of Medical Laboratory Science Incorporated



ACL Futura

Expanding the Vision of Coagulation Analysis

Coagulation



IL continues its tradition of leadership in coagulation analysis with the ACL Futura™ system. This versatile random-access analyser provides both turbidimetric (clotting) and absorbance (chromogenic) channels on board.

With 16 channels reading simultaneously, throughput is dramatically improved.

ACL Futura is a PC-based, menu-driven system that is as easy to use as it is flexible. The system holds up to 120 samples for true walkaway productivity. Samples can be added continuously during analysis. You can also insert emergency (STAT) samples at any time for priority testing.

Two precision pipettors equipped with liquid sensors aspirate and dispense all liquids. ACL Futura holds up to 34 reagents in their original vials.

Its powerful software provides DMS capabilities (patient data storage), bidirectional communication with a mainframe, a Quality Control program and reaction curve display.

With an IL system you're assured of the most sophisticated, reliable diagnostic tools available. In addition IL analysers and reagents are perfectly matched for consistently superior analytical performance.

Contact Coulter on Free Call 0800 442 346 or Free Fax on 0800 442 347.
Coulter Electronics (NZ) Ltd,
PO Box 20266, Glen Eden, Auckland.

COULTER

Instrumentation Laboratory

Partners for Excellence

New Zealand Journal of
Medical Laboratory Science
Volume 50 Number 2
May 1996
ISSN 1171-0195

Current Comment

The Industry and the Laboratory – Our Changing Roles

Ross Hewett
Manager, Laboratory Systems Division
Boehringer Mannheim NZ Ltd
Auckland

NZ J Med Lab Science 1996 50(2) 46-47

The role of the Medical Laboratory Scientist has changed in the last 15 years in line with the advances in technology. This changing role has been to some, an opportunity to create new careers in commerce, whilst still retaining their knowledge and skills as a Technologist.

Those of us who took this option have seen the relationship between the laboratories and commercial industry change. Whereas before, the expectation of commerce was to provide glassware, chemicals and basic equipment, nowadays the expectation is quite different. We need to employ specialist technologists in defined areas as sales representatives, application specialists, operator trainers, and product managers. Laboratory personnel seek advice from industry on laboratory design, equipment options both medium and long-term, management of work flow and department reorganisation.

As cuts to Lab personnel occur, the reliance on industry increases in areas of technical support and troubleshooting.

With those companies who have adopted the philosophy of converting Medical Laboratory Technologists into sales people rather than the opposite, what can the laboratory expect from them.

They are one of the laboratory's greatest assets. With the modern sales liaison, the laboratory should utilise their financial and technical expertise to make cost-efficient purchase decisions.

The old aggressive sales stereotype has given way for the most part to educated professionals who use a softer touch and are supported by greater resources.

Today's breed generates business by meeting customer needs, especially in areas of reliability and ongoing after sales service. If they don't have immediate answers, they are supported by a top level team of specialists who do, both nationally and globally.

Some laboratory technologists may argue that sales reps have a limited perspective, that they are driven by getting the sale and avoid responsibility for the consequences. Such issues as near patient testing and technology replacing technologists are such examples.

The thrust of the current health reforms and the advances of technology have these consequences, not sales reps. It's a case of blaming the messenger rather than managing the change.

Laboratory technologists and managers argue that they are the experts, as there is no substitute for day to day laboratory experience. It must be remembered that many reps have that same background.

Some of the country's best technologists have responded to the call of a career in marketing. They are being targeted by companies who can fully compensate them for their expertise and talent.

A company's employees can help a laboratory in many ways. The major functions and areas are:

1. Sales Representatives: They are the company's eyes and ears (and in some cases mouthpieces). They will tell you about new products and inform their company of your needs. This influences new product development and support functions. A well prepared salesperson and laboratory technologist will have a productive win/win sales encounter. They keep you informed and may give you leads to information from other sales sources.
2. Application Support Staff: Companies are taking more responsibility for providing greater levels of education and technical support. These people train the laboratory staff on new instrumentation and provide the ongoing support. They keep you updated on technical changes and troubleshooting remedies.
3. Product & Marketing Managers: They are responsible for the need or release of new products. They facilitate customer seminars and user group meetings. A Product or Marketing Manager will identify key decision makers or opinion leaders for new or innovative tests or products. They will keep a lab informed as to the long-term development of new products and strategic direction of technology.
4. Customer Services can provide some of the above, however their prime function is to easily facilitate the day to day business relationship between the laboratory and the company. Order processing, standing order delivery and product allocation are such functions. Companies can arrange loans and exchanges to keep labs supplied when back orders are delayed. Customer Services help with accounts or problems with deliveries or couriers.
5. Technical Service Engineers: As instruments become more sophisticated and technically complex, so does their maintenance. Gone are the days of technologists with screwdrivers pulling to bits flow analysers or spectrophotometers in typical Kiwi DIY fashion. Long-term reliability, up-time and reduced turnaround time are key requirements by laboratories as more work is loaded onto less equipment.

   Preventative maintenance and service agreements are critical ingredients to a modern automated laboratory. The equipment needs expert technicians to maintain and troubleshoot.
6. Managers of these companies need to know the overall ingredients that makes a commercial organization work in such a specialised and defined market. The mix of all the above functions, like any reagent formulation, must be optimised to provide both a satisfied customer and a financial result for the company. Whilst a background in Medical Laboratory Science is not essential, it certainly helps with overall company strategic planning and empathy with the market. Their opinion and knowledge to any laboratory is a resource too few take advantage of. They provide financial analysis to argue for new equipment or options for long-term

planning. They should be consulted about conferences because it's their final decision that makes company participation, and therefore financial support an option.

7. The final expectation a laboratory should have from a company is trust. A company should value its clients and care about the relationship it has with them. A company's employees should keep all information about a client confidential, especially in this new era of laboratory competition. Without a lab's loyalty, trust and confidence, a company, regardless of who they are, will not survive in a competitive market.

In conclusion, the "us and them" attitude towards company personnel is changing in the most part to one of mutual consultation and partnership. From the day to day running of a laboratory to the long-term strategic direction of Medical Laboratory Technology, the scientific industry has a significant influence.

As technicians progressed to technologists and then to scientists, so have commercial travellers progressed to the trade and then to a professional scientific industry. To some it may be semantics, to others however, it is an evolutionary process that reflects the growing up of a profession both in the workplace and in commerce.

AUSTRALIAN INSTITUTE OF MEDICAL SCIENTISTS

National Science Meeting

6 – 11 October 1995

On behalf of the Organising Committee, I extend a warm invitation to all Medical Scientists, Technical Officers and interested parties, to attend the above conference to be held in the Convention Centre in Adelaide, South Australia.

The conference theme is Aboriginal Health and will cover this and many other related topics and scientific endeavours.

Persons interested in taking part in the scientific programme by presenting a paper or poster should contact:

Mr John Stirling
c/- Histopathology Dept
Flinders Medical Centre
BEDFORD PARK SA 5042

For further information please contact:
SAPMEA Conventions
80 Brougham Place
NORTH ADELAIDE SA 5006

Brian Matthews
Chairperson Organising Committee

NEW ZEALAND JOURNAL OF MEDICAL LABORATORY SCIENCE

The New Zealand Journal of Medical Laboratory Science is published quarterly (March, May, August & November) on behalf of the New Zealand Institute of Medical Laboratory Science (Inc) by Institute Press Ltd, Auckland.

The Journal is indexed in the Cumulative Index to Nursing and Allied Health Literature (CINAHL).

Subscriptions:

Subscriptions to the Journal for non-members requiring delivery in New Zealand is $NZ33.00 for 1 year surface mail paid. Subscriptions to the Journal for non-members requiring delivery overseas is $NZ39.60 for 1 year surface mail paid. All subscriptions except for single issues are due in February. Single issues are $NZ12.00 Surface mail paid. Members of the NZIMLS should send their enquiries and address changes directly to the Executive Officer of the NZIMLS, PO Box 3270, Christchurch.

Advertising:

Advertisement bookings and enquiries should be addressed to the Advertising Manager: Trish Reilly, 48 Towai St., St Heliers, Auckland 5. Phone: (09) 575 5057.

Editorial:

All editorial matter, including submitted papers, press releases and books for review should be sent to the Editor: Rob Siebers, Department of Medicine, Wellington School of Medicine, PO Box 7343 Wellington South. Phone: (04) 385 5999 (Ext: 6838). Fax: (04) 389 5725, E-mail: rob@wnmeds.ac.nz.

Contributors and advertisers are responsible for the scientific content and views. The opinions expressed in the Journal are not necessarily those of the Editor or Council of the NZIMLS.

Information for Contributors:

The Journal publishes original, review, leading & technical articles, short communications, case reports and letters in all disciplines of Medical Laboratory Science as well as related areas of interest to Medical Laboratory Scientists (eg) epidemiology, public & community health, education, ethics, computer applications, management, etc. All papers published will be in the form known as the "Vancouver Style" or Uniform Requirements for Manuscripts Submitted to Biomedical Journals. Concise details are listed below while full details may be found in the *NZ J Med Lab Science* 1991; 45 (4): 108-11 or from the Editor.

Papers submitted to the Journal are refereed and acceptance is at the discretion of the Editor. Papers with substantive statistical analysis and data will be reviewed for appropriateness by the Statistical Adviser. No undertaking is given that any article will be published in a particular issue of the Journal. The copy deadline for each issue is the first of the month prior to the month of publication.

Manuscripts:

Submitted papers (**in duplicate**) should be typewritten, in double spacing throughout on one side of A4 paper. Generally each component of the manuscript should begin on a new page in the following sequence.

* **Title of paper**, authors (including first name and qualifications), and institution(s) where the work was carried out. Address for the corresponding author should also be given.

* **Abstract and keywords**. Abstracts should be structured and contain concise and precise information regarding the study's **Objective(s), Method(s), Result(s)** and **Conclusion(s)**. List up to 4 keywords using *Index Medicus* medical subject headings.

* **Text**, in the order of **Introduction, Materials and Methods, Results, Discussion and Conclusion**.

* **References** should follow the style adopted by the US National Library of Medicine as used in *Index Medicus*. Refer to papers in recent issues of the Journal for guidance (or see *NZ J Med Lab Science* 1991; 45 (4): 108-11). Authors are responsible for accuracy of all references.

* **Illustrations** must be provided with a suitable legend typed on a separate sheet. Graphs should be 2-3 times larger than they would appear in the journal and contain a minimum of lettering Legends for these should also be typed on a separate sheet. Photographs should be original sharp, glossy black & white prints. Authors wishing to submit colour photographs must contact the Editor in the first instance.

* **Tables** should be typed on a separate page complete with a title at the top and footnotes at the bottom. The tables should be numbered as they appear in the text and must *not* contain vertical lines.

* **Acknowledgements** should be made to people and/or organisations who have made substantial contributions to the study. Authors are responsible for obtaining consent from those acknowledged. Financial contributions towards the study from granting bodies or commercial organisations must be stated.

Two copies of the manuscript are to be addressed to the Editor NZ J Med Lab Science, c/- Department of Medicine, Wellington School of Medicine, PO Box 7343, Wellington South, together with a letter from the corresponding author stating that the work is original, is not under consideration for publication elsewhere, and in the case of multi-authorship that all authors have contributed directly to the planning, execution, analysis or to the writing of the paper.

EDITORIAL

The TH Pullar Memorial Address

Rob Siebers, Editor

Each year the NZIMLS Council invites a person who has made a significant contribution towards Medical Laboratory Science to deliver the TH Pullar Memorial Address at the Annual Scientific Meeting. This prestigious address was initiated by council upon recommendation by R. Kennedy and K. Fletcher in April 1967 in honour of Dr. TH Pullar who passed away on August 29, 1966. The proposal was that this Memorial Address was to be given at Conference, that prominent pathologists at that time be invited to give the Address, and that it be published in the Journal. From 1967 until 1974 a number of pathologists (1967:Dr FW Gunz; 1968: Dr PP Lynch; 1969: Dr SE Williams; 1970 Dr NP Markham; 1971: Dr WL Kenealy; 1972: Dr PB Herdson; 1973: Dr DT Stewart) presented the TH Pullar Memorial Address. From 1974 this Address was delivered predominantly by members of our Institute, nm. Harry Hutchings, John Case, Desmond Philips (twice), Rod Kennedy, Bob Allan, Janet Marsland, Brian Main, Burt Nixon, John Whiteley (President AIMLS), Jan Parker, Ron MacKenzie, Barrie Edwards and Walter Wilson.

So who was Dr TH Pullar and why does the NZIMLS honour his name each year at the Annual Scientific Meeting?

Thomas Henry Pullar was born in Auckland in 1907. His father was a General Practitioner and returned with his family to Scotland where Thomas was educated at George Heriot's School in Edinburgh. He subsequently attended the Universities of Glasgow and Sheffield, the latter where he graduated MB ChB with Honours in 1929.

He started his professional career as Biochemist to the Sheffield Royal Hospital in 1930, and then spent 2 years as Assistant Pathologist at the Sunderland Royal Infirmary obtaining the London Membership of the Royal College of Physicians there in 1933. For the next three years he was Clinical Pathologist at the Middlesex Mount Vernon Cancer Hospital, and then in 1937 moved to New Zealand to take up appointment as Pathologist at the Palmerston North Hospital, a position he held for the next 25 years. He was admitted MRACP in 1951 and elected FRACP in 1962. A foundation member of the new Zealand Society of Pathologists he was elected MRCPA in 1965.

During his professional career he was for a number of years External Examiner Pathology and Microbiology to the University of Otago, pioneered the introduction of BCG vaccination in New Zealand, and for many years was an active member of the New Zealand Cancer Society. His main outside interests were fishing and golf.

Thomas Pullar, or 'Thos' as he was affectionately known, was a champion and great friend of New Zealand Medical Laboratory Technologists. For many years he was involved with the gradual building up of professional laboratory standards throughout the country, and with the formation of the Medical Laboratory Technologists Board. He was intensely concerned and involved with the training and welfare of Medical Laboratory Technologists. He helped draft conditions of employment and prepared new syllabi for the Intermediate Examinations.

Deteriorating health necessitated a lighter work load and in 1963 he moved to the milder climate of Tauranga engaging in part time private laboratory practice there. During the last year of his life he visited medical laboratories throughout the country supervising and setting up Technologists' exams and introducing new educational training schemes. Thomas H Pullar was a friend, teacher and lifelong champion of New Zealand Medical Laboratory Technologists and thus it is fitting that to this day our Institute continues to recognise his many contributions to our profession through the TH Pullar Memorial Address.

Review Article

Le(a+b+); Phenotype and Genotype

Steve Henry, FNZIMLS, PhD, DrMedSc; Research Fellow;

Department of Clinical Chemistry and Transfusion Medicine, Sahlgrens Hospital,

Göteborg University, Göteborg, Sweden.

NZ J Med Lab Science 1996 50:2 50-53

Abstract

The Lewis system is a complicated histo-blood group system in which the carbohydrate antigens are formed by the interaction of Lewis and secretor fucosyltransferases. The detection of Lewis antigens on red cells depends on many variables. These include the degree of interaction of the Lewis, secretor and ABO glycosyltransferases, together with the factors involved in the secondary acquisition of the glycolipid antigen from plasma. These issues and others concerning the Lewis and secretor systems have recently been reviewed in detail[1]. This review will focus on the Le(a+b+) phenotype, a common phenotype of the South Pacific.

General Introduction

The Lewis histo-blood group system essentially consists of two major serologically defined antigens, Le^a and Le^b. These oligosaccharide antigens are synthesized by the sequential action of glycosyltransferases. The final Lewis antigenic determinants are defined by the presence of one or two fucoses, on either or both of the two terminal saccharides of the type 1 precursor chain. The presence, position, and linkage of these fucoses determine the differences between the type 1 precursor (Galß1→3GlcNAcß1-R), H type 1 (Fuc∝1→2Galß1→3GlcNAcß1-R), Le^a (Galß1→3(Fuc∝1-→4)GlcNAcß1-R) and Le^b (Fuc∝1→2Galß1→3(Fuc∝1→4) GlcNAcß1-R).

The two fucosyltransferases responsible for the synthesis of the Le^a and Le^b glycoconjugates have been cloned, sequenced and expressed[2,3].

The *Lewis* gene codes for an ∝(1,3/1,4) fucosyltransferase. This can add fucose to the subterminal GlcNAc of either the type 1 precursor or H type 1 to form the Le^a and Le^b antigens respectively[2]. There are also alleles which code for inactive (or partially active) proteins, and these are responsible for the Lewis-negative phenotypes[2,4]. The Lewis transferase is expressed in about 90% of Europeans and controls the expression of Lewis antigens (Le^a and Le^b) in the secretory compartments[5]. Those 10% of individuals not expressing Lewis substances are known as Lewis-negative.

The *secretor* gene codes for an ∝(1,2) fucosyltransferase that can add a fucose onto the terminal Gal of the type 1 precursor to form H type 1, the precursor of Le^b[3]. There are also inactive alleles at the secretor loci, which code for inactive proteins, and these are responsible for the nonsecretor phenotype[3,10]. Recently an allele which codes for a poorly active secretor transferase has been described in Asians[11], Indonesians[12] and Polynesians[13]. The secretor transferase is expressed in 80% of Europeans and controls the expression of H in the secretory compartments. The expression of H substances in secretions allows the A and/or B glycosyltransferases to synthesize blood group epitopes of the individual in secretory fluids, hence the name secretor. Those 20% of individuals not expressing H and/ or related A and/or B substances in secretions are known as nonsecretors.

As a consequence of polymorphism of the genetically independent Lewis and secretor systems and interaction of their transferases, four major red cell and salivary ABH secretor phenotypes are common in Europeans. These are; Lewis-negative secretors and nonsecretors both red cell phenotyping as Le(a-b-), Lewis-positive nonsecretors whose red cell phenotype will be Le(a+b-), and Lewis-positive secretors whose red cell phenotype will be Le(a-b+) (as reviewed in [1]).

The Le(a+b+) Red Cell Phenotype

In 1951 a new red cell Lewis phenotype known as Le(a+b+) was first described[14]. In this genetic survey of Maoris the Le(a+b+) phenotype was found to be present in 11% of individuals tested, all group O. Over the next few decades occasional papers appeared reporting the presence of the Le(a+b+) phenotype in various populations (table 1). Polynesians have a high incidence of the phenotype, which was reported to range from 10-40% in the different ethnic groups[15]. The incidence is now realised to be higher because many Le(a+b-) individuals in the original study were incorrectly phenotyped Le(a+b+) samples (see below). In general the Le(a+b+) phenotype remains most notable for its virtual absence in Caucasians[16].

Another notable feature of the Le(a+b+) phenotype was that there was an association with the ABO system. Most examples of the Le(a+b+) phenotype occurred in group O individuals. As the ABO and Lewis systems are not in genetic linkage, these results suggested that many Le(a+b+) phenotype individuals were being incorrectly phenotyped as either Le(a+b-) or Le(a-b-). This was exemplified when it was shown that commercially available monoclonal, goat and human anti-Lewis reagents were unreliable in their ability to define Lewis phenotypes in Polynesians[17]. It was found that of 108 Polynesian samples phenotyped, 17 individuals (16%) gave different phenotypes with alternative reagents. Later in 1990, using several monoclonal Lewis reagents, the Le(a+b+) phenotype was clearly demonstrated in most individuals previously phenotyping as Le(a+b-), of both Polynesian[16] and Asian[18] descent. These anomalous phenotypes cast doubt on actual phenotype frequencies reported for populations that have the Le(a+b+) phenotype (table 1). For example, in Chinese the frequency of Le(a+b-) varies from 0% to 10% and Le(a+b+) from 13% to 25% when different antisera are used[20]. We estimate that the frequency of the Le(a+b+) phenotype in Polynesians is 15-45% (the range reflecting the different ethnic groups), and the Le(a+b-) phenotype is virtually absent.

The Le(a+b+) phenotype is clearly very common in many

populations, but its detection is reagent dependent. Today the monoclonal anti-Lewis reagents which are commercially available do not usually detect the phenotype, although they are the same clones as those which can detect the Le(a+b+) phenotype (unpubl. observ.). This is because commercial reagents are over-diluted and used by techniques which do not enhance the detectability of the poorly expressed Le^b antigen on Le(a+b+) red cells, ie direct saline agglutination versus enzyme techniques. As a consequence, despite the ability to be able to detect this phenotype, it is not routinely detected because the reagents in current clinical use are not reliable for accurate Lewis phenotyping.

The Le(a+b+) phenotype can occasionally be seen with Caucasian samples[21] (table 1). This is usually because in some individual increased amounts of Le^a are expressed, which is detected by potent anti-Le^a reagents, usually monoclonals. These individuals are however not true Le(a+b+) individuals and their serology is different, with weak Le^a reactions and a salivary secretor phenotype[17]. In true Le(a+b+) individuals the Le^a reactions are usually very strong and they have the salivary partial-secretor phenotype. It is also possible to detect Le^a antigens on Le(a-b+) cells using sensitive techniques, such as enzyme treated red cells and the antiglobulin technique[22], but again these cells are not considered at Le(a+b+). The Le(a+b+) phenotype also occurs in another population; infants of all races[23]. Infants usually phenotype as Lewis-negative at birth, however, this depends on the type and potency of the reagent used[24]. It is generally accepted that there is a maturation of Lewis and secretor enzymes[24,25] which causes a progressive change in the phenotype of an infant from Le(a-b-), to Le(a+b-), then Le(a+b+), before finally becoming Le(a-b+) in infants of the Lewis-positive, secretor-positive genotype[22]. As a consequence of this, you cannot rely on an infant's Lewis phenotype until at least 2 years of age. It should however be noted that the infant Le(a+b+) phenotype is unlike the adult Le(a+b+) phenotype, the latter having a salivary partial-secretor phenotype and the former a secretor phenotype[27].

The Associated Partial-Secretor Phenotype

In 1970 an important paper on salivary substances and the Le(a+b+) phenotype appeared when Sturgeon and Arcilla studied the salivary substances in Japanese families and correlated their findings with the Le(a+b+) phenotype[17]. They reported for the first time that the Le(a+b+) phenotype is associated with poor secretion of ABH substances and postulated the concept that the phenotype was caused by an inefficient secretor gene they called *Se*[17]. These findings were supported by others who also found poor expression of salivary substances in Le(a+b+) Australian aborigines[26,27]. Two decades later, the association of the poor expression of salivary substances was re-examined in a large group of Polynesians[28]. Like the original report, poor expression of salivary ABH substances was correlated with the Le(a+b+) phenotype. Furthermore, most Polynesian individuals of the Le(a+b-) phenotype were also found to have low levels of salivary ABH and Lewis substances. The term "partial-secretors" was adopted to describe these individuals who had less salivary ABH substances than secretors but more than non-secretors. Similar observations were also made in Chinese[20].

The alternative possibility that the Le(a+b+) phenotype was caused by a strong Lewis tranferase rather than a weak secretor transferase was made unlikely in light of the finding of Le(a-b-) partial-secretors[28]. These individuals were actually described in the early literature[29] (and as revised in[30]).

The Lewis Glycolipids of Le(a+b+) Individuals

Evidence collected from serological analysis strongly suggested that a weak secretor transferase existed and was responsible for the Le(a+b+) phenotype. Such a concept was highly compatible with the accepted biosynthetic pathway for Lewis antigens (as reviewed in[9]). If the secretor transerase is inefficient, less H type 1 will be formed and more Le^a will result. Less H type 1 means less Le^a (hence weak Le^b serological reactions) and less ABH substances are able to be formed (hence partial-secretion). Obviously in non-group O individuals competition from the A and B transferases would make even less H type 1 available for conversion into Le^b (hence its poorer detection rate in non-group O individuals).

This hypothesis (based on serological data) for the Se^w transferase was tested by extracting Lewis glycolipids from plasma, red cells and small intestinal epithelial cells of Le(a+b+) individuals[31-34]. When these results were contrasted with individuals of other "common" Lewis phenotypes it was concluded that unlike all other Lewis phenotypes, Le(a+b+) individuals co-expressed significant levels of Le^a and Le^c. Examination of the glycolipids of the red cell membrane showed that both glycolipids were expressed and there was quantitatively much less Le^a in the red cell membrane than in Le(a-b+) individuals. In intestinal epithelial cells the results were concordant with those of plasma[31]. Similarly, the plasma glycolipid profiles of individuals of the Polynesian Le(a+b-) phenotype who secreted ABH and/or Lewis substances were the same as Le(a+b+) individuals, suggesting the correct phenotype of these individuals should be Le(a+b+)[34]. The biochemical evidence was therefore all strongly in support of an inefficient transferase which altered the equilibrium between the secretor and Lewis transferases.

An interesting and unexpected observation was made, which was also biosynthetically compatible with the concept of a weak secretor transferase. It was noted that when the inefficient Se^w transferase was believed to be present (eg Le(a+b+) and/or partial-secretors), extended glycolipids were more predominant than in secretors[31,34]. The biosynthetic pathway for Lewis antigens was revised to include a postulated precursor extension pathway which operates until secretor or Lewis fucosylation modifies the precursor[32].

The Le(a+b+) Genotype

In 1995 the group led by Dr Lowe in Michigan published the sequence of the secretor transferase (FUT2) which allowed for the screening of secretor mutations[3]. We sequenced the coding region of the secretor gene of 10 Polynesians and found a new point mutation. This missense mutation with an A→T substitution at nucleotide 385 resulted in a change of amino acid 129 from isoleucine to phenylalanine. The mutation, when appearing in a homozygous state (or heterozygous with a nonsecretor allele), associated with the rare red cell Le(a+b+) and salivary partial-secretor phenotype[12,13]. This mutation was also studied in an Indonesian pedigree where it correlated with the Le(a+b+) and partial-secretor phenotype[12]. In Chinese the same mutation was identified and correlated with the Le(a+b+) phenotype[11]. Expression of the mutation in a cell line (COS-7) concluded that it results in a secretor fucosyltransferase which is more susceptible to proteolysis and its catabolism is accelerated[13]. These results conclude that the molecular basis for the Le(a+b+) and associated partial-secretor phenotype is an amino acid change in the α 1,2 fucosyltransferase coded for by an A→T mutation in nucleotide 385 of the FUT2 gene.

The Future of the Le(a+b+) Phenotype

The Le(a+b+) phenotype has been clearly established as a real phenotype of the Lewis system but unfortunately it still remains largely unrecognised because of the poor quality of Lewis reagents routinely available. Although mistyping a Le(a+b+) individual as Le(a+b-) or even Le(a-b-) will probably not have any transfusion implications, this error may be important in other areas such as transplantation and disease studies. Until serological reagents are improved this problem will not be resolved. With the recent advances

made with molecular genotyping it is now possible to detect the alleles responsible for the Le(a+b+) and partial-secretor phenotypes. It is however still too early for molecular genotyping to be used as a replacement for phenotyping. It is now important to establish the genotype frequencies for the Lewis and secretor alleles in different populations and to correlate these results with phenotypes.

The Le(a+b+) phenotype is common in many populations, especially those of the South Pacific. Why the Le(a+b+) phenotype should exist is still unknown. The biological significance of the Lewis system still remains obscure, and at best can only be indirectly linked with various disease processes. It is however known that the Le(a+b+) phenotype confers on an individual's mucosal epithelial cells a glycoconjugate profile that is different from that found in any other Lewis phenotype. This different glycoconjugate profile may be related to some selective biological pressure because carbohydrate antigens are known to interact with microbes[35].

The high incidence of the Le(a+b+) and partial-secretor phenotypes, and the unique glycoconjugate profiles in these individuals suggests that some common but as yet unidentified biological pressure exists in the South Pacific[36]. Molecular biology will soon be able to offer the tools to allow the biological significance of this system to be systematically evaluated.

Table 1:

Adult populations and ethnic groups in which the Le(a+b+) and/or partial-secretor phenotypes have been recorded. Phenotype frequencies are not indicated because these have not yet been accurately established.

Populations with Le(a+b+) and/or partial-secretor phenotypes

Polynesian	Asian	Others
Cook Islander[15]	Chinese[37]	American Negro[38]
Maori[5, 15]	Japanese[21, 29]	Australian aborigine[21, 26, 27]
Niuean[15]	Malay-Polynesian[37]	Caucasian[27, 39, 40]
Samoan[15]		Indonesian[4]
Tongan[15]		Réunion Islander[32]
Tokelauan[15]		Siberian[16]

References

1. Henry SM, Oriol R, Samuelsson BE. Lewis histo-blood group system and associated secretory phenotypes. *Vox Sang* 1995; 69: 166-82.
2. Kukowska-Latallo JF, Larsen RD, Nair RP, Lowe JB. A cloned human cDNA determines expression of a mouse stage-specific embryonic antigen and the Lewis blood group ∝(1,3/1,4) fucosyltransferase. *Genes Dev* 1990; 4: 1288-303.
3. Kelly RJ, Rouquier S, Giorgi D, Lennon GG, Lowe JB. Sequence and expression of a candidate for the human Secretor blood group ∝(1,2) fucosyltransferase gene (FUT2). Homozygosity for an enzyme-inactivating nonsense mutation commonly correlates with the non-secretor phenotype. *J. Biol Chem* 1995; 270: 4640-9.
4. Mollicone R, Reguigne I, Kelly RJ, Fletcher A, Watt J, Chatfield S, Aziz A, Cameron HS, Weston BW, Lowe JB, Oriol R. Molecular basis for Lewis ∝(1,3/1,4) fucosyltransferase gene deficiency (FUT3) found in Lewis-negative Indonesian pedigrees. *J Biol Chem* 1994; 269: 20987-94.
5. Elmgren A, Rydberg L, Larson G. Genotyping of Lewis blood group status. (Abstract). *FEBS Special Meeting on Biological Membranes, Helsinki* 1994;
6. Elmgren A, Rydberg L, Larson G. Genetic heterogeneity among Lewis negative individuals. *Biochem Biophys Res Commun* 1993; 196: 515-20.
7. Koda Y, Kimura H, Mekada E. Analysis of Lewis fucosyltransferase genes from the human gastric mucosa of Lewis-positive and -negative individuals. *Blood* 1993; 82: 2915-9.
8. Nishihara S, Yazawa S, Iwaskai H, Nakazato M, Kudo T, Ando T, Narimatsu H. ∝(1,3/1,4) fucosyltransferase (FucT-III) gene is inactivated by a single amino acid substitution in Lewis histo-blood type negative individuals. *Biochem Biophys Res Commun* 1993; 196: 624-31.
9. Oriol R, Le Pendu, J, Mollicone R. Genetics of ABO, H, Lewis, X and related antigens. *Vox Sang* 1986; 51: 161-71.
10. Henry S, Mollicone R, Lowe JB, Samuelsson B, Larson G. A second nonsecretor allele of the blood group ∝(1,2) fucosyltransferase gene (FUT2). *Vox Sang* 1996; 70: 21-5.
11. Yu L-C, Yang Y-H, Broadberry RE, Che Y-H, Chan Y-S, Lin M. Correlation of a missense mutation in the human *Secretor* ∝1,2-fucosyltransferase gene with the Le(a+b+) phenotype: a potential molecular basis for the weak *Secretor* allele (*Se*[w]). *Biochem J* 1995; 312: 329-32.
12. Henry S, Mollicone R, Fernandez P, Samuelsson B, Oriol R, Larson G. Homozygous expression of a missense mutation at nucleotide 385 in the FUT2 gene associates with the Le(a+b+) partial secretor phenotype in an Indonesian family. *Biochem Biophys Res Commun* 1996; 219: 675-78.
13. Henry S, Mollicone R, Fernandez P, Samuelsson B, Oriol R, Larson G. Molecular basis for erythrocyte Le(a+b+) and the salivary ABH partial-secretor phenotypes. Expression of a ∝FUT2 secretor allele with an A→T mutation at nucleotide 385 correlates with reduced ∝(1,2) fucosyltransferase activity. *Glycoconj J* 1996; (in press).
14. Simmons RT, Graydon JJ, Semple NM, Taylor GND. Blood, taste and secretion: a genetical survey in Maoris. *Med J Aust* 1951; 1:425-31.
15. Henry SM, Simpson LA, Woodfield DG. The Le(a+b+) phenotype in Polynesians. *Hum Hered* 1988; 38: 111-6.
16. Mourant AE, Kopec A, Domaniewska-Sobczak K. The distribution of human blood groups and other polymorphisms. Oxford University Press, Oxford. 1976; 548-76.
17. Henry SM, Simpson LA, Benny AG, Woodfield DG. Investigation of Polynesian Lewis phenotypes. Variability in detection of Lewis antigens by monoclonal, goat and human antisera. *N Z J Med Lab Technol* 1989; 43: 64-7.
18. Henry SM, Dent AM, Harding Y. Detection of an Le[a] epitope peculiar to Polynesians. In proceedings of the second international workshop and symposium on monoclonal antibodies against red blood cells and related antigens. Chester MA, Johnson U, Lundblad A, Löw B, Messeter L, Samuelsson B. Secretariat, Lund, 1990: 75.
19. Lin-Chu M, Broadberry RE. The Lewis phenotypes among Chinese in Taiwan. In Proceedings of the second international workshop and symposium on monoclonal antibodies against human red blood cells and Chester MA, Johnson U, Lundblad A, Löw B, Messeter L, Samuelsson B. Secretariat, Lund, 1990: 79.
20. Broadberry RE, Lin-Chu M. The Lewis blood group system among Chinese in Taiwan. *Hum Hered* 1991; 41: 290-4.
21. Sturgeon P, Arcilla MB. Studies on the secretion of blood group substances I. Observations on the red cell phenotype Le(a+b+x+). *Vox Sang* 1970; 18: 301-22.
22. Cutbush M, Giblett ER, Mollison PL. Demonstration of the phenotype Le(a+b+) in infants and adults. *Br J Haematol* 1956; 2: 210-20.

23. Lin M, Shieh SH. Postnatal development of red cell Le^a and Le^b antigens in Chinese infants. *Vox Sang* 1994; 66: 137-40.
24. Oriol R. ABO. Hh, Lewis and secretion. Serology, genetics and tissue distribution. In Blood cell biochemistry, molecular basis of major blood group antigens. Cartron JP, Rouger P. Plenum Press, New York, 1995: 37-73.
25. Mollicone R, Candelier JJ, Mennesson B, Couillin P, Venot AP, Oriol R. Five specificity patterns of (1-3)-∝-L-fucosyltransferase activity defined by use of synthetic oligosaccharide acceptors. Differential expression of the enzymes during embryonic development and in adult tissues. *Carbohydr Res* 1992; 228: 265-76.
26. Vos GH, Comley P. Red cell and saliva studies for the evaluation of ABH and Lewis factors among the Caucasians and Aboriginal populations of Western Australia. *Acta Genet Stat Med* 1967; 17: 495-510.
27. Boettcher B, Kenny R. A qualitative study of Le^a, A and H antigens in saliva of Australian Caucasians and Aborigines. *Hum Hered* 1971; 21: 334-45.
28. Henry SM, Benny AG, Woodfield DG. Investigation of Polynesian Lewis phenotypes. Evidence of a weak secretor phenotype. *Vox Sang* 1990; 58: 61-6.
29. Lewis M, Kaita H, Chown B. The blood groups of a Japanese population. *Am J Hum Genet* 1957; 9: 274-83.
30. Race RR, Sanger R. Blood groups in man, 5th ed. Davis, Philadelphia. 1968: 310.
31. Henry SM, Woodfield DG, Samuelsson BE, Oriol R. Plasma and red cell glycolipid patterns of Le(a+b+) and Le(a+b-) Polynesians as further evidence of a weak secretor gene, Se^w. *Vox Sang* 1993; 65: 62-9.
32. Henry SM, Oriol R, Samuelsson BE. Detection and characterisation of Lewis antigens in plasma of Lewis-negative individuals: Evidence of chain extension as a result of reduced fucosyltransferase competition. *Vox Sang* 1994; 67: 387-96.
33. Henry SM, Samuelsson BE, Oriol R. Immunochemical and immunohistological expression of Lewis histo-blood group antigens in small intestine including individuals of the Le(a+b+) and Le(a-b-) nonsecretor phenotypes. *Glycoconj J* 1994; 11: 600-7.
34. Henry SM, Oriol R, Samuelsson BE. Expression of Lewis histo-blood group glycolipids in the plasma of individuals of Le(a+b+) and partial secretor phenotypes. *Glycoconj J* 1994, 11: 593-9.
35 Karlsson K-A, Angström J, Bergström J, Lanne B. Microbial interaction with animal cell surface carbohydrates. *APMIS Suppl* 1992; 100: 71-83.
36. Mollicone R, Cailleau A, Oriol R. Molecular genetics of *H, Se, Lewis* and other fucosyltransferase genes. *TCB* 1995; 4: 235-42.
37. Lin-Chu M, Broadberry RE, Chang FJ. The distribution of blood group antigens and alloantibodies among Chinese in Taiwan. *Transfusion* 1988; 28: 350-2.
38. Edwards JM, Reed RM, Greenwell P, Johnson P. Anomalies in the Lewis blood group system. *Transfusion* 1982; 22:411.
39. Simmons RT, Jakobowicz R. The Lewis blood antigens and antibodies. *Med J Aust* 1951; 1: 497-500.
40. Brendemoen OJ. Further studies of agglutination and inhibition in the Lea-Leb system. *J Med Clin* 1950; 36: 335-41.

Cyclic Neutropenia – A Case History

Ailsa Bunker, Staff Technologist, Haematology Department, Middlemore Hospital
Otahuhu, New Zealand

NZ J Med Lab Science 1996 50;21 54-56

Introduction

Cyclic (cyclical) neutropenia has been attributed to a regulatory abnormality affecting blood cell formation at the stem cell level. The utilisation and distribution is normal but the precise defect in these cells is not yet known. There are many theories. A stem cell defect and/or immunological abnormalities are considered to play a part in this disease[1,2,3,4].

Also, recently T-lymphocytes have been implicated in cyclic neutropenia pathogenesis. A persistent inversion of the T-helper/T-suppressor ratio has been described in at least one patient. It has also been suggested that the defect could be an insufficient production of granulocyte-macrophage colony stimulating factor by lymphocytes.[3]

It is fascinating to find out the cause of this disorder but at the coal face of Medical Laboratory Science it will always be important to identify the condition so it can be treated.

It is a rare condition characterised by regular intervals of neutropenia, often showing a 20-21 day cycle, with a duration of 3 to 6 days.[1,2] Clinically patients have problems when neutropenic, but these are generally not life-threatening.[1,2] About one third of cases have family members with either cyclic or chronic neutropenia,[1] some of whom show autosomal dominance.[2]

Clinical manifestations include fever, mouth ulcers, skin infections, joint pain, malaise, lymphadenopathy, abdominal pain and anorexia.[1,2]

Case History

A 26-year-old woman with profound neutropenia, low grade fever (37.3°C) and vomiting was referred to Haematology by her GP. She also had 2 painful mouth ulcers and tender cervical glands.

She first had a neutropenia documented by her GP three weeks before presentation and this was said to vary since then. Results then were: WBC 3.2x10^9/l, Neutrophils 0.16 x10^9/l, Hb 118g/l, platelets 410x10^9/l.

At referral a full examination, medical history and a battery of laboratory tests were taken.

The cause at this stage was unclear for several reasons. She had had a recent drug history which included flagyl, penicillins, ceclor, bactrim, bromocryptine and rulide.

On top of this she was due to have a colonoscopy the following day but this was cancelled because of the neutropenia. It was thought that the vomiting may have been due to the colonoscopy preparation.

The patient had a 10 year history of mouth ulcers and recently during the previous six months, which were post partum, had had four episodes of mastitis, oral candidiasis, Vincent's angina, a urinary tract infection and an episode of night sweats. She also was said to have mild iron deficiency, presumed secondary to the demands of the recent pregnancy.

Differential diagnoses were proposed (Table 1) and all but drug induced and cyclic neutropenia were excluded by examination of laboratory tests and history and examination of the patient. Blood cultures, a mid-stream urine and a chest x-ray were taken and showed no infection. On serological examination she was shown to have had a previous but not current EBV (Epstein Barr Virus) infection (IgG +ve, IgM -ve), and her ANF (Antinuclear Factor) was negative, thus excluding SLE. (Systemic Lupus Erythematosis)

Table 1 – Differential Diagnosis

- viral illness
- drugs
- familial/cyclical neutropenia
- systemic lupus erythematosis
- severe sepsis
- myelodysplastic syndrome

Haematologically she showed a neutropenia with the neutrophils at 0.18 x 10^9/l. Other haematological findings were:

Hb 121g/l, MCV 80.7 fl, WBC 2.3 x10^9/l,
Lymphs 1.54x10^9/l, Platelets 352 x 10^9/l,
Coagulation screen, renal and liver function tests were normal.

Her antenatal blood test results were reviewed and were shown to have normal WBC and neutrophil numbers. (The white cell counts ranged from 4.7 to 6.2 x 10^9/l and absolute neutrophil counts 2.44 – 4.2 x 10^9/l).

Sequential blood counts were requested and through the month showed her white cell count to vary. The following month, a cyclical pattern of neutropenia began to emerge.

Serial blood counts continued and after three months, a clear pattern of cycling of the patients' neutrophils was established with counts ranging from 0.16 to 3.6 x 10^9/l.

As shown in Figure 1, her counts fall approximately every three weeks and then return to normal. This being well established no bone marrow examination was performed.

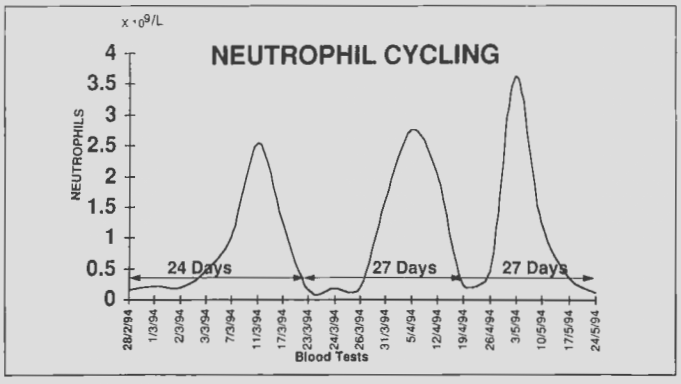


Figure 1 Neutrophil Fluctuation

Table 2 – Treatment Options

- Treat the symptoms only:
  infections with antibiotics
  arthralgia with analgesics
- Some response shown to prednisone therapy
- G-CSF and GM-CSF being trialed with promising effects

The patient was not aware of any other family member that has this haematological problem though she did mention that her younger brother also has relatively frequent mouth ulcers and it is possible he also has the condition. (This has not been confirmed)

During her neutropenia she develops symptoms of mouth ulceration, tender cervical lymphadenopathy and occasionally systemic flu-like symptoms.

It was suggested at this stage that she continued mouth cares, especially when neutropenic and if she became unwell with a marked fever to seek urgent medical attention so systemic antibiotics could be administered.

G-CSF (Granulocyte Colony Stimulating Factor), generic name filgrastim = rh G-CSF, one of the cytokines was mentioned as a possible treatment for cyclic neutropenia but unfortunately it is expensive, (approximately $1,000 for 5 vials), not easily obtainable and requires subcutaneous injection so it was thought that at this stage a "wait and see" approach would be best although if she were to undergo a surgical procedure it would be considered.

The patient's neutrophil counts through the remainder of the year rarely reached normal levels. She was still having problems with mouth ulcers and had a dry crusting scalp lesion that was being treated by a dermatologist with rifampicin and flucloxacillin.

The haematologists once again discussed the possible therapeutic alternatives and decided the patient might benefit from a trial of GM-CSF (Granulocyte-monocyte Colony Stimulating Factor) generic name Molgramastin, in order to maintain an adequate white count to allow healing of her scalp infection. GM-CSF was considered because Middlemore pharmacy had a supply available which was free for trial from a drug company.

The potential side effects (Table 3[15]) were discussed with the patient and she agreed to a trial.

Table 3 – Toxicity of GM-CSF

Non dose related

- fever
- bone pain
- myalgia
- rarely catheter thrombosis, and splenomegaly

High dose related i.e. > 32µg/kg/day

- oedema
- weight gain
- effusion and pulmonary emboli

Figure 2: This graph shows 85 blood test results taken over a period of about 2 years. As you can see it shows clearly the neutrophils fluctuating, the lymphocytes remaining relatively constant and the total white count moving with the fluctuations of the neutrophils.

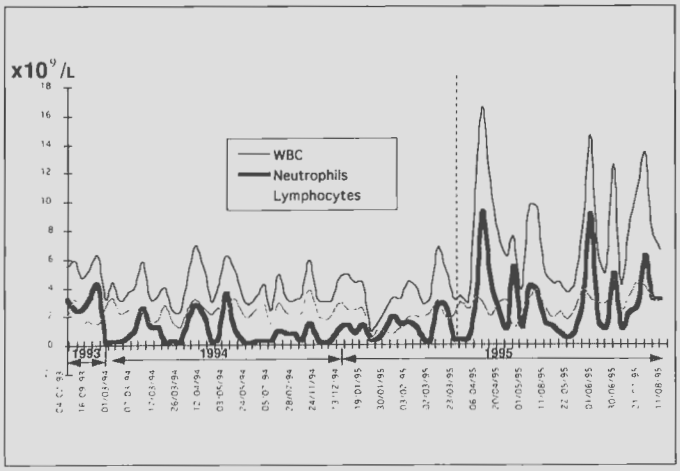


Figure 2 – Graph of WBC/Neuts/Lymphocytes for 1994-95

She was no longer developing severe neutropenia, indeed her neutrophil count had been 3x10⁹/l at the beginning of the month, a time usually associated with a low neutrophil count. She is keen to continue treatment with GM-CSF, her only concern now is for the continuous supply of GM-CSF, and that she would like to become pregnant and GM-CSF is not approved for pregnancy. Although it is noted that pregnancy often improves the cyclic neutropenia[16] she would need to be off this drug before she became pregnant. The treatment of cyclic neutropenias has in the past been reactive but now as the cytokines are being researched and used more perhaps a true cure may be made and the quality of life for these people greatly improved.

On March 23rd the patient had her first dose of GM-CSF at the low dose of 1ug/kg (i.e., 50 ug daily) for 4 weeks. (This is marked by the vertical line)

Following the GM-CSF's administration the neutrophil counts remained low for about a week, but by day 14 it was 3 x 10⁹/l with a total WBC of 11.3 x 10⁹/l.

During the first week of her injections she had a problem with bony pain but this resolved. After about two weeks red itchy welts appeared around the injection sites which took several days to clear.

At this stage it was difficult to tell if the patient had had a definite clinical response to GM-CSF although the white cell count certainly was the highest she had had for months.

Treatment continued with GM-CSF on alternate days and topical treatment was given for the itch (hydrocortisone and antihistamine). Over the next month the patient reported she felt well with her energy levels greatly improved and her head was healing. (She was also on flucloxacillin and topical Manuka honey for her scalp lesion). Her urticarial lesions around the injection sites had diminished significantly since starting the alternated day GM-CSF programme.

Treatment continued but a boost of GM-CSF doses to daily was thought to be needed for about 5 days per month to cover the predicted neutropenic periods. This was decided after a bout of neutropenia at the end of May, as can be seen from the dip on Figure 2. This coincided with symptoms of flu, myalgia and mild fever.

The patients' scalp infection still remained a worry since ceasing treatment with antibiotics and Manuka honey. The only other side effect has been eosinophilia which fluctuates with the neutrophils and has been up to 4.5 x 10⁹/l.

When seen at clinic in mid August, 1995, the patients' results were as shown in Table 4.

Table 4 – Blood sample results at clinic – August 1995

		(Normal Range)
Hb	117g/l	(115-165)
WBC	16.7 x 10^9/l	(4-11)
Neutrophils	7.68 x 10^9/l	(2.2-7.5)
Lymphocytes	4.98 x 10^9/l	(1.0-3.9)
Monocytes	0.50 x 10^9/l	(0-0.9)
Eosinophils	4.18 x 10^9/l	(0-0.5)
Platelets	443 x 10^9/l	(150-400)

Conclusion

Cyclic neutropenia is the never ending story of haematology. It is not usually life-threatening, comes with a myriad of clinical presentations, including dermatological, and needs many frequent blood tests so that it will not be missed or dismissed as a laboratory error. This requires a great deal of tolerance and understanding on the part of the patient.

Finally a quote from the 1991 4th Edition of Haematology

"A diagnosis of cyclic neutropenia can be made only by several white blood cell counts and differentials at least three times per week for a minimum of six weeks".[1]

Acknowledgement

Thanks to Drs Hilary Blacklock and Sharon Jackson, Consultant Haematologists, Middlemore Hospital for permitting me to document this case report of their patient and help with preparation of this manuscript.

References

1. Dale D C, Neutropenia. In *Haematology*, 4th Edition, edited by Williams J W, Bentler E, Ersler A J, Lichtman M A, 1991: 809.
2. Goldman J M, Granulocytes, Monocytes and their benign disorders. In *Postgraduate Haematology*, 3rd Edition, edited by Hoffbrand A V, Lewis S M, 1989: 319-20.
3. Rodenas J M, Ortega N, Herran M T, Tercedor J, Pinar A, Quero J H, Cyclic Neutropenia: A cause of recurrent aphthous stomatitis not to be missed. *Dermatology*, 1992; 184(3): 205-7.
4. Athens J W, Neutropenia. In *Wintrobe's Clinical Haematology*, 9th Edition, Volume 2, edited by Lee G R, Bithell T C, Foerster J, Athens, J W, Lukens J N, 1993: 1603-5.
5. Schriber J R, Negrin R S, Use and Toxicity of the Colony Stimulating Factors, *Drug Safety* 1993; 8: 457-88.
6. Pajor A, Szakacs Z, Pregnancy in cyclic neutropenia. *Gyn Obstet Invest* 1991; 32: 189-90.

Winner of the Med Bio Journal Award for the March 1996 issue was Michelle Dougherty from Medlab Hamilton for her article *"Cyclospora cayetanensis*: An emerging intestinal pathogen in New Zealand".

Occasional Article

Life in Sweden: A Personal Account from a Visiting Postdoctoral Researcher

Steve Henry, Research Fellow;
Department of Clinical Chemistry and Transfusion Medicine, Sahlgrens Hospital, Göteborg University, Göteborg, Sweden.

NZ J Med Lab Science 1996 50(2) 57-58

In 1994 my wife Helen, children Jessie (8), Zak (3) and I made the difficult decision of resigning from my job, selling our home, and moving to Sweden for 2 years so I could do a postdoctoral fellowship. We left in September 1994, and after twenty three flying hours (plus a few more in airports), arrived exhausted to a beautiful Swedish autumn day; clear skies, sun and about 4°C. Our adventure had begun.

The city of Göteborg (Gothenburg in English) was founded in 1621. Göteborg is located on the west coast of Sweden and currently has close to 500,000 inhabitants. It is the second largest city of Sweden, surpassed only by the capital, Stockholm. Göteborg is a beautiful city with cafés, tree lined avenues, canals, trams, a major port, lots of parks, and an archipelago.

The first thing one seems to do when visiting a foreign country is to make comparisons with home. The most obvious dissimilarities were the foreign language (and the way that what you read is unrelated to its pronunciation!), lefthand drive cars, a population where most people live in apartment buildings, the woods, the high cost of living, and a regulated society. In fact 70% of the economy is controlled by some level of government or its subsidary. The list of dissimilarities goes on but there are also many similarities, being that the people appear the same as NZers, although there is a definite "Swedish look", McDonalds is everywhere, and Sweden is a beautiful country. Almost all Swedes can speak fluent English, and they have similar values and morals to NZers. Although there are many cultural differences they are not too great. Swedes are generally more open-minded than NZers, but tend to keep to themselves. Although the culture may appear affluent, this affluence is only maintainable if both parents are in full-time employment. In general a NZer in the middle to upper income bracket would have a higher standard of living than a Swede of the same status. One of the most striking cultural differences seen in Sweden is the role of women. Women are equal without a doubt, and to help achieve this equality the State takes over a great deal of the child-rearing role. Sweden has an extremely generous welfare system, a fact which is reflected in the huge tax demand.

The weather. Göteborg has the same latitude as the Southern tip of Greenland and Siberia, but the weather is relatively mild because of the gulf stream. Compared with Auckland the weather and seasons are very different, and at opposite times of the year of course. We arrived in Autumn, which is a beautiful season with the leaves on most trees changing to vivid reds and yellows before carpeting the ground. The temperatures usually range from about 0 to 10°C and snow is occasional. It rains frequently but sunny days are also common, and although the temperature does not usually rise above 8°C it is not cold. Although this may sound like a contradiction, it should be appreciated that the cold here is generally dry and not biting like the cold of NZ.

In winter, the longest season, we experience subzero temperatures for several months, with the temperature averaging about minus 5°C and occasionally dipping to lows of minus 20°C. Sunny days are infrequent, but when they come they are glorious. Unfortunately there are usually very long periods of time when the sun is not seen. In January 1996 we officially had a total of 18 hours of sun, the rest of the time it was overcast. During the peak of winter the sun rises (in theory) about 9am, moves around the horizon and sets about 3:30pm. A consequence of this lack of sun over the winter months is depression and a disturbance in sleeping patterns, a medical fact which afflicted our children! It snows reasonably frequently in winter but rarely in large amounts. There are of course exceptional days like in November 1995 when we had a snow storm which dumped about half a metre of snow in 12 hours.

Spring is my favourite season in Sweden. The change from winter to spring is usually a dramatic event. Within weeks the temperature rises to above 10 degrees and all the plants develop buds. They don't open immediately, they just wait like a loaded spring. Then for some reason, some change occurs, and in a synchronised fashion, everything explodes into colour within a few days. The green is fresh, the colours of the flowers are vivid and the smells are breathtaking. Life completely changes and the people of Sweden transform along with the season.

Summer is brief. At the peak of summer the days are long, with sun-up about 4am and setting after 10:30pm. Unfortunately it lasts only about 6 weeks, but it is really welcome, and recharges one' batteries in preparation for the coming winter. Temperatures are in high 20s and can even reach the 30s. It is a time to enjoy, with a large percentage of the population going to either the lakes or beaches to swim and sunbathe (topless of course!). Getting work done during this time is difficult as many shops and businesses are closed and nobody feels like working.

Because the seasons are so dramatically different they become events, and heathen customs are also associated with certain seasonal events. In winter, the festival of light (Lucia) is celebrated, and at the end of winter two festivals, one to scare away the evil witches and both to celebrate the arrival of spring. The peak of summer is celebrated with an all-night party known as mid-summers eve.

My position in Sweden is as a postdoctoral researcher in the Glyco- and Transplanation Biology Group, Department of Clinical Chemistry and Transfusion Medicine, Göteborg University. My

research involved identifying, cloning and expressing the candidate Se^w gene which was believed to be responsible for the Polynesian Le(a+b+) and partial-secretor phenotypes. The molecular biology work progressed extremely well, and by sequence analysis we quickly identified in Polynesians a candidate Se^w allele. During the sequencing work a second nonsecretor allele was also identified in Polynesians[1]. Cloning and expression of the candidate Se^w allele was more involved and took a further 9 months to complete. Eventually we were able to express the enzyme encoded by the Se^w allele and prove that it resulted in less enzyme activity than normal[2]. The molecular basis for the Le(a+b+) and partial secretor phenotypes were thus resolved, and we have moved on to other problems related with carbohydrate blood group systems.

Current work involves determining the structures of elongated glycoconjugates which are associated with the Lewis and secretor systems. This latter work is very complex and involves structural determination by a team using such tools as ^1H NMR and tandem mass spectrometry together with preparative procedures. I am also investigating other related problems including studies into the origin of plasma Lewis glycolipids, and the possibility of artificially including carbohydrate antigen expression in organs. The ultimate objective of my research is towards the goal of understanding the role of glycoconjugates in transfusion, transplantation and disease.

Life in Sweden was not all work. In winter the ponds and numerous lakes in Göteborg freeze and it was not long before we became the owners of ice skates and enjoyed these open spaces. The sea also freezes but as this can be dangerous we only took the opportunity to walk on it near to the shore. Snow is a big attraction of course and we all became experts at tobagganing on the slopes behind our house.

In the summer 1995 we travelled up to the Swedish/Norwegian border, hired a large Canadian canoe, loaded it with our tent, sleeping bags and food and set off for a week, canoeing down through the lakes. At night we camped where we liked and by day we canoed for 3 hours to our next camp site. The lakes were beautiful, we only saw a few people and we had a memorable adventure. Two essential items on such a trip are a very effective mosquito repellent and a good insect proof tent, both items we were thankful we had.

Another attraction of Göteborg is a big amusement park, Scandinavia's biggest (2.8 million visitors every year). This was a favourite with the kids, although it was rather expensive. About 2 hours inland is another amusement park called Sommarland. This place is huge and includes a massive outdoor swimming complex. We stayed in a holiday cottage (stuga) at Sommarland for 4 days and enjoyed every day. Also about 2-3 hours inland from Göteborg is the "glassregion". This is where the famous crystal works such as Kosta Boda, Åfors, Orrefors, Sea, etc are found. If you venture into this region you can watch crystal being blown, purchase very reasonably priced crystal, or admire the things you can't afford!

Off the coast of Göteborg is an archipelago, on which many fishermen and small communities are based. This is also a defence area and so most of it is prohibited to "aliens". You can, however, visit the outermost island (Vrångö) by ferry and disembark for a walk and swim. Strangely, you cannot be on the ferry while it travels to the outer island because it passes through the restricted military zone. Catch 22! They trick is to give the kids a chewy sweet and refrain from speaking English until you get to Vrångö. (Psst, I have some nice video footage of the archipelago). In winter the sea freezes, and if it has been cold for long enough, the ferry service closes and they open official roads so that you can drive to the islands!!

Going to Sweden for 2 years was part of my career plan as well as a family adventure. Scientific research in Sweden is of a very high standard and extremely well funded. NZ research is of an equivalent standard, but funding in NZ is much harder to secure. The laboratory services in Sweden are excellent and also extremely well funded. Sweden, despite its apparent wealth, is however facing major financial difficulties, and I am sure all facets of social security and health are going to undergo some of the cost cutting measures experienced in NZ several years ago. This will be a hard road for the Swedes because they are a very socialistic people, and any suggestion of reducing the level of social support will not be well received.

The beauty of Sweden is very real, and emphasised by the harshness and contrast of the seasons. Sweden will always have a place in my heart, but will never supersede my love for New Zealand.

References

1. Henry S, Mollicone R, Lowe JB, Samuelsson B, Larson G. A second nonsecretor allele of the blood group $\alpha(1,2)$ fucosyltransferase gene (FUT2). *Vox Sang* 1996; 70: 21-5.
2. Henry S, Mollicone R, Fernandez P, Samuelsson B, Oriol R, Larson G. Molecular basis for erythrocyte Le(a+b+) and the salivary ABH partial-secretor phenotypes. Expression of a FUT2 secretor allele with an A→T mutation at nucleotide 385 correlates with reduced $\alpha(1,2)$ fucosyltransferase activity. *Glycoconj J* 1996; in press.

The VG Autospec tandem mass spectrometer and several members of the Glyco- and Transplantation Biology Group. From the left, Drs Steve Henry, Sohbat Ghardashkani, Mikael Gustavsson and Professor Bo Samuelsson. Mass spectra are recorded on this hybrid tandem instrument with a magnetic sector as MS-1 and an orthogonal time-of-flight device as MS-2 (AutoSpec-TOF, VG Analytical, Manchester, UK).

Editor's Note:
Steve Henry is a medical laboratory scientist who started his career in 1979 at Greenlane Hospital before specialising in Transfusion Medicine at the Auckland Region Blood Centre. Steve is a Fellow of the NZ Institute of Medical Laboratory Science and has a PhD in Biochemistry (Auckland) and a doctorate in Medical Science (Göteborg). Steve will be returning to NZ in July 1996 to pursue a research career.

South Island Seminar 1996

A Report thereof . . .

Continuing Education, we are glad to report, is alive and well in the South. No less than 120 eager intellects attended the great Southern MOLS hunt, held in the metropolis of Methven (Gateway to the skifields, and nothing to do with taps at all). Included in that record number were a few refugees from the north, mainly expatriates returning for some clean fresh non-moisture-saturated mountain air. There was good representation from most laboratories, not least being an entire busload from Canterbury Health Labs.

Most gratifying was the real sense of camaraderie shown from the beginning. The importance of being able to rise above street-level competitiveness, and interact at a professional level, should not be undervalued; our professional credibility depends on it. The sharing of scientific data without regard for preceived market-place advantage must be encouraged. The spread of papers proferred, from all sectors of the wider laboratory scene, was most encouraging.

The Saturday seminar has steadily grown in popularity over the yeas since it was resurrected from torpor of the 70s. Its lack of formality and the generalised nature of the programme provides a good springboard for first-time presenters, who are encouraged through a substantial award providing assistance with travel to the main NZIMLS conference.

The staff at Centrepoint Resort Hotel, ensured, through their hard work and attention to detail, that the event ran particularly smoothly. Of course, no-one comes to these events just for the food, but it certainly helps when this minor detail is attended to with culinary flare. The days of good ol' kiwi "Pea/pie and spud" are long gone – even in Methven. Not even parasitologist extraordinaire Graham Paltridge, who regaled us with tales of maggotry just before lunch, could disuade us from the display – including the rice and pasta salads. Well done Centrepoint – we'll be back next year.

A record number of papers were offered, with minimum cooercion (Plan C, which was to have an uninterrupted session of country music provided by the Methven Yodellers Society, did not have to be activated – much to the disappointment of all three Society members). The content and quality of presentations were a credit to all who participated, and shear delight for our esteemed Journal editor, who had gleaned many of them for later publication without having to rely on the more easily extracted, but less reliable promises usually made as the social hour progresses.

Because it is necessary to keep the South Island venue central to ensure optimum opportunity to attend, Marlborough is not a suitable site, despite much enthusiasm for a venue that could include a Wine Trail. Some exposure to Marlborough's main attraction was provided however, from a most unlikely source, (a Salvationist presenter, ably coached in the finer points of wine-speak by a local publican). The Trail was to provide brief respite from intellectual overload, but was eventually abandoned in favour of discussion on content of the programme – the level of interest shown did not fade, even after lunch.

Comment on Content: Basically Biochemistry Session (Chaired by Trevor Rolinson)

Gordon Sutton, from Medlab South opened the first session with a well contructed presentation of the introduction of a new quantitiative urine protein test. The DMA Microprotein Kit, (available from Med Bio Ltd) is readily automated, linear to 1.5g/l with C.V.<5%, detects all significant proteins. A prozone problem experienced with one batch and traced to bacterial contamination of reagent was discussed. Useful discussion followed. A good first-time presentation.

First-time presenter (veteran section) John Kitto, of Southern Community Laboratories, Christchurch, evangelised on behalf of ALT/AST ratios, exhorting us make better use of available liver panel data and provide value-added information and criteria for supplementary testing. 20% of LFTs will be abnormal, of these some 10% will not be of liver origin, warranting further investigation for evidence of muscle breakdown. All tests need to include utilisation of a certain brand of Yellow Tip to be optimally effective it seems.

John's recent discovery of several new substrates (SCL + MLS + CHL + CCL) operating in the atmosphere around Christchurch in the presence of a catalyst (RHA) is likely to add considerably to the winter smog problem on account of the amount of heat and gas generated. It is expected however that reaction equilibrium will be reached without too much damage to what is left of the godzone layer. It was gratifying to see that even though each reactant hopefully looks for signs of substrate depletion in the others, the atmosphere of professionalism was not tainted at the seminar. It will be important, over the next few years, for us to ensure that our profession is not corroded by the atmosphere in which we are all obliged to operate.

Frances Cadman, from Medlab South, presented next with a timely reminder that modern drug treatment can have its down side. Salazopyrine is a very effective anti-inflammatory agent, but can induce neutropoenia in susceptible people. It needs careful monitoring.

The sharing of uncommon cases is an important function of this seminar. Many of us work in relative isolation and on smaller population densities, with less exposure to abnormal pathology. Presentations such as these keep us in touch and alert to potential problems.

Sandi Southby, also from Canterbury Health, raised the issue of Neonatal Bilirubin, haemolysis interference and the use of Icterus index. Those of us involved with the National Womens Bilirubin survey, are familiar with the problems of comparing chemical with direct measurement systems Sandi reports an incidence of 95% heelprick collects showing haemolysis, raising the issue in discussion of lab responsibility for ensuring proper collection technique in tests such as Bilirubin where interference is highly significant. Fine tuning our CV% is of little help to the patient whose original sample is badly collected or handled poorly in transit. A good presentation on a basic issue, and a problem area for all of us in chemistry.

Mainly Microbiology Session: (Chaired by Janet Wilson)

Paul Bau, Medlab South, and another first-time presenter, gave us a good overview of Leptospirosis in New Zealand. A good reminder of our close links to rurality. Thanks to burgeoning possum population, leptospirosis needs to be considered as a potential differential diagnosis of any unwell logger, tree-topper, or tramper, as well as the better recognised occupational-hazard groups such as farmer, vets and meat workers. The disease is on the increase and will pop up anywhere in the country.

Rob Siebers, an enlightened North Islander, asked very politely if he could attend, and offered to present an update of his work as part of the Asthma Research Unit at the Wellington Clinical School. What ensued was a comprehensive paper, well illustrated (in a manner befitting our esteemed editor) on the role of the House Dust Mite as an asthma-inducing agent.

Not many of us were aware of the fact that we each shed daily enough skin to feed 10,000 mites for three months! They in turn reward us by excreting a highly allergenic protein (Derp1), which is almost impossible to avoid unless we move to Antarctica (though

Christchurch is less of a hazard than Wellington). Fascinating comparison studies of Derp1 levels in all sorts of areas revealed some interesting differences. e.g. synthetic pillows are worse than feather, and girls clothing is more hazardous than boys. Look out for the published version, it will be a useful resource for us doing the frontline allergy screening.

The Dunedin Virology team of Debbie Langford and Peter Johns presented a wide ranging overview of viral infections with graphic illustrations that left nothing to the imagination, and had many of us considering celibacy as a preferred lifestyle (fleetingly). The slides should be compulsory viewing for the anti-vaccination fringe group. A very good resume of what lurks out there. The handout was a much appreciated summary.

I was somewhat suspicious when Graeme Paltridge specifically asked for the slot just before lunch. Sure enough, the gleefully graphic dissertation on the niceties of myaisis – maggot infestation. A case of stage 2 larval Dermatobia hominis, acquired in Peru but popping up in New Zealand served as the index case for a broader look at these tropic beasties. We still enjoyed our rice and pasta salads though Graeme.

Helpfully Haematology session (Chaired by Ben Harris, even)

Brent Bishop from Southern Community Lab, Dunedin, reminded us of the need to be alert and respond promptly to the dangerous combination of Haemolysis/Elevate LFTs/Low Platelets, which can appear rapidly in pregnancy. This DIC-like syndrome has a 26% maternal mortality.

Graeme Bennett, Timaru Hospital continued the focus on coagulation problems with two separate cases. Heparin-Induced Thrombocytopenia occurs in two types, a milder type 1 which rarely drops the platelet count below 80 and recovers promptly after heparin is stopped. Type II is more serious, is immunologically based, has a delayed onset and slower recovery, with extra risk of morbidity and mortality (30%).

Graeme's second paper evoked considerable discussion on the issue of screening for the relatively common genetic defect (7%) – Factor V Leiden. This abberant Protein C is defective in the normal clotting feedback mechanism, clotting once triggered, does not stop. DVT is particularly common in this group, who require lifelong warfarin control. The APC resistance test is definitive, but is not widely available. Someone should look more carefully at a cost/benefit analysis, or come up with yet another immunochemical strip test.

Three good case presentations leading to wider discussion and much enhanced awareness.

Two first-time presenters from Canterbury Health, Diane Murton and Lia Kubala gave two very different looks at causes of methaemoglobinaemia, occurring at the opposite ends of the lifespan. Diane's case of congenital cytochrome b reductase deficiency in a neonate flown in from Greymouth, won her the Med Bio travel award for excellent presentation. Lia's equally fascinating case of brown blood in an elderly lady being crossmatched, uncovered a long list of potential causes, none of which were conclusively proven. This survivor of multiple medical interventions remains a haematological enigma.

General Session:

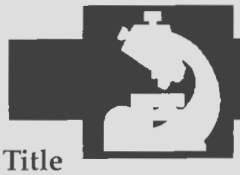
John Newton is another of the northerners seeking respite from the fast lane of Wellington life (as well as catching up with daughter Penny). An update on his involvement with the Tuatara recovery programme over the years gave us a good look at this well-known, but little understood, national treasure. Lizard libido problems don't make headlines but seemed to generate considerable interest amongst the more senior members of the audience.

Trevor Walmsley gave us an overview of the Internet, and how nerds should go about surfing on it. Fascinating stuff, but I am having difficulty working the keyboard with my toes.

Diane Whitehead highlighted a side effect of a falling road toll – fewer organ donors. Raising awareness of the donor option would facilitate harvesting of available organs. An uncomfortable topic, but one that gives hope (life, even) from others loss – Support needed.

Jan Deans discussion on seminal appraisal practices, highlighted the need for greater consensus on this matter. It needs to be addressed by a more specialised group.

Lorraine Craighead from Balclutha is currently sitting the Massey Diploma extramurally. Its content is generally acknowledged as imbalanced (even by Massey), reflecting available tutors rather than need. The discussion topic needs wider airing and Institute action.



THE NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE (INC.)

Title	**Jim Le Grice Award**
Nature	An annual award in memory of Jim Le Grice to sponsor a full time student, qualified staff technologist or qualified technical assistant to the Annual Scientific Meeting.
Eligibility	1. Any student who is a member of the NZIMLS and in full time tertiary education. 2. Any qualified technical assistant or staff technologist with less than 5 years total work experience. (Work experience to be verified on application form).
Conditions	No conditions apply to student applications. However, qualified staff will present a paper or poster at the Annual Scientific Meeting.
Applications	Applications should be completed on the official application form published in the NZIMLS Journal and available from the Executive Officer, NZIMLS, PO Box 3270, Christchurch.
Selection	Will be made by ballot by the convenor of the NZIMLS Awards Committee.
Amount	The prize awarded will vary yearly and will consist of travel to and from conference, accommodation and registration with the successful applicant making all arrangements.
Term of Award	Initially offered in 1995 and subsequent 9 years with a review at that time.

INSTITUTE BUSINESS
Office Bearers of the N.Z.I.M.L.S. 1995-1996

President
Dennis Reilly
Diagnostic Laboratory, Auckland

Vice President
Shirley Gainsford
Valley Diagnostic Laboratory, Lower Hutt

Secretary/Treasurer
Paul McLeod
Microbiology Dept., Nelson Hospital

Council
Leanne Mayhew, Chris Kendrick, Les Milligan, Trevor Rollinson, Ann Paterson

Executive Officer
Fran van Til
P.O. Box 3270, Christchurch
Phone/Fax (03) 313-4761.

Please address all correspondence to the Executive Officer, including Examination and Membership enquiries.

Editor
Rob Siebers
Dept. of Medicine, Wellington
School of Medicine, P.O. Box 7343
Wellington South.
E-Mail: rob@wn meds.ac.nz

Membership Fees and Enquiries
Membership fees for the year beginning April 1, 1996 are:

For Fellows – $98.40 GST inclusive

For Members – $98.40 GST inclusive

For Associates – $43.80 GST inclusive

For Non-practising members – $40.00 GST inclusive

All membership fees, change of address or particulars, applications for membership or changes in status should be sent to the Executive Officer at the address given above.
Members wishing to receive their publications by airmail should contact the Editor to make the necessary arrangement.

Membership Report – February, 1996

Membership	04.05.96	13.02.96	19.09.95	19.07.95
	994	1006	1079	1084
Less resignations	12	11	68	6
Less G.N.A.	8	15	7	6
Less deletions	–	–	–	–
Less deceased	2	1	2	–
Less duplications	–	–	–	–
	972	979	1002	1072
Plus applications	27	12	4	5
Plus reinstatements	3	3	–	2
Total	**1002**	**994**	**1006**	**1079**

Composition

Life Member (Fellow)	11	12	12	12
Life Member (Member)	9	9	9	9
Fellow	20	21	21	21
Member	621	618	621	645
Associate	265	258	266	311
Non Practising	49	49	50	54
Honorary	27	27	27	27
Total	**1002**	**994**	**1006**	**1077**

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE 1996 CALENDAR

28 June	Nomination forms for the election of Officers and Remits to be with the Membership (60 days prior to AGM)
1 July	Annual Staffing Survey
9/10/11 July	Fellowship examinations
19 July	Nominations close for election of Officers (40 days prior to AGM)
7 August	Ballot papers to be with the membership (21 days prior to AGM)
14 August	Annual Report and Balance Sheet to be with the membership (14 days prior to AGM)
21 August	Ballot papers and proxies to be with Executive Officer (7 days prior to AGM)
26-27 August	Council Meeting – Auckland
28 August	AGM – Auckland
27-30 August	**50th Anniversary Annual Scientific Meeting – Auckland**
6 November	QTA examinations
14/15 November	Council Meeting – Wellington
20/21 November	Specialist Certificate examinations

New Members
J. FAULKNER, ARBC, P. RYALL, V. JERAM, Wellington Pathology, S. CHAPMAN, Taranaki, L. SHARP, Valley Diagnostic, D. TURKOVIC, Valley Diagnostic, C. CAMERON Waikato, R. NICHOLLS, Medlab South, A. STADE, Wellington Pathology, G. MOORE, Canterbury Health, L. GRAYLING, Taranaki, A. JAMIESON, Palmerston North, J. CASTLE, Auckland, A. CHEETHAM, Waikato, L. CLARKE, Wellington Pathology, A. HINTON, Medlab, V. GARAM, Hutt, J. MITCHELL, Wanganui, J. WYPYCH, Hawkes Bay, L. JAYET, Medlab South, L. ROY, Dunedin, S. DE JONG, Dunedin, C. THOM, Rotorua Diagnostic, K. GAVIN, Medlab Bay of Plenty, M. WARD-ALLEN, Medlab Bay of Plenty, R. CLARK, Rotorua, K. PEARCE, Diagnostic

THE NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY SCIENCE (INC.)

Title	NZIMLS Student Award.
Donor	New Zealand Institute of Medical Laboratory Science
Nature	The purpose of this award of $200 is to encourage the presentation of scientific papers at the Annual Scientific Meeting by undergraduate students of Medical Laboratory Science.
Eligibility	All undergraduate students at a recognised course of training in medical laboratory science at Massey or Otago Universities or at the Auckland Institute of Technology are eligible.
Judging	All students wishing to have papers considered for this award are required to make an application to the convenor of the awards committee through the Executive Officer, no later than two weeks before the Annual Scientific Meeting. This application must include a brief synopsis of the paper to be presented. Responsibility for selecting the best paper will rest with the convenor who will seek the advice of appropriate special interest group convenors or the journal editor where necessary. The decision of the convenor of the awards committee will be final.
General	Papers for consideration must have been completed and presented prior to the completion of undergraduate training. Students should consider submitting the paper in publishable form to the journal editor within one month of oral presentation.

A Spectrum of Microbiology, Christchurch, September 29 – October 4, 1996

Joint ASM-NZMS meeting. This will be the biggest Microbiology Conference and Trade Exhibition held in New Zealand. All aspects of microbiology will be covered For information, contact:

ASM & NZMS '96 Secretariat
GPO Box 128
Sydney
NSW 2001
Australia
Telephone (61) 2 262 2277
Fax (61) 2 262 3135 or (61) 2 262 2323

NZIMLS 50th Anniversary Annual Scientific Meeting Reunion

If you know of any past member(s) of the profession that may have not received notification of the Reunion to be held at NZIMLS 50th Anniversary Scientific Meeting on 28th and 29th August 1996 at the Ellerslie Convention Centre, Auckland, please notify the NZIMLS Secretariat, PO Box 3270, Christchurch or tel/fax on 03 313 4761.

murex

INTERNATIONAL TRAVEL AWARD

Applications and nominations are invited for this prestigious award. The Award sends our winner to an international scientific meeting of their discipline.

This is one of the most significant awards the Institute presents and rewards individual effort in support of the profession. The award provides you with Congress fees, return airfare, plus a daily accommodation allowance to a maximum value of $5,000.

All practising Fellows, Associates and Members of the NZIMLS in disciplines associated with Immunohaematology, Immunology, Virology and Microbiology are eligible to apply or be nominated. If you believe a colleague deserves this award, then we recommend you complete the Application form as their nominator. Murex stress again, that this award is available to all laboratory personnel (NZIMLS members) in the above-mentioned disciplines.

Applications will be judged on your professional and academic abilities together with your active participation in your discipline of Medical Laboratory Science.

Applications/Nominations must be on the official form and received by the Executive Officer, NZIMLS no later than —

5.00pm, June 30th, 1996

Late applications or nominations will not be accepted.

The decision as ratified by the council of the NZIMLS will be final.

The successful person would be expected to report back to the AGM of the NZIMLS on return.

Current office bearers of the IMALT or employees of Murex Diagnostics New Zealand cannot apply.

Application & Nomination Form

murex

INTERNATIONAL TRAVEL AWARD

APPLICATION	NOMINATION

(STRIKE OUT THAT WHICH IS NOT APPLICABLE)

DATE:.....

NAME:

.....AGE:.....

(APPLICANTS NAME OR PERSON BEING NOMINATED)

ADDRESS:.....

.....

.....

PROFESSIONAL EXPERIENCE:.....

(POSITIONS HELD)

.....

.....

.....

.....

.....

.....

LIST YOUR ACHIEVEMENTS IN YOUR DISCIPLINE:.....

.....

.....

.....

.....

.....

.....

.....

.....

WHICH MEETING DO YOU WISH TO ATTEND? .....

.....

.....

.....

Additional application forms can be obtained from the Executive Officer

I wish to apply for this Murex International Travel award for the following reasons:
(In less than 200 words)

.....

.....

.....

.....

.....

.....

.....

.....

.....

.....

.....

.....

.....

.....

.....

.....

I agree to abide by the terms of the award and the decision of the judges.

SIGNED:
Nominee/Applicant: .....Date: .....
(Delete as appropriate)

This application form must be accompanied by references from:

(a) The director or senior medical officer in charge of your laboratory

(b) Any other unrelated individual.

APPLICATIONS MUST BE RECEIVED NO LATER THAN 5.00PM ON JUNE 30, 1996

POST TO: NZIMLS EXECUTIVE OFFICE, P O BOX 3270, CHRISTCHURCH

Murex Diagnostics New Zealand

P O Box 22-305

Otahuhu

Auckland 6

New Zealand

Telephone (09) 276 9970

Fax (09) 276 0704

NZIMLS CONTINUING EDUCATION

SPECIAL INTEREST GROUPS

Liftout

Transfusion Science

Special Interest Group

Convenor: Sheryl Khull, Transfusion Medicine, Palmerston North Hospital
Members: Ray Scott, Auckland Regional Blood Centre; Sue Baird, Blood Bank, Invercargill Hospital; Marie Willson, Blood Bank, Gisborne Hospital; Diane Whitehead, Transfusion Medicine, Christchurch Hospital; Suzanne Williams, Blood Bank, Dunedin Hospital, Dunedin; Kaye Fissenden, Blood Bank, Timaru Hospital; Christine van Tilburg, Auckland Regional Blood Centre

The Latest

Welcome to our newest member – Christine van Tilburg from Auckland. You can get to know Christine from her personal view of the NICE Weekend, published later in this newsletter.

Roger Austin has "gone beyond" Blood Banking and is on the "up and up" into hospital administration, so he's decided to end his time on the TSSIG. Roger has been with us since the very beginning.

Sue Baird has moved from Rotorua back to Invercargill, but is still involved in TSSIG.

Lots of people at the NICE Weekend said they felt that everyone else knew all the latest gossip. I'm sure I don't know half the gossip, but I'll publish what I've heard and maybe you can drop me a line and share the bits you know!

Palmerston North Hospital Lab has been contracted out to Med Lab Central (an SGS company), with the loss of many jobs. The new combined hospital/community lab operation is based on the hospital campus. Transfusion Medicine was left out of the contracting process – rather like the Blood Bank at North Shore, I guess. I hear that Tauranga lab is undergoing a similar process but that it will include Blood Bank.

Alison Wilson from Auckland Regional Blood Service attended the NICE Weekend with a rather large bump in her bathing suit. Congratulations and best wishes, Alison.

Is this the kind of gossip people want in the TSSIG newsletter? If so, drop me a line and tell me some more.

What else do people want from TSSIG? We do what we can to get interesting things in the journal; help with the NICE Weekend and seminars/workshops/conference; are looking for a replacement for the Transfusion medicine Audio Updates, so you can collect MOLS points without leaving home; organise examiners and generally advise NZIMLS on Transfusion Science matters. If there's something else you think you would like, just drop me a line.

1996 N.I.C.E. Weekend

Another weekend too good to just be called "nice". Over fifty participants spent the weekend "immersed" in Transfusion Science. Rather than say too much about it, I have sent some photos which I hope the editor will allow to be printed.

The winner of the Abbott award for the best presentation at the NICE Weekend was Margaret Dickinson from Auckland Regional Blood Service. Margaret told us about the factors under our control which affect the Factor VIII level of plasma we send to CSL and therefore the final product yield, and demonstrated dramatically the monetary effect that even a small improvement could make. A well-deserved win, Margaret.

The list of abstracts is published below. If you want to know more about any of the presentations, ask someone who went or contact the presenter directly. Christine's personal view of some of the papers follows.

Nice Weekend 1996

The annual TSSIG trek to Taupo took place on 12-13th April, and with trepidation I packed my presentation and headed South. Public speaking is not one of my favourite occupations, and this was to be my first Nice Weekend, so it was all a bit frightening!

However, on arrival Friday afternoon my fears were laid to rest by the friendly greeting we received. Time for a quick drink and then off to the TSSIG meeting, where I was welcomed to the team, and given the task of writing this piece.

A social evening then ensued, old friends reappeared from the mists of time, and it was far too late when I finally got to bed.

Saturday dawned too soon, and it was a race to breakfast before the start of the first session at 9am.

The first item gave a call for national unity in the service, this was to become a recurring theme as the weekend progressed, with much discussion about the need for standardization of methods, techniques and improved communications.

With 50 speakers, covering a diverse range of topics, over two very full days, there is not enough space to describe them all. Some lasting impressions were left by the Taranaki contingent with their professional, high-tech presentations. Walter Wilson, from the Blood Transfusion Trust pointed out the contractual liabilities involved in forecasting plasma production, and the financial repercussions of contaminating the CSL plasma pool.

Yersinia enterocolitica was another hot topic, with case histories, microbiological overviews, reports on progress in research and on testing. A suggested protocol for a donation testing pilot study provoked much discussion.

Sheryl's description of 'The Chillybin' for emergency caused an enthusiastic response amongst the Blood Bankers.

Transfusion reactions, antibody identification, tissue transplantation (including a graphic slide show of cadaver skin harvesting) equipment and reagents, new technology, plasma production, QC, QA, the list goes on and on. All presentations were of a professional standard and everyone came away having learnt something.

Margaret Dickinson, QC Department, ARBS, won the Abbott Laboratories NICE Weekend Travel Award, with her thought provoking presentation 'We Can Afford Quality'. She alerted us to the cost savings that would result from 0.1U/mL increase in FVIII levels in the plasma shipped to CSL. The suggestions on how to achieve this increase should prompt us to review our plasma handling practices.

I thoroughly enjoyed the whole weekend (well, maybe not giving my talk) it was great to put faces to voices heard over the phone. The chance to talk shop away from the hectic pressures of work doesn't come often. I made some new friends, ate far too much, talked too much and slept too little.

In fact I had a wonderful time, and recommend that anyone who has the opportunity in 1997 goes.

It really is a NICE WEEKEND!

Christine Van Tilburg
Production Coordinator
Blood Product Department
ARBS

Margaret Dickinson – Winner of Abbott award for best N.I.C.E. presentation receiving her award from David Ackeroyd of Abbott Diagnostics.

N.I.C.E. Presentation Abstracts

BTS – Quick March!
Ray Scott
Auckland Regional Blood Centre
Auckland Hospital
Auckland
Blood Transfusion Services in New Zealand, like other areas of the Health Service have been subjected to considerable operational and organisational change with resulting positive and negative effects. This presentation will briefly highlight the elements of change experienced throughout the service with the intention of provoking discussion on systems and direction.

Accountability in a Small Laboratory
Elizabeth Fisher
Laboratory
Masterton Hospital
Masterton
In a small blood bank a greater number of tasks fall to fewer people. How do staff remain accountable and when is enough enough.

BPR in THL
Roger Austin
Path Project
Taranaki Base Hospital
New Plymouth
Business Process Re-engineering is the 90's way of organising work processes. Progress along "Straightening the Path for Patients" will be presented along with an outline of BPR.

"Oh No! Suzy's Off to the Dentist Again!"
Bronwyn Kendrick
Department of Transfusion Medicine
Palmerston North Hospital
Palmerston North
Palmerston North Transfusion Medicine installed the Quatro SP-400 in May 1994. A light-hearted look at the teething problems of "Suzy" from then until now.

QASAR Upgrades 1994-1996
Neil Woodmansey
Quatro Biosystems
Manchester
United Kingdom
- Microplate and Gel Card Security Modules
- Blood Grouping Workforce Software
- Cooled Reagent Boats
- Automated Sample Security Module
- QASAR II

April Fool's Day
Nicola Beamish
Blood Bank
Taranaki Base Hospital
New Plymouth
A book review.

A Space Odessey
Leonie Robinson
Stores Dept.
Auckland Regional Blood Centre
Auckland
An intense investigation into the complex business of our supplies and your demands. A lighthearted show and tell session. (CSL P.E.S.)

Materials Management
Lorraine Rimmer
Auckland Regional Blood Centre
Auckland Hospital
Auckland
A brief overview of the Materials Management Quality System which we have set up at Auckland Regional Blood Service.

The ARBC Red Cell Serology Survey
Jill Faulkner
Quality Control Laboratory
Auckland Regional Blood Centre
Auckland
1. Overview of the survey content and timetable.
2. Points to note:
   - scoring variation
   - method variation
   - overall performance
3. This survey is a useful tool for Charge Technologists to track staff competency.

We Can Afford Quality
Margaret Dickinson
Quality Control Laboratory
Auckland Regional Blood Centre
Auckland
Factor VIII supply costs New Zealand citizens over $7 million annually.

Costs are incurred throughout the chain from donor recruitment to supply of CSL Factor VIII concentrates to the recipient.

On average, whole blood donors give 244 units of Factor VIII per donation and pheresis donors 670 units of Factor VIII per donation.

In the 1994-95 year, 130,000 whole blood donations and 6580 pheresis donations were processed to satisfy the NZ demand (7,200,000 units of Factor VIII).

Factor VIII is an extremely labile clotting factor but with care and protection, survival is increased dramatically.

Survival → Improved Yield → Reduced Costs
Opportunities within **our** control for improving Factor VIII survival include donor selection, anticoagulant selection, collection technique, processing times, plasma storage and transport temperatures.

QA Management Masters Degree, Massey University
Malcolm Rees
2nd Field Hospital
Linton Camp
Palmerston North
A quality management/quality assurance paper is being proposed for the new Master of Medical Laboratory Science degree being offered at Massey University. The paper will be organised and run from the Department of Production Technology who have the expertise in quality practices.

This paper describes a report being prepared by myself providing a link between the two groups. Comments are welcome on the subject matter being considered for inclusion in the report.

General topics being considered for inclusion are as follows. Principles of quality management, developing a quality management system, services and service quality, total quality management, quality improvement, statistical quality control, occupational health and safety.

Laboratory specific topics being considered are as follows: Legislation and regulations, accreditation, activity measurements, control charts, calibration and verification of standards, error (human or otherwise), imprecision, competency testing, near patient testing, method comparisions, rational use of laboratory tests.

"Whilst the Trust Understands Your Concerns, It Has No Responsibility" ...
Max Love
Blood Bank
Hutt Hospital
Lower Hutt
Report of problems faced by a Blood Centre when a fund raising campaign to raise funds for a neighbouring centre was run within its donor collection area.

Chaga's Disease Screening in Christchurch
Steve Gibbons
Canterbury Health Laboratories
Christchurch Hospital
Christchurch

CJD and Blood Transfusion
Fiona McCormack
Blood Bank
Taranaki Base Hospital
New Plymouth
CJD has become a newsworthy item recently as the result of a highly publicised case at Taranaki Health Care Ltd. This presentation reviews the world literature on the disease and its transmission by blood transfusion and highlights some areas of concern at the way the media handled their investigation.

Report of Hepatitis C Lookback Programme

Andrew Mills
Waikato Blood Transfusion Services
Waikato Hospital
Hamilton
The Health Department initiated Hepatitis C testing of all blood donations in New Zealand in July 1992, however a Hepatitis C antibody test became available for usage within New Zealand during 1990. The New Zealand Blood Transfusion Service advocated the introduction of Hepatitis C testing of all blood donations in 1990. When the Health Department decided to undergo a retrospective Hepatitis C testing programme they chose the dates of August 1990 to July 1992 to test donations from donors who had not subsequently donated blood. My presentation is a summary of the Hepatitis C Lookback Programme conducted by Waikato Regional Blood Centre.

Yersinia the Hidden Risk
Lola Prikkel
Blood Bank
Auckland Hospital
Auckland
Having recently given up piles of agar plates, Bunsen burners and anaerobic smells for test tubes, eye pieces and red cell agglutination I find I cannot get away from micro-organisms.

This talk will touch on microbiology and clinical aspects of Yersinia.

All Infected Blood Transfusion
Patricia Joy
Blood Bank
National Womens' Hospital
Auckland
A case presentation of a fatal transfusion reaction to Yersinia infected blood at Greenlane Hospital, Auckland in October 1995.

Yersinia Detection in Autologous Donations
Grant Bush
Blood Bank
Tauranga Hospital
Tauranga
Having reduced our storage time of allogenic blood down to twenty-one days, I decided to investigate the storage time of autologous donations.

It was not possible to decrease the length of expiry of the autologous donations so I investigated testing the donors and/or donations for yersinia enterocolitica infection.

I looked at methods for the detection of yersinia antibodies, antigens and enterotoxins, but found that no method proved of value at this time.

Economics of Y. enterocolitica Testing
David Fisher
Laboratory
Masterton Hospital
Masterton
A comparison of the cost of short dating donor blood versus extended storage following testing for Y. enterocolitica.

Yersinia enterocolitica – Report on Progress of Current Research at Massey University
Chris Kendrick
Dept of Microbiology and Genetics
Massey University
Palmerston North
The problems of blood contaminated with the organism **Y. Enterocolitica** have become alarmingly evident on the NZ scene in the late '80s and 1990s. Possible reasons for the introduction of this organism into the blood supply are presented along with a report on the developing research programme investigating this problem at Massey University.

New Kid on the Block
Diane Matheson
Transfusion Medicine
Lakeland Health Ltd
Rotorua
A case study of a recent Rh antibody titre is presented with some interesting post-natal developments.

Detective Work
Debbie Mason
Auckland Regional Blood Centre
Auckland
The Red Cell Serology Laboratory at the Auckland Regional Blood Centre often receives referred samples for antibody identification and/or further investigation We have recently had several case studies This presentation will include a brief overview of some of these and detail one in particular.

Microwave Elution
Leisa Cournane
Blood Bank
Southland Hospital
Invercargill
To evaluate the effectiveness of microwave radiation in dissociating warm reactive IgG from red cells, Rubins' Diethyl Ether method was compared to that of microwave radiation. Seventeen samples of red cells with known phenotypes were incubated with specific antisera in order to sensitise the red cells. DATs were then performed on the samples to ensure that the cells were sensitised. Once sensitised the two different methods for obtaining an eluate were performed on the antibody coated red cells. The eluates were then put up in parallel with screening cells and some with panel cells by ICT to detect and identify any antibody present. Antibodies were detected and identified successfully in 16 out of 17 eluates obtained by microwave treatment and in 15 out of 17 eluates obtained by Rubins' Diethyl Ether. Obtaining an eluate by microwave treatment takes 10 minutes while obtaining an eluate by Rubins' Diethyl Ether takes 1 hour. Microwave treatment also produces more eluate than Rubins' Diethyl Ether

Of Course it's Negative – NOT!
Raewyn Clark
Transfusion Medicine
Lakeland Health Ltd
Rotorua
The case of the unexplained anti-D and its impact on the testing regime of blood donors is examined.

Tails of the Unexpected
Adrienne McKay
Canterbury Health Laboratories
Christchurch Hospital
Christchurch
A case study involving the incorrect assumption of a genotype of an antenatal patient presenting with atypical red cell antibodies.

Titre-ing on the Brink
Tony Morgan
Immunohaematology Department
Napier Hospital
Napier
Antenatal testing may be performed by two laboratories in Hawkes Bay or one in Christchurch.

If an antibody worthy of titrating is discovered, this titration could then be done by a different laboratory in Christchurch or even one in Auckland! Has the time come for "standardisation", within laboratories, of titration techniques and if so, which test book will be our reference!

To Screen . . . . . Or Not to Screen
Susan Duncan
Wanganui Diagnostic Laboratory
Wanganui
A review of antenatal screening over the past eight years in a small provincial laboratory.

How Come?
Faye Martin
Immunohaematology
Memorial Hospital
Hastings
A brief presentation showing how an incorrect result given to the Immunohaematology Department can lead

to results with the query, *"How Come"*?

Albumex ™ The First Year of Production
Vito Micucci
CSL Bioplasma
Broadmeadows
Australia
The manufacture of Albumex 5 and Albumex 20 commenced at CSL's new Broadmeadows facility in October '94. This product replaced 5% NSA and 20% NSA in the second half of 1995. Albumex is manufactured using a combination of Cohn fractionation and chromatographic methods. Data from production batches demonstrates that this process consistently generates albumin of high purity (>99% albumin), high monomer content (>99%), low albumin levels (10ppb) and exceptionally low endotoxin levels.

This presentation will outline the results obtained thus far, and compare the properties of albumin produced at the Broadmeadows plant, Albumex, with CSL's previous product, NSA.

The clinical relevances of these differences will also be discussed.

It's freezing But It's Fun
Christine Van Tilburg
Auckland Regional Blood Centre
Auckland
A brief review of the manipulation and storage of stem cell, bone marrow, cord blood harvests, performed by the Blood Products Cryogenics Department at ARBS.

Babe in the Blood Bank
Stephen Silk
Blood Bank
Hutt Hospital
Lower Hutt
A current perspective on special blood and component transfusion requirements for the neonate.

How Low Can You Go?
Jacqui Jones
Auckland Regional Blood Centre
Auckland Hospital
Auckland
In late January, three Jk(a-b-) units were collected and sent to Hong Kong on the day of collection. This paper outlines the temperatures recorded by the accompanying data logger.

Potassium in Stored Blood
Anne Jamieson
Dept of Transfusion
Palmerston North Hospital
Palmerston North
Potassium is the major intracellular cation, maintained at a high concentration by the sodium/potassium pump. In stored blood, declining energy levels lead to a rise in plasma potassium. When transfused, high potassium concentrations can interfere with normal neuromuscular function and place demands on the renal system to restore homeostasis.

Is Fresh Whole Blood Straight From the Donor Ever Indicated?
Warwick Henry
Blood Bank
Nelson Hospital
Nelson
Very occasionally we are pressured by surgeons to provide fresh whole blood for patients whose situation is desperate and there seems to be little left to control haemorrhage. Despite the appropriate blood components being available, there seems to be some "mystery" in fresh whole blood.

"Just Give Me Some Blood NOW!"
Sheryl Khull
Dept of Transfusion Medicine
Palmerston North Hospital
Palmerston North
A very small minority of patients are exsanguinating so rapidly that there is no time for any kind of pretransfusion testing. The challenge is to keep them alive for those critical first few minutes until compatible blood is available. Together with our Emergency Department, we have put together a system which provides blood fast when the need is truly urgent.

A Non-Layered Cocktail
Sue Evans
Wellington Regional Blood Services
Wellington Hospital
Wellington
Nycroprep™ Mixer is a new product for the spreading of lymphocytes from peripheral blood. Experiments were carried out in parallel with our standard methods to assess the Nycoprep™ Mixer's suitability for routine use in my laboratory.

DNA Platelet Typing
Kathie Figgins
Tissue Typing Laboratory
Auckland Regional Blood Centre
Auckland
Where we are at with a new technology.

DNA Technology – Past, Present and Future
Holly Perry
Auckland Regional Blood Centre
Auckland
DNA Technology is going space age!
A review of where we've been and where we're going.

The Risks of Plasma Supply
Walter Wilson
Blood Transfusion Trust
Wellington

Update on the Blood Transfusion Service and the Trust
Walter Wilson & Steve Gibbons
Blood Transfusion Trust
Wellington

A Problem Crossmatch
Belinda Reilly
Waikato Regional Blood Centre
Waikato Hospital
Hamilton
This will just be a small presentation about a patient from Rotorua who we received blood on to crossmatch 3 units. The person ended up having anti-Jk^a and anti-Jk^1. The talk will be about the outcome of my panels, patient antigen typing, use of 2 molar urea and crossmatching with a Jk(a-b-) donor to come to my conclusion.

Cadaver Solid Organ Transplant Statistics – 1995
Sandy Beckman
Tissue Typing
Auckland Regional Blood Centre
Auckland
A review of the Cadaver Donors tested at the Auckland Tissue Typing Laboratory in 1995. Comparing the origin, blood groups and subsequent distribution of the retrieved organs with previous data.

Your Chance of a Match
Margaret Ushakoff
Tissue Typing
Auckland Regional Blood Centre
Auckland
The chances of a matched sibling bone marrow transplant. A statistical look at the 93 families tissue typed in 1995.

Skin Alive!
Teh Liew Cheng
Tissue Bank
Auckland Regional Blood Centre
Auckland
ARBS Tissue Bank was set up in July 1995. The bank processes donated cadaver skin tissue for use in patients suffering from burns or chronic ulcers. The bank will include other tissues like femoral heads and cadaver bones in the near future. Current human tissues that are banked in Auckland include bone marrow cells, peripheral stem cells, corneas, heart valves, sperm, ova and bones. The Skin Bank is part of the ARBS Tissue Bank. The operations of the skin bank involve the:
• accepting potential cadaver donors through the Tissue Transplant Co-ordinator

- screening of cadaver donors
- harvesting or procuring of the skin from the cadaver
- processing of the skin
- packaging of the skin
- controlled freezing of the skin
- storage of the skin
- accreditation of the donation
- issue of the skin

These operations are governed by the strict code of GMP as in the processing of blood and blood products.

What More Could We Have Done?
Alison Wilson
Auckland Regional Blood Centre
Auckland Hospital
Auckland
A 53-year-old multiparous Nuiean woman was admitted for surgery for a pelvic tumour. She had no previous history of transfusion and was post-operatively transfused three units of red cells, issued on the basis of a negative antibody screen. Nine days later she was noted to be pale and jaundiced and her haemoglobin was 25g/L. The resulting investigation of the transfusion reaction will be discussed.

Baby; Look What You've Done To Me Now!
Ailsa Signal
Department of Transfusion Medicine
Palmerston North Hospital
Palmerston North
An unusual and complicated case of Placenta Praevia involving massive transfusion is presented.

ABO-Incompatible Blood Transfusion
William Perry
DiaMed New Zealand
Auckland
It is the thing that all blood bankers dread. We set up our techniques and procedures to prevent it from happening.

We hope and believe it will never happen in our laboratories.

It happened in my laboratory once. This presentation will describe the incident and discuss the lessons that we learnt from it.

Transfusion Reactions
Simon Campbell
Blood Bank
Tauranga Hospital
Tauranga
Fred, Hilary, Alan and friends join us this weekend to highlight the clinical picture associated with the dreaded phone call that we hate to hear . . .

Transfusion Reactions – 99% Routine
Gerry Heta
Auckland Regional Blood Centre
Auckland Hospital
Auckland
The interesting 1% are examined.

Biochemistry

Special Interest Group

Convenor: Alison Buchanan
Clinical Biochemistry
Main Building
Auckland Hospital
Ph (09) 307 4949
Ext 7553
Fax (09) 307 4939

The Biochemistry Special Interest Group is arranging a one day seminar at the August conference in Auckland.

The Title:

Something old, something new
What you hear ain't always true
Are things topical always true?
Join us here and share your view.

The Topics:
Sweat testing and Cystic Fibrosis – Why is the old test still vital for C.F. diagnosis? Ask a C.F. clinician.
Troponin I, T or whatever – Which letter will they think of next?
Faecal Pancreatic Elastase – Aha! So you thought faeces belonged in the Microbiology Lab.
Maternal Serum Screening – What are they doing it for? What does it mean? Come along and find out.

Other topics to be advised. More information is available from the convenor at the address above.

Kindly fill out the registration form on the blue flyer within this journal.

Come along and join us for a great day. See you at conference.

P.P.T.C. News – The Samoa Course

In November 1995 Mike Lynch, the Tutor Co-ordinator and Marilyn Eales, the NZIMLS representative on the PPTC Management Committee, visited Western Samoa to conduct the final examinations for the second cycle of the course.

Three students satisfied the examiners in both written and oral examinations and passed in all four subjects – Haematology, Microbiology, Biochemistry and Blood Bank. Four students did not meet the examiners requirements. Reasons for this have been analysed and recommendations for improving some aspects of the course have been made to the appropriate authorities.

A very colourful graduation ceremony was held on Thursday, 7th December, 1995. The laboratory staff of the hospital are to be congratulated on all their hard work to make this ceremony the success that it undoubtedly was. The floral decorations in the hall, the beautiful leis presented to the graduates and officials participating in the ceremony and the refreshments and food provided after the official function were all provided by the laboratory staff at the hospital. The tables were laden with delicious Samoan food.

An outline of the graduation programme is itemised below:

4.45p.m. Guests seated.
5.00p.m. Welcome Speech
Dr. V.F. Asaua, Director of Laboratory Services.
5.15p.m. Prayer
Rev. Sulufaiga Samasoni
Hymn: Vivi'i atu ia
Laboratory staff.
6.00p.m. Official opening
Hon. Sala Vaimili II, Minister of Health.
6.20p.m. Graduation Address
Dr. D. Parkinson, WHO Representative.
6.40p.m. Awarding of Certificates
Dr. Taulealeausumai LTE Enosa, Director General of Health.
Hymn: Pese 371
Laulau Mai Pea – Laboratory staff.
7.00p.m. Overview of training programme
Mr. M. Lynch, Co-ordinator PPTC
Address to graduates and tutors
Marilyn Eales, NZIMLS Representative P.P.T.C. Committee.
7.30p.m. Speech by Graduate.
Makerita Leolaga.
Christmas Carol – Laboratory Staff
Closing Remarks – Organising Committee

The Pacific Way

Points to note from the Samoan Visit

1. The agreement between the Samoan Government and the New Zealand Foreign Affairs Department via the PPTC is one of *true partnership*. New Zealand providing the course content, reference material and conducting the assignments and examinations. The senior staff of the laboratory at Central Hospital Apia, using the material and providing lectures and guidance to the students. It is true there are problems and some of these have been identified. For the partnership to work it requires a conscientious effort by staff of the PPTC and by the senior staff of the Laboratory Services in Apia.
2. A satisfactory air conditioning unit was only installed in the Haematology Department when a Cell Dyn Automated Cell Counter was purchased!!! It seems that no matter what the daily temperature, expensive machines warrant air conditioning more than people. Many laboratories in New Zealand have experienced the same situation. The air conditioning unit in the hospital in Apia is now so satisfactory that everyone heads for the Haematology Department on arrival at the Laboratory Services to cool down.

The Third Three Year Training Course

This course began in February 1996 with six students. The students appointed are:
Lailani Eli
Lulu Ah Mu
Uili Pesata
Itugi'ia Eti
Susau Savei

Risk of Relapse in Leprosy

Until the introduction by WHO of the standard regimes using multidrug therapy (MDT) for the treatment of leprosy, there was a general unwillingness to release patients from treatment. This was mainly due to the high risk of relapse after Dapsone monotherapy. After almost a decade of MDT implementation and after releasing more than 4 million patients, it was necessary for WHO to review the risk of relapse following WHO-recommended MDT. The results of this study carried out on more than 20,000 Multi-bacillary (MB) and 50,000 Pauci-bacillary (PB) patients, revealed that the risk of relapse is very low, 0.77% for MB and 1.07% for PB, nine years after stopping MDT. In comparison to Dapsone monotherapy, the risk is ten times lower. Thus, over the last decade MDT implementation has probably prevented close to half a million relapses.

Blood Bank Training Workshop, held at the Province Hospital, Qui Nhan, Vietnam 25 September – 30 September 1995

The workshop was of a five day duration and was run by Mr Stewart Dixon, Medical Laboratory Scientist, PPTC, Wellington, New Zealand.

The course covered the basic aspects of blood bank technology and was attended by 24 technicians from the Province Hospital and district hospital laboratories.

Mr Nguyen Van Tho acted as interpreter throughout the whole workshop and without this invaluable contribution the workshop would not have been possible.

Two trainees were identified in this group as candidates for further training at the PPTC in New Zealand.

Makerita Leolaga giving her speech on behalf of the graduates. In background from left Dr V F Asaua, Tilau Lopa and Fetalaiga Vasa.

Tilau Lopa (Tokelau) receiving the award for top student.

The tutors of the course: Malo – Microbiology; Fai'inu – Acting Charge Technologist; Tala – Blood Bank; Back, Fa'apulo – Biochemistry.

Tutor co-ordinator PPTC Mike Lynch and Tutor Tala – Blood Bank.

Dr V F Asaua, Director of Laboratory Services.

New Products and Services

New From bioMérieux
Albicans ID

For immediate isolation and identification of C.albicans, try Albicans ID media. Albicans ID is ready-to-use plated media, which enables isolation of yeasts and immediate identification of Candida albicans (70 to 80% of the isolated yeasts of superficial samples are C.albicans). C.albicans are coloured blue due to a specific hexosaminidase chromogenic substrate incorporated in the medium. Other yeasts give white colonies.

With a sensitivity of 97% and a specificity of 99%, Albicans ID shows superior performance compared to classical identification tests for C.albicans. Furthermore, Albicans ID shows very clearly the presence of multiple yeasts in the same sample

Inhibition of bacterial flora is achieved by combining two antibiotics (Gentamicin and Chloramphenicol) within the medium.

This medium is becoming very popular. For more information, please contact us.

Med-Bio Enterprises Ltd, Phone 03 349 4950, Toll Free 0800 733 599

New from Socorex
Colour Code
CALIBRA Micro-pipettes

The CALIBRA micro-pipettes are based on the Solid Calibration system, providing for both digital entry and digital display of the volume.

Two cylindrical cams fitted with precalibrated steps give the instrument high performance and long time calibration stability. The two positions of the volume setting wheel, one for each cam, allow an instant, easy volume selection and eliminates tedious windings.

The modern design and smooth shape of the pipette allow it to fit easily in the hands and all models have colour coding related to the size of the tips used.

The line includes single channel pipettes covering volumes from as low as 0.2µl up to 10ml.

Lightweight and soft plunger stroke care for user comfort, allowing fatigue-free operation. Robustness and durability are guaranteed even though repeated autoclaving at 121°C, fully assembled.

Med-Bio Enterprises Ltd, Phone 03 349 4950, Toll Free 0800 733 599

New Catalogue for 1996

Many new products have been introduced to DAKO's expanding line of products. DAKO supplies a wide spectrum of high-quality antibodies, monoclonal and polyclonal, for both diagnosis and research. They also have a wide range of products for Clinical Immunochemistry, Clinical Microbiology and Flow Cytometry. The latest addition to the DAKO range is a fully automated Autostainer.

The 1996 catalogue is colour coded, which makes it very easy to find what you are looking for. For your free copy, please contact us.

Med-Bio Enterprises Ltd, Phone 03 349 4950, Toll Free 0800 733 599

Pharmacia and Upjohn Diagnostics (formerly Pharmacia Diagnostics)

New Zealand Medical and Scientific Ltd has been appointed New Zealand distributors for Pharmacia and Upjohn Diagnostics. Allergy diagnostics constitutes the major area of business for Pharmacia and Upjohn Diagnostics. Products are also marketed for asthma monitoring "ECP UniCAP" and CDTect™ for monitoring alcohol related disease.

UniCAP™ *In-Vitro* Tests in the Diagnosis of Allergy

"The UniCAP 100 Instrument is structured to handle all the steps from request to result. It automatically distributes the samples, immunocap and reagents and processes all the steps from incubation, washing, measuring to calculation. The UniCap *In-vitro* immunoassay system is fast, fully integrated and automated. In a single desk top unit, UniCAP lets you complete up to 48 different determinations from among 450 different allergens, complete with automatic documentation in less than 3 hours. UniCAP gives you – a quantitative method for measurement of IgE antibodies in serum – clinically relevant allergen panel with more than 400 allergens – and effective anti-inflammatory treatment can be followed by measuring serum ECP"

For more information contact New Zealand Medical and Scientific Ltd, free phone 0508-634 1036.

CSL Biosciences established in New Zealand

Effective May 1, 1996, CSL Biosciences Division will commence operation in New Zealand.

Judy Woodard, formerly from the Auckland Regional Blood Centre will join CSL Biosciences as the Technical Product Specialist based in Auckland.

Under the new structure a number of activities will take place.

CSL will launch a range of Reagent Red Blood Cells sourced from New Zealand donors. These include TrioNZ™, AntiglobNZ™ & ReverNZ™.

Another major change will be the transfer of the Ortho Diagnostic & Hemoliance product group (MLA) from Intermed Scientific.

The Oncor products for "In Situ Hybridization & Molecular Biology" will also be marketed by CSL Biosciences.

Medica Pacifica Ltd will continue to provide Logistic & Market Management of the CSL Biosciences products.

For further inquiries please call George E Bongiovanni on Freephone 0800-688 882 or Freefax 0800-688 883.

New Cytometry Products from Coulter

Flow Count™

Flow Count™ fluorospheres allows an absolute count to be obtained directly on a flow cytometer. The product consists of polystyrene fluorospheres at a known concentration which are stable in both, size and fluorescence intensity. The concentration of fluorospheres is an assayed value allowing absolute count determination on any sample directly on the Coulter Epics XL in real time. Flow Count™ fluorospheres is a ready to use product.

New Monoclonal Antibodies

CD95-RD1	P/N 6607003	CYOT-STAT™CD3-PC5	P/N 6607010
CD28-FITC	P/N 6607004	CYOT-STAT™CD8-PC5	P/N 6607011
CD28-RD1	P/N 6607005	CYOT-STAT™MsIgG1-PC5	P/N 6607012
CD31-RD1	P/N 6607006		

For more information please contact:

Coulter Electronics Pty Ltd on Free Call 0800 442 346

Advertisers In This Issue

NZIMLS 50TH ANNIVERSARY ANNUAL SCIENTIFIC MEETING
27 - 30 AUGUST 1996
REGISTRATION FORM

SECTION A:	**DELEGATE DETAILS**	
Title [Prof/Dr/Mr/Mrs/Ms]	Family Name	Given Name
Name to appear on badge:		
Institution/Organisation		Position held
Contact Address:		
Telephone:		Facsimile:
Major Interest (Disclipine):		
ACCOMPANYING PERSON(S)		
Title	Family Name	Given Name
Title	Family Name	Given Name

SECTION B:	**REGISTRATION**		
Category	Early Bird Registration before 30 June 1996	Registration after 30 June 1996	Total
Full Registration - Member	$320.00	$370.00	
Full Registration - Non Member	$400.00	$450.00	
Full Registration - Full-time Student	$200.00	$250.00	
Day Registration - Member	$150.00		
Day Registration - Non Member	$175.00		
Day Registration - Full-time Student	$100.00		
TOTAL: SECTION B			NZ$

SECTION C:	**ACCOMMODATION**
Quality Hotel Logan Park ❑ Single ❑ Twin ❑ Double @ $110.00 + GST per night per room **A deposit of one night's accommodation of $110 + GST must be paid to secure booking.**	NZ$
I have arranged to share with..... **(If two people share a room it is important for one person only to make the reservation on their registration form and send ONE deposit).**	
Arrival/Check in Date:..... Approximate Arrival Time:.....am/pm Departure/Check out Date:.....	
Please send me information on: ❑ Admiral Motel ❑ Remuera Motor Lodge ❑ Other	

SECTION D:	SOCIAL PROGRAMME		
Date	Event	No. of Tickets Required	Amount
27 August 1996	Jim Le Grice Ice Breaker (included in Registration Fee) Extra Ticket(s) at $20.00 per ticket		- $
28 August 1996	Casual Dining (to be paid on the night)		
29 August 1996	NZIMLS 50th Anniversary Golden Ball at $65.00 per ticket		$
TOTAL SECTION D:			NZ$

SECTION E:	FRIDAY WORKSHOPS	30th August 1996
90 Minute Sessions - Limited to 30 registrants		**Full Morning Workshops - Limited to 60 Registrants**
1. Latest Developments in Computing Software/Network		10. Surviving Corporate Culture
2. Temperature Measurement		
3. Barcode Technology		
4. S.O.S. Safe Management of Hazardous Substances		
5. Advances in Laboratory Automation and Related Systems		11. Site Visit: Starship, Auckland Hospital, Philson Library, Medical Books Skin and Tissue Bank, ARBC
6. Generation and Application of Data on Biological Variation (Reference Ranges)		
7. Reengineering in the Laboratory		
8. Doing Better Business by Phone		
9. Telecommunications Hardware Solutions		

Please list three workshops you would like to attend in order of preference:

1. _____

2. _____

3. _____

SECTION G:	PAYMENT OF FEES
Section B: Registration Fees	$
Section C: Accommodation	$
Section D: Social Programme	$
TOTAL SECTION G:	NZ$

Please complete the form, retain a copy for your records and mail immediately with your payment to: NZIMLS Conference Secretariat, P O Box 3270, Christchurch, New Zealand.

Please make cheques payable to: "NZIMLS 96 Conference"

"going for gold"

SOME OTHER VIEWS YOU'LL DISCOVER

WHILST WORKING FOR CORINTH MEDICAL

In order to get the most out of your working holiday in Britain, you'll want the expert care you can only gain from Corinth. With almost thirty years experience we've grown to be the leading specialist Employment Agency in our field by helping Medical Laboratory Scientists from all over the world combine business with pleasure; giving them the opportunity to work with the UK's leading hospitals and helping them take time out to enjoy the different cultures and sights. What's more, as well as top rates of pay and an excellent package of benefits, you won't be tied to any binding contracts and you won't be charged for our services.

We'll simply offer you the widest selection of jobs and supply friendly and confidential advice on all aspects of working through an agency.

Want us to put you in the picture?

If so, complete the coupon below and send it to our Recruitment Manager, **Kate Williams** AIBMS and she will send you our FREE 'Working Holidays in Britain' Information Pack. Alternatively, you can **Call Us Toll-Free** on **0800 441586** (24 hours).

I'd like to know more
about Corinth Medical.
Please send me your 'Working Holidays in Britain' brochure.

Name _____

Address _____

_____

Tel: _____

Anticipated date of travel: _____ Month _____ Year BSNZ

Corinth Medical

Post to: Corinth Medical, 5 Theobald Court, Theobald Street Borehamwood, Hertfordshire WD6 4RN. ENGLAND
Tel: 00 44 181 207 0234 Fax: 00 44 181 207 6894

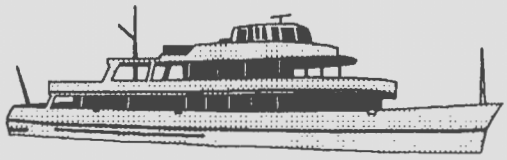
THE PROFESSIONAL STAFFING AGENCY

Immunology

Special Interest Group

Pre Conference
WORKSHOP & AGM
ON TUESDAY AUGUST 27 1996

The Auckland Branch Invites you to the City of Sails
for a Discussion Workshop
(Virology/Immunology)
and
the Annual General Meeting of ISIG
while
CRUISING ON THE BEAUTIFUL WAITEMATA HARBOUR



PROGRAMME: AGM
LIGHT LUNCHEON
DISCUSSION WORKSHOP

TOPICS:

- Proposal to draw up a Master Directory of test referrals
- Progress with Degree Courses
- Technical Problems relating to Testing
- Quality Control
- Open Forum for your Ideas.

COST: $25

Full details will be supplied on receipt of your Registration Form (below).

Registration Form for ISIG Cruising Workshop and AGM

NAME: .....

CONTACT: .....

IDEAS FOR DISCUSSION: .....

.....

Closing date for Registration: Friday 19 July 1996

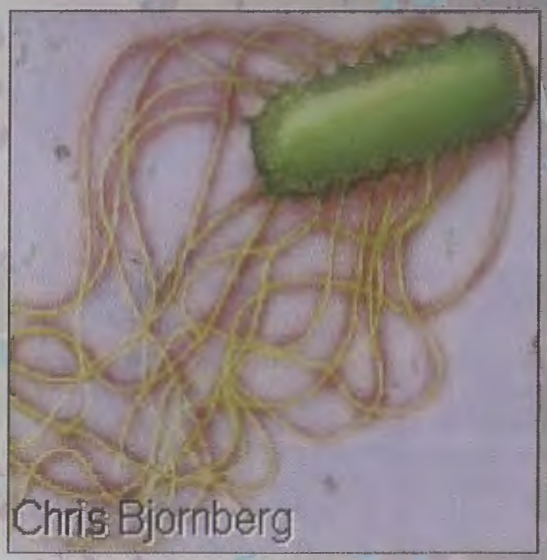
Please make cheques payable to ISIG Workshop and send with Registration Form to:

David Haines
Virology/Immunology Dept
Auckland Hospital
Private Bag 92024
AUCKLAND

WELLCOLEX *E.coli* 0157

ESCHERICHIA

COLOURED LATEX



Escherichia coli 0157

Distinguishing features :

Gram negative bacillus, with peritrichous flagella ; non-sorbitol fermenting and verocytotoxigenic. Responsible for food poisoning with potential for severe complications including haemolytic uraemic syndrome and haemorrhagic colitis

Identification of *Escherichia coli* 0157

- **Latex agglutination test**
  simple, proven technology enabling one-off testing and speedy turnaround times
- **Smooth red latex**
  ensures clear interpretation and accurate results
- **Test time of only 30 seconds**
  rapid diagnosis and prevention of serious complications
- **Complete kit**
  including controls, sticks and cards so there are no hidden costs to hamper budgeting
- **Excellent Sensitivity and Specificity**
  accurate diagnosis

Wellcolex *E.coli* 0157	ZC60 (50 tests)
Test Latex (pink cap and label)	1 dropper bottle (2ml)
Control Latex (grey cap and label)	1 dropper bottle (2ml)
Positive Control (red cap and label)	1 dropper bottle (2ml)
Negative Control (blue cap and label)	1 dropper bottle (2ml)
Disposable Reaction Cards	40
Disposable Mixing Sticks	3 x 100

Enteric Range: Antisera, Wellcolex Colour Salmonella, Wellcolex Colour Shigella, Wellcolex *E.coli* 0157

murex

Experts in Enterics

murex

PH. : (09) 276 9970
FAX: (09) 276 0704

50 years

The New Zealand Institute of Medical Laboratory Science

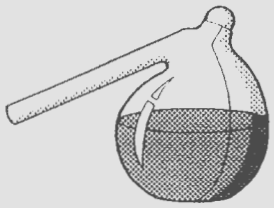
NZIMLS 50th Anniversary Auckland Scientific Meeting

27th - 30th August 1996

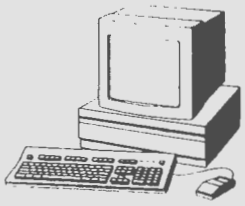
"going for gold"

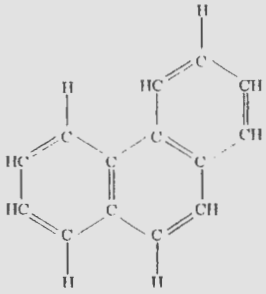
BIOCHEMISTRY SPECIAL INTEREST GROUP

SEMINAR



Something old something new
(What you hear ain't always true).
(Are things topical always true)?
(Join us here and share your view)

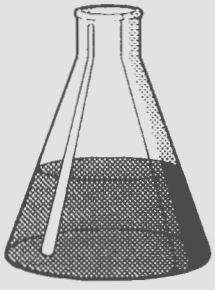




Venue: Conference Venue
Ellerslie Convention Centre
Ellerslie Race Course
Greenlane Road
Auckland

Date: Tuesday 27th August 1996

Time: Coffee served at 10.30 hours
First session at 11.00 hours
Lunch and Afternoon Tea provided



Topics to include:

- Sweat testing and Cystic fibrosis
- Tropinins
- Faecal pancreatic elastase
- Maternal serum Screening

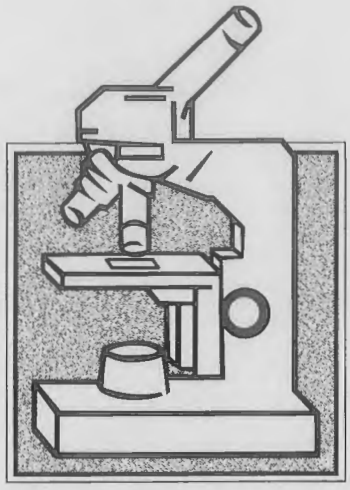
For further information - see BSIG notes in the NZIMLS Journal

✂----------✂

Registration Form for BSIG Seminar to be held on Tuesday 27th August 1996 commencing at 10.30am		
Name:		Telephone:
Laboratory Contact Address:		
Registration fee:	NZIMLS Member $35.00 Non NZIMLS Member $45.00	Amount Enclosed
To register, please send completed form and cheque for the appropriate amount to the Conference Secretariat, P O Box 3270, Christchurch.		

POSTGRADUATE PROGRAMS

DIRECT ENTRY INTO A MASTERS FOR REGISTERED MEDICAL LABORATORY SCIENTISTS



At Queensland University of Technology, we offer direct entry into our masters programs for medical scientists who are registered with the New Zealand medical laboratory scientists registration board. So, if you're looking to upgrade your qualifications and enhance your career, QUT is the ideal place to start.

FACULTY OF SCIENCE POSTGRADUATE COURSES

The faculty offers a range of postgraduate research and coursework programs in medical science.

PhD and research masters include specialisations in:

- chlamydia diagnosis and control
- protein based diagnostics
- arbovirus pathogenesis program
- DNA based diagnosis
- molecular endocrinology of cancer.

A coursework masters degree in medical science is available through the Master of Life Science.

We also offer a Graduate Diploma in Biotechnology.

QUT'S POSTGRADUATE PACKAGE

- world-class research laboratories and centres like the Cooperative Research Centre for Diagnostic Technologies which support postgraduate courses and research programs
- research programs that can be commenced in Australia and completed in New Zealand
- expert staff, many of them practising professionals and leading researchers
- coursework masters which can be completed in three semesters or one calendar year
- flexible study programs designed to meet the demands of your busy lifestyle

For more information, phone +61 7 3864 2917, fax +61 7 3864 1534 or email j.vidgen@qut.edu.au

QUT

A university for the real world

Queensland University of Technology GPO Box 2434 Brisbane Australia 4001 World Wide Web Site: http://www.qut.edu.au/

DAKO

NEW ANTIBODIES

For use in Immunohistochemistry

DAKO MONOCLONAL MOUSE ANTI-HUMAN

FOR USE ON ROUTINELY PROCESSED TISSUE

Progesterone Receptor

■ **Reacts with the nuclei of cells from various histologic subtypes of breast carcinoma including infiltrating ductal, lobular, mucinous and carcinoid. Labels the nuclei of normal breast and uterine epithelial cells on routinely processed tissue. This product is an excellent addition to an extensive line of breast marker antibodies.**

DAKO Progesterone Receptor, 1A6. Product code M3529.

DAKO POLYCLONAL RABBIT ANTI-HUMAN

FOR USE ON ROUTINELY PROCESSED TISSUE

pS2

■ **Reacts with the pS2, or pNR-2, protein. pS2 protein is an indicator of a functional oestrogen regulatory system. pS2 is a potential prognostic indicator for the clinical responsiveness of breast carcinoma to hormone therapy. This antibody labels epithelial cells in a number of primary mammary carcinomas.**

DAKO pS2. Product code A0095.

DAKO POLYCLONAL RABBIT ANTI-TdT

FOR USE ON ROUTINELY PROCESSED TISSUE

TdT

■ **Terminal Deoxynucleotidyl Transferase (TdT) is expressed by the majority of cortical thymocytes, and elevated levels of TdT have been documented in neoplasms derived from immature lymphoid cells. High enzyme activity has been reported in tumour cells of lymphoblastic leukaemias, lymphoid blast crisis of chronic myeloid leukaemias, and the majority of acute lymphoblastic leukaemias (ALL).**

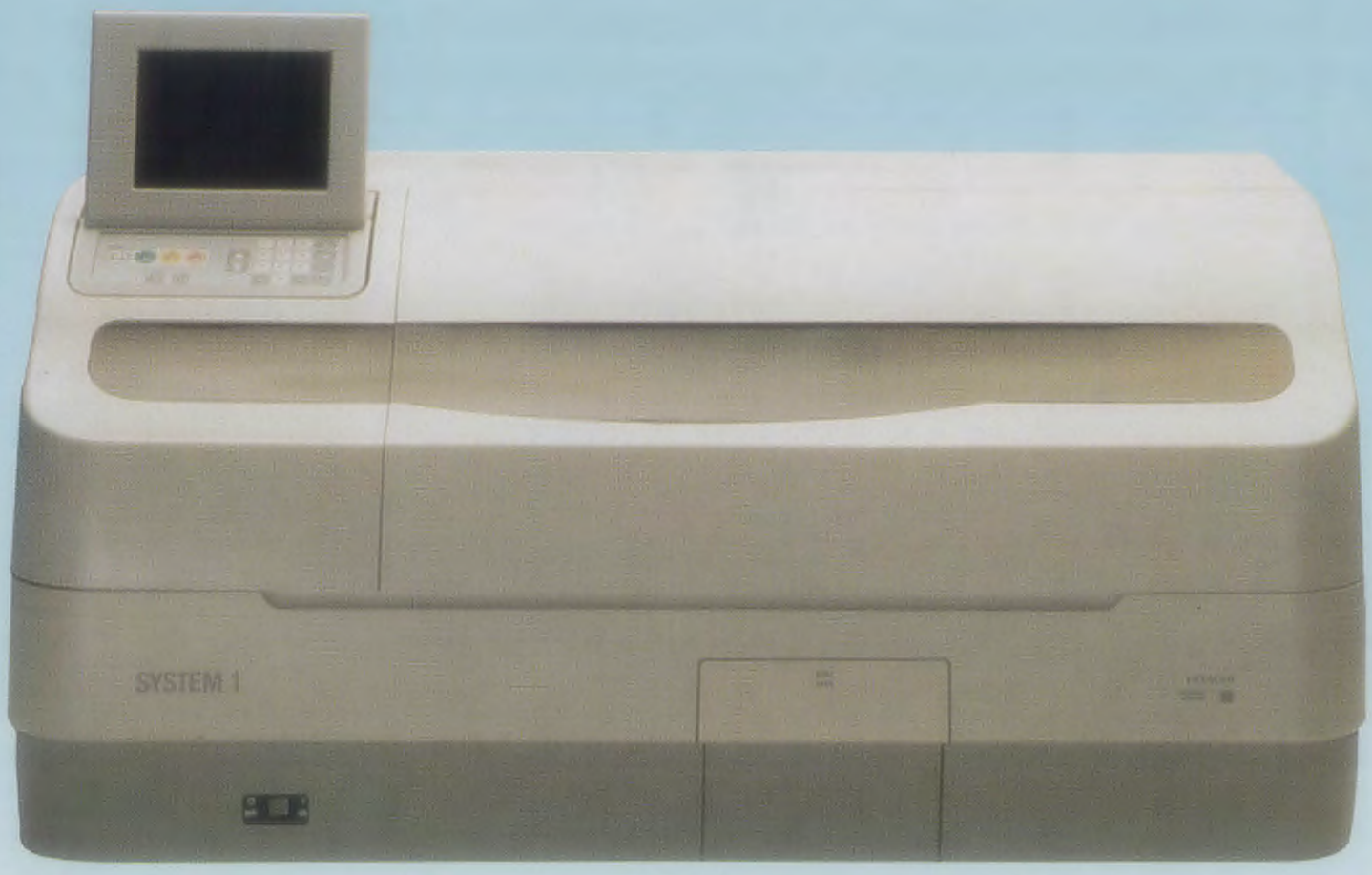
DAKO TdT. Product code A3524.

For additional information call Med-Bio Enterprises Ltd. 0800 733 599.

Laboratory Systems from Boehringer Mannheim

Elecsys

Immunology for tomorrow



mannheim boehringer