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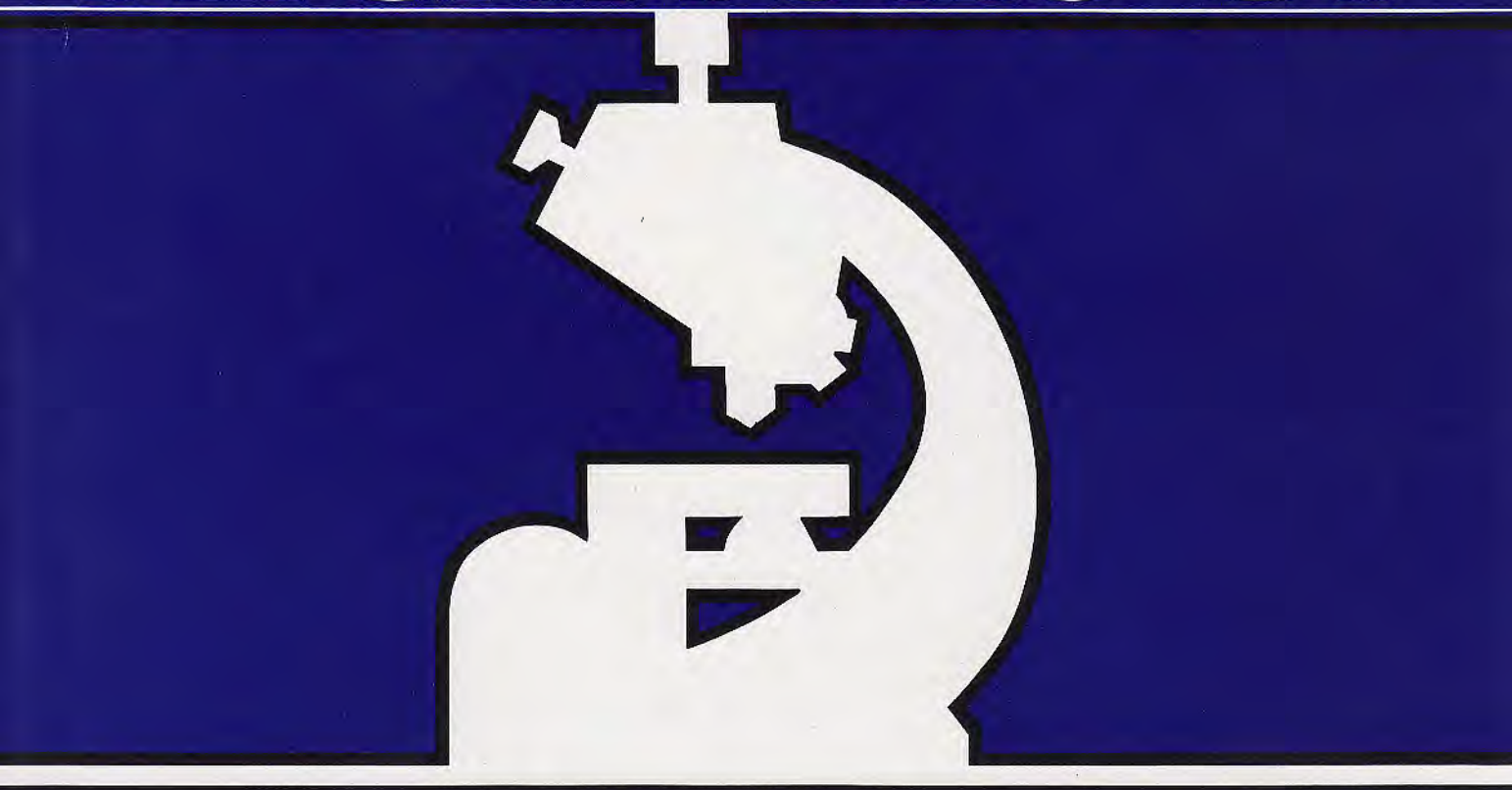

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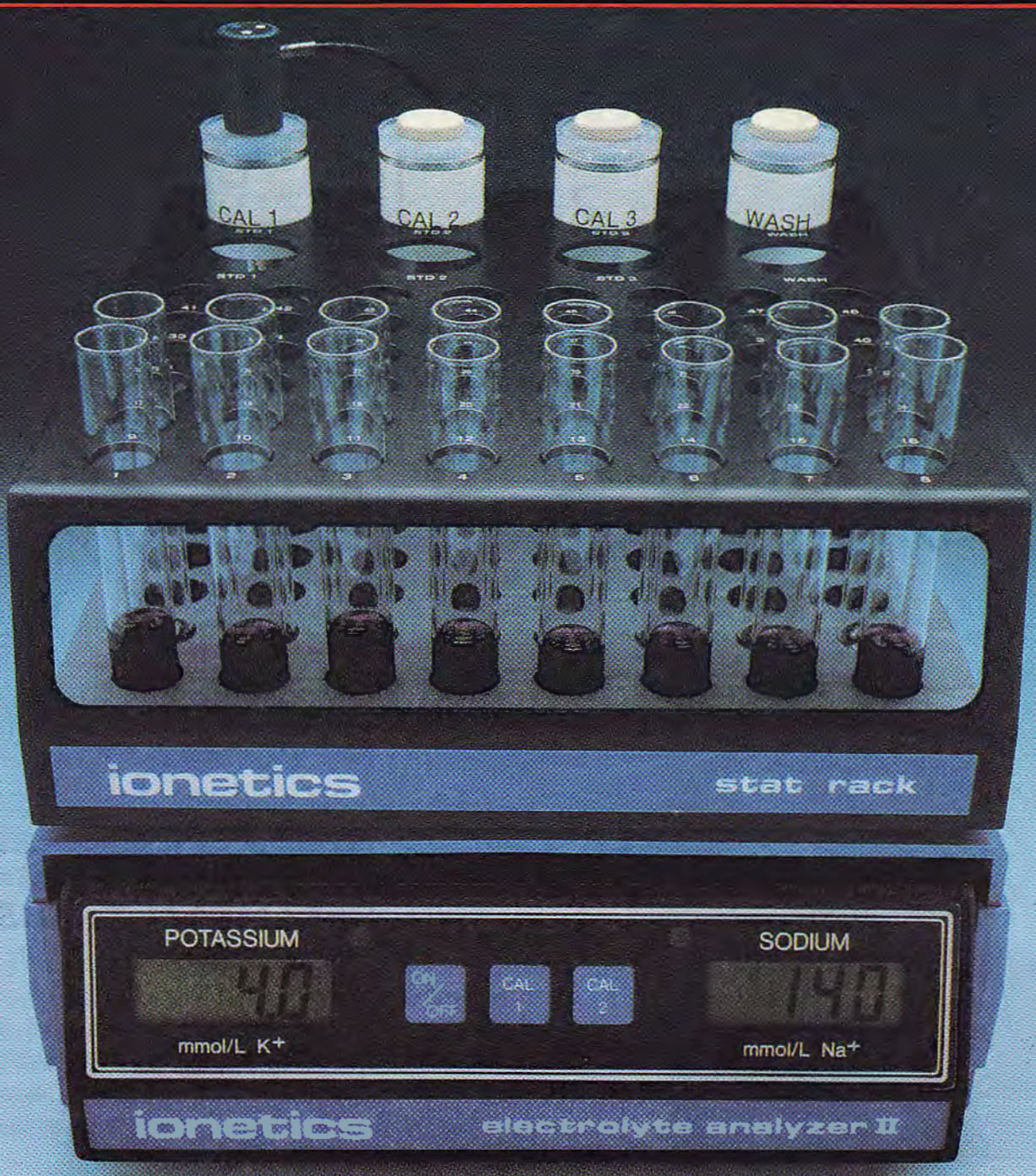

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TABLE OF CONTENTS

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Investigation of Lewis Phenotypes in Polynesians. : Variability in Detection of Lewis Antigens by Monoclonal, Goat and Human Anti-sera.

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Abstract

Commercially available monoclonal, goat and human Lewis antisera (Le^a, Le^b) were evaluated for red cell phenotyping of Polynesians. These antisera did not always give concordant phenotypes when tested in parrallel. No single anti-Le^b reagent could detect all the Le^b antigens defined by either of the three antisera used, suggesting that no single antisera was capable of accurate Lewis phenotyping of Polynesians. Monoclonal anti-Le^b failed to react with many of the Le(a+b+) samples although it did consistently detect the Le^b antigen in Le (a-b+) samples indicating a clear specificity for this antigen. Score results indicated that the non detection of Le^b in Le(a+b+) samples could not entirely be attributed to a lower titre of the monoclonal anti-Le^b. These results suggest there may be an Le^b epitope common to all Polynesian Le(a-b+) individuals but not common to all Le(a+b+) individuals.

It is proposed that some Polynesians have an Le^b antigen with a structural difference in the recognition area, a difference that is reactive (at least partially) with polyclonal sera but not recognised by monoclonal sera.

Key words

Lewis antigen, Lewis antisera.

Introduction

Most antibodies in man and animals are heterogeneous mixtures of molecules with similar but not identical specificities. These polyclonal antibodies probably react with many different epitopes on the same antigen. In contrast monoclonal antibodies by the nature of their preparation will recognise only a single epitope of the multiple epitopes carried on some blood group antigens. With the introduction of monoclonal antibodies into routine immunohaematology several different monoclonal antibodies have revealed variance in their ability to detect antigens defined by polyclonal sera. These include a monoclonal anti-M that undiluted, reacts with M negative red cells [1], the detection rate of weak ABO variants [2] and recognition of different epitopes of the Gerbich antigen [3].

Lewis antigens are well defined oligosaccharide structures built on common precursor chains and involving the interaction of the *Le* and *Se* genes [4]. Detection of these antigens is generally made using saline reactive human or goat reagents, and more recently monoclonal reagents.

In recent years, the Lewis system has become increasingly important in the field of renal transplantation [5-7] and colonic carcinoma [8]. As the incidence of renal disease is high in Polynesians [9,10], and because the frequency of Lewis phenotypes in Polynesians appear to differ from those of Europeans [11] a survey was undertaken to establish the reliability of monoclonal, goat and human sera in determining Polynesian Lewis phenotypes.

Materials and Methods

Samples

Blood samples were obtained over a period of 3 months from Polynesian (Pacific Islanders and Maoris) blood donors resident in the North Island, New Zealand. All Polynesians were first identified by sight and only those predominantly of polynesian ancestry were accepted for this study. The ethnic distribution for the 108 samples was 57 Maoris, 35 Samoans, 7 Cook Islanders, 5 Tongans and 4 Niueans. Samples for controls were collected from 85 predominantly non group O European blood donors. The ABO distribution for these control samples was 13 group O, 46 group A, 21 group B and 5 group AB.

Approximately seven millilitres of venous blood was collected into 1mL of citrate phosphate dextrose anticoagulant (Becton Dickson, Rutherford, NJ). Blood samples were stored at 4°C and phenotyped within 3 days of collection.

Saliva was obtained from 102 of these Polynesians, processed and tested as described elsewhere [12].

Antisera

Mouse monoclonal anti-Le^a (111037), anti-Le^b (113057) and human anti-Le^a (111057) and anti-Le^b (111047) were supplied by Biotest (Frankfurt, WG). Anti-Le^a (LA346A1) and anti-Le^b (LB544A2) of goat origin were supplied by Ortho Diagnostic Systems (Raritan, NJ).

Human ABO antisera were supplied by Biological Laboratories Ltd., Auckland, N.Z.

All testing was carried out by the manufacturers recommended methods and appropriate controls were used at all stages. Agglutination reactions were read with a 10 X magnification eyepiece and scored by the numerical system of Marsh [13] (12 = ++++, 10 = +++, 8 = ++, 5 = +, 3 ±, 2 = w, 0 = no agglutination).

Anti-Le^b inhibition Study

The ability of Ortho goat anti-Le^b (LB545A3) to detect the Le(a+b+) phenotype after inhibition of anti-$Le^{b\,H}$ activity with group O Le(a-b-) secretor saliva [14] was tested. Five hundred microlitres of saliva from a Caucasian group O Le (a-b-) secretor was added to a 2mL aliquot of anti-Le^b (Test). A dilution control using 500 μL of saline (group O Le(a-b-) non-secretor saliva was not available) added to a second 2 mL aliquot of anti-Le^b (Control) was tested in parallel. The treated anti-Le^b reagents were incubated at RT for 1 hour before they were used.

Table I: *Distribution of concordant and discordant Lewis phenotypes using human, goat and monoclonal sera.*

Race	No	Concordant				Discordant
		a+b-	a-b+	a-b-	a+b+	????[1]
Maori	57	12	35	4	1	5
Samoan	35	9	15	3	2	6
Cook Is	7	2	4	0	0	1
Tongan	5	1	2	0	1	1
Niuean	4	0	0	0	0	4
Controls (Caucasian)	85	22	52	5	0	6

[1] ???? — Abbreviation used when phenotypes obtained using the three different antisera were not in agreement.

Table II. *Polynesian and control phenotype discrepancies.*

Number/Group				Lewis phenotypes derived from results		
A	B	O	n	Mono	Goat	Human
Polynesians						
0	1	4	5	a+b-	a+b+	a+b+
1	1	2	4	a+b-	a+b+	a+b-
0	0	1	1	a+b+	a+b+	a-b+
1	0	0	1	a-b+	a-b+	a-b-
0	0	4	4	a+b+	a-b+	a-b+
1	0	0	1	a+b+	a+b-	a+b-
0	0	1	1	a+b-	a+b-	a+b+
3	2	12	17			
Controls						
0	5	0	5	a+b+	a-b+	a-b+
0	1	0	1	a+b+	a-b+	a+b+
0	6	0	6			

n = number tested; Mono = monoclonal antisera;
Goat = goat antisera; Human = human antisera.

Thirteen of 14 goat sera defined Le(a+b+) and 7 random Le(a-b+) Polynesian samples from this survey as well as 5 Caucasian Le(a-b+) control samples were recovered from liquid nitrogen. All samples were deglycerolised, washed 6 times and prepared as 5% cell suspensions in saline. Twenty microlitres of each suspension was added to two glass tubes, then 50 μL of the appropriate antiserum added. After 30 minutes incubation at RT, tubes were centrifuged and the agglutinations graded.

Results

Samples from 108 random Polynesians and 85 Caucasian controls were tested with three Lewis antisera of different origin.

The distributions of the Lewis phenotypes using each of these sera is summarised in Table I along with the discordant results, recorded as the Le(????) phenotype. Discordant results are those where one or more of the three antisera gave a different phenotypic interpretation.

The results of the 17 Polynesian and 6 control samples that gave serological discrepancies are summarised in Table II. Goat antisera detected 5 Le^b reactions in concordance with human sera that were undetected with monoclonal sera and

Table III. *Number of weak reactions in Le(a+b-) and Le(a-b+).*

		Number of weak reactions*						
		Polynesian			Control			
	n	Mono	Goat	Human	n	Mono	Goat	Human
Anti-Le^a	24	1	11	11	22	0	5	12
	%	4	46	46		0	23	55
Anti-Le^b	56	7	0	34	52	5	1	19
	%	13	0	61		10	2	37

* weak reactions = score 2-8,
n = number tested; Mono = monoclonal antisera;
Goat = goat antisera; Human = human antisera.

Tabie IV. *Inhibitory effect of Le(a-b-) secretor saliva on goat anti-Le^b*

		Average Anti-Le^b Score		Number of negative results	
Lewis Phenotype	n	Test Saliva	Control Saline	Test Saliva	Control Saline
Polynesian					
Le(a-b+)	7	6.3	8.0	1	0
Le(a+b+)	13	1.7	4.6	10	2
Caucasian					
Le(a-b+)	5	10.0	10.0	0	0

5 Le^b reactions undetected by either human or monoclonal sera. Goat antisera in concordance with monoclonal sera detected 1 Le^a and 1 Le^b reaction not detected by human sera. Monoclonal antisera detected 4 Le^a and 1 Le^b reaction not detected by either human or goat sera. Human sera detected 1 Le^b reaction which was undetected by both the goat and monoclonal sera.

Monoclonal anti-Le^a detected weak reactions (scores of 3) in 6 of the 17 group B Le(a-b+) controls whereas this reactivity was detected in only one of these samples by human sera, and not detected with the goat sera.

Monoclonal anti-Le^b reacted with only 36% (5 of 14) samples defined as Le(a+b+) but with 100% of the samples defined as Le(a-b+) by the goat antisera.

To allow comparison of reactivity between sera, the results from the Le(a+b-) and Le(a-b+) phenotypes were divided into the number of strong and weak reactions obtained (Table III). Weak reactions were defined as those with a score within the score range of 2-8. Monoclonal anti-Le^a detected only 1 weak reaction in 46 samples compared with 16 weak reactions with goat sera and 23 with human sera. Goat anti-Le^b detected only 1 weak reaction in 108 samples compared with 12 weak reactions with monoclonal sera and 53 with human sera.

Average Le^b reaction scores for the Le(a-b+) samples showed that human anti-Le^b reacted marginally weaker against Polynesian Le^b antigen than that of the controls (average scores of 8.3 and 9.4 respectively). The more reactive monoclonal and goat sera showed no variation, with similar respective scores of 10.9 and 11.4 for both the Polynesians and controls.

Polyclonal goat anti-Le^b was mixed with non-secretor saliva to inhibit anti-Le^b activity [14]. If this activity was present then a reduction in potency of the sera under test would be expected. A dilution control using saline was tested in parallel (group O Le(a-b-) non-secretor saliva was not available). Eight Le(a+b+) and 1 Le(a-b+) sample failed to react with the saliva treated anti-Le^b but were still reactive with the control anti-Le^b (Table IV). Two Le(a+b+) samples failed to react with the control anti-Le^b, demonstrating that as little as a 20% dilution of anti-Le^b, can make the antisera non-reactive.

Discussion

The monoclonal, goat and human Lewis antisera used for phenotyping Polynesians in this study were not in concordance for the determination of Lewis phenotypes. No single antisera detected all the expected Le^b antigens, indicating that no single antisera was reliable for the determination of Polynesian Lewis phenotypes.

Anti-Le^a

The detection of 4 Le^a reactions in the Polynesian Le(a-b+) samples by the monoclonal sera (Table II) was not unexpected because of the strong reactivity of this sera. As Le(a-b+) samples are known to have Le^a antigen [15] this

detection of Le^a is probably related to the strength of the monoclonal sera. This increased sensitivity of monoclonal anti-Le^a for Le^a antigen has been previously reported [16,17].

It is possible the weak Le^a reaction of the group B Le(a-b+) control samples is due to the presence of more Le^a antigen or the steric presentation being more favourable to the monoclonal anti-Le^a sera. The reaction is unlikely to be due to anti-B contamination as the majority of group B samples tested were negative.

Monoclonal anti-Le^a could be considered to be too strong because it detected weak Le^a reactions on Le(a-b+) samples whereas human sera could be considered too weak, as it failed to detect an Le^a reaction in a Le(a+b-) sample.

Anti-Le^b

Monoclonal anti-Le^b failed to detect the Le^b antigen on many of the polyclonal sera defined Le(a+b+) samples. The possibility that this failure was due to reagent potency was examined. However the human sera which was determined to be the least reactive of the three antisera (Table III) detected 5 Le^b reactions in concordance with goat sera, that were not detected by the monoclonal sera (Table II). In addition monoclonal anti-Le^b reagent detected one reaction which was undetected by both human and goat anti-Le^b.

Using monoclonal antisera the Le(a+b+) phenotype, as defined by goat or human sera is less commonly detected. Unlike previous reports [17,18] that indicate monoclonal anti-Le^b is superior to polyclonal sera, the monoclonal anti-Le^b used in this study was not as sensitive as the polyclonal sera in detecting the Le^b antigen in Polynesians.

Association of the Le(a+b+) phenotype with ABO groups and H antigen reactivity [11,19] appear (at least in part) to be due to reactivity with anti-$Le^{b\ H}$, or a similar H reactive antibody, present in polyclonal anti-Le^b (Table IV). Reactivity with this component of polyclonal sera [20] would cause a greater detection rate of a weak Le^b antigen expression in H reactive samples. The combined effect of the anti-$Le^{b\ L}$ and anti-$Le^{b\ H}$ components of polyclonal sera may allow the threshold of agglutination to be achieved with some samples, an effect not possible with monoclonal sera. Some Polynesian red cell samples which typed as Le(a+b-) probably have Le^b antigen present but at levels too low to support agglutination. This is supported by the finding that a 20% reduction in the potency of the goat anti-Le^b sera resulted in some Le^b antigens becoming undetectable.

Comment

The secretor gene which is responsible for the formation of Le^b antigen is not a regulator gene but rather a structural gene closely linked to the H gene [21]. The *Se* gene codes for an alpha-2-L-fucosyl-transferase present in epithelial tissue and able to transform both type-1 and type-2 precursor chains. The Le(a+b+) phenotype may be the result of a change in the normally found equilibrium of the Secretor and Lewis gene coded transferases. Whichever gene is involved the net effect would be a decreased production of type-1-H chains from available precursor chains. Once the type-1 chain has been converted into Le^a substance by the action of the *Le* gene coded transferase, the *Se* gene coded transferase cannot convert the chain into type-1-H hence lower levels of Le^b antigen would result.

Theoretically the Le(a+b+) phenotype could be caused by a more efficient than usual *Le* gene competing more successfully than the *Se* gene for type-1-precursor chains; alternatively a less efficient secretor gene could give the same result. The finding of weak secretor phenotypes in Le(a-b-) individuals in a Le(a+b+) Japanese family study [22,23], and by ourselves in Polynesians [12], suggests the presence of a weak secretor gene (Se^w).

It would be expected that a Se^w gene would cause partial secretion and the red cell Le(a+b+) or Le(a+b-) phenotype. Further evidence to support this hypothesis is provided by our recent findings of salivary substances in almost all Polynesians regardless of their red cell phenotype [12,24].

The amount of genetically independent precursor chains produced and the ability of the red cells to uptake glycosphingolipids, variables not able to be measured in this study, could also be involved in the phenotypic expression of Lewis antigens. We suggest that the failure of anti-Le^b antigen on the red cells of some Polynesians may be due to low levels of Le^b antigen, and/or epitopical differences in the currently recognised Le^b antigen.

Acknowledgements

The authors would like to thank Christine Glavas, Linda Pinder, Walter Wilson and Bill Capper for their help.

References

1. Nichols M.E., Rubinstein P. Two types of monoclonal anti-M antibodies. *Transfusion* 1981; **21**: 631-632.
2. Gane P., Vellayoudom J., Mollicone R., et al: Heterogeneity of anti-A and anti-B monoclonal reagents. Agglutination of some weak ABH erythrocyte variants and recognition of synthetic oligosaccharide and tissue antigens. *Vox Sang* 1987; **53**: 117-125.
3. Rouger P.H., Lee H., Juszczak G., et al: Murine monoclonal antibodies against Gerbich antigens. *J Immunogenet* 1983; **10**: 333-335.
4. Oriol R., Le Pendu J., Mollicone R. Genetics of ABO, H, Lewis, X and related antigens. *Vox Sang* 1986; **51**: 161-171.
5. Oriol R., Gerhard O., Chun C., et al: The Lewis system and kidney transplantation. *Transplantation* 1980; **29**: 397-400.
6. Pfaff W.W., Howard R.J., Ireland J., et al: The effect of Lewis antigen and race on kidney graft survival. *Transplantation Proceedings* 1983; **15**: 1139-1141.
7. Spitalnik S., Pfaff W., Cowles J., et al: Correlation of humoral immunity to Lewis blood group antigens with renal transplant rejection. *Transplantation* 1984; **337**: 265-268.
8. Koprowski H., Brockhaus M., Blaszczyk M., et al: Lewis blood-type may affect the incidence of gastrointestinal cancer. *Lancet* 1982; **1**: 1332-1333.
9. Dawson K.P. A comparative study of the clinical patterns of acute glomerulonephritis from a high and low incidence area of New Zealand. *NZ Med J* 1982; **95**: 262-264.
10. Smith A.H., Pearce N.E. Determinants of differences in mortality between New Zealand Maoris and non-Maoris aged 15-64. *NZ Med J* 1983; **97**: 101-108.
11. Henry S.M., Simpson L.A., Woodfield D.G. The Le(a+b+) phenotype in Polynesians. *Hum Hered* 1988; **38**: 111-116.
12. Henry S.M., Benny A.G., Woodfield D.G. Investigation of the Lewis phenotypes in Polynesians. Evidence of a weak secretor phenotype. Submitted *Vox Sang* 1989.
13. Marsh W.L. Scoring of hemagglutination reactions. *Transfusion* 1972; **12**: 352-353.
14. Issitt P.D. Applied blood group serology. 3rd ed. Miami: Montgomery Scientific, 1985.
15. Cutbush M., Biglett E.R., Mollison P.L. Demonstration of the phenotype Le(a+b+) in infants and adults. *Brit J Haemat* 1956; **2**: 210-220.
16. Cowles J.W., Cox M.T., McMican A., et al: Comparison of monoclonal antisera with conventional antisera for Lewis blood group antigen determination. *Vox Sang* 1987; **52**: 83-84.
17. Longworth C., Rolih S., Moheng M., et al: Mouse monoclonal anti-Le^a and anti-Le^b as routine grouping reagents. *Transfusion* 1985; **25**: 446.
18. Messeter L., Brodin T., Chester M.A., et al: Immunochemical characterization of a monoclonal anti-Le^b blood grouping reagent. *Vox Sang* 1984; **46**: 66-74.

19. Vox G.H., 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* 1967; **17**: 495-510.
20. Francois A., Sansonetti N., Mollicone R., et al. Heterogeneity of Lewis antibodies. A comparison of the reaction of human and animal reagents with synthetic oligosaccharides. *Vox Sang* 1986; **50**: 227-234.
21. Oriol R., Danilovs J., Hawkins B.R. A new genetic model proposing that the *Se* gene is a structural gene closely linked to the *H* gene. *Am J Hum Genet* 1981; **33:** 421-431.
22. Lewis M., Kaita H., Chown B. The blood groups of a Japanese population. *Am J Hum Genet* 1957; **9**: 274-283.
23. Race R.R., Sanger R. Blood groups in man. 6th ed. Oxford, London: Blackwell Scientific Publications, 1975.
24. Henry S.M. The serology and genetics of the Le(a+b+) Phenotype in Polynesians. NZIMLT Fellowship thesis, Submitted Jan 1989.

Using the Correlation Coefficient in the Determination of Reference Intervals

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Presented at the 1988 NZACB — NZIMLT Conjoint Conference, Rotorua

Abstract

Reference intervals are critically important for the interpretation of laboratory results. Conventionally, a reference interval for a given test is defined as the central 95% of values observed within a healthy population. There is no reason to believe that this rigid adherence to the central 95% as a means to distinguish 'normal' from 'abnormal' values is optimal for all tests. Because of this and because the health of the reference population is usually unverifiable (i.e. any given reference population will contain an unknown proportion of sick but asymptomatic subjects), Merkouriou and Dix (*Stat Med* 1988, 7: 377-385) have argued that health should be abandoned as a criterion of reference data. Instead they suggest distinguishing "typical" (normal) from "atypical" (abnormal) values by purely statistical criteria. Typical values are thus defined as those exhibiting a linear relationship with percentiles on a value versus percentile plot; linearity being assessed by the Pearson product-moment correlation coefficient, *r*. The general applicability of the method is illustrated with data from both 'healthy' and 'unhealthy' populations.

Introduction

Although reference intervals are essential for the interpretation of patients' results, reference information is said to be the weakest data provided by clinical laboratories (1). If you think about this for a moment you will realise this to be so. We go to great lengths to produce accurate and precise test results — very often exceeding clinical requirements — only to have them compared with reference data that is at best uncertain and at worst inappropriate. Many of our reference intervals are compromises, the effects of age, sex, time and/or other variables being largely ignored.

Readers will be familiar with the conventional strategy for determining reference intervals as promulgated by the International Federation of Clinical Chemistry (IFCC)(2) and outlined in Table 1. Difficulties can arise, however, in several of these steps. Let us consider some of them. Firstly, obtaining a population of healthy individuals in numbers sufficient for statistical analysis poses both logistical and ethical problems. Very often laboratories rely on blood donors and/or hospital staff for their reference individuals; two groups that are not necessarily representative of a population for which reference data is being sought. Even if a reference population can be assembled the health of the individuals remains undefined. Such a sample will inevitably contain an unknown number of sick but asymptomatic individuals with common disorders such as cancer, ischemic heart disease and diabetes, and this in turn must lead to uncertainty as to the accuracy of the reference intervals obtained.

Secondly, we must ask the question: is a healthy population appropriate anyway? Consider the often quoted example of patients with suspected myocardial infarction admitted to a coronary-care unit. Their enzyme results will probably be compared with a reference interval derived from a healthy ambulant population when in fact it seems more logical that they should be compared with a reference interval derived from age-matched patients with similar symptoms but who have not experienced myocardial infarction (3).

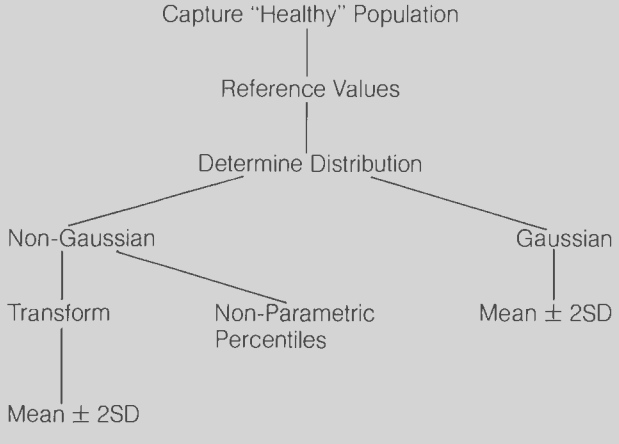
Thirdly, the width of the reference interval needs to be considered. Conventionally, we have come to accept a reference interval as encompassing the central 95% of observations within a healthy population and biochemists it seems have not considered alternatives but rather thay have become obsessed with determining this 95%. The many elegant mathematical procedures advocated to transform skewed distributions to the more manageable gaussian form attest to this. However, notwithstanding the above comments, there is no reason to believe that this rigid adherence to the central 95% as a means to distinguish normal from abnormal values is optimal for all tests. Indeed, recent reference creatine kinase data for men gave a 97.5th percentile upper reference limit of 300 U/L which was considered too high if used in the diagnosis of myocardial infarction, (4) and the unsuitability of the 97.5th percentile of plasma cholesterol concentration as an upper reference limit is well known (3, 5). What in fact does this 95% span tell us? Well, all that it does say is that on average 5% (1 in 20) of apparently healthy subjects will have a value outside this range; it says *nothing* about the probability of an unhealthy subject having a value outside, or for that matter inside, the reference interval.

Because of considerations such as these Merkouriou and Dix(6) have argued that health should be abandoned as a criterion for reference intervals. Instead they suggest that objective statistical criteria should be used to separate "typical" (normal) from "atypical" (abnormal) values. How then do we decide what are typical and atypical values?

Method

Looking at a skewed frequency distribution (characteristic of many biochemical tests) of creatine kinase values in young women (Fig 1, inset), it is not immediately obvious where the typical values lie, or clear as to the position of appropriate reference limits. Such values, though, become conspicuous on a cumulative percentage plot (Fig 1). Notice the conspicuous deviation from linearity in the tails. Values above about the 90th percentile and below about the 3rd percentile are clearly different, that is atypical, from the linear spread of values inbetween. Linearity on the value-percentile plot is thus a natural objective definition for typical behaviour. At this point, it must be stressed that the region of the 90th percentile has no general significance and Merkouriou and Dix are not proposing it as a substitute for the 97.5th percentile. Rather, the region of the 90th percentile is a boundary between typical values specific for the data in Figure 1. As we shall see later, the points of deviation from linearity are not fixed at any given percentile but depend on the shape and content of the frequency distribution. Our goal then is to locate accurately those points of deviation. The authors give the following method:

Table 1: *IFCC Strategy for Reference Interval Determination.*



TECHNICAL ASSISTANTS EXAMINATION COMMITTEE

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The 1988 examinations were conducted on 10 and 11 May. There were 98 candidates for the examination with 92 gaining the Certificate of Qualified Assistant. The pass rate was 94% compared with 90% the previous year.

Breakdown of figures are:

	1988		1987	
	Sat	Passed	Sat	Passed
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General Certificate	8	7	10	8
Haematology	20	17	13	9
Histological Technique	9	9	10	9
Medical Cytology	4	3	5	5
Medical Microbiology	19	19	26	25
Mortuary Hygiene & Technique	2	2	1	1
Radioisotope & Radioassay Technique	1	1	1	1
Immunohaematology	13	13	10	9
Immunology (Microbiology)	4	4	8	8
Special Certificates	5	4	2	2
	98	92	102	92

OVERSEAS AID COMMITTEE

Members of the Committee are E. Norman (Convener), M. Eales and S. Gainsford.

The Institute continued to support the P.P.T.C. during the 1988-89 year.

This was mainly in the form of the donation of an appropriate text book to each course graduate and of financial support for the Quality Assurance Programme for Pacific Island Laboratories.

Once again thanks are due to the many individual Institute members who support and assist the P.P.T.C. and especially to Marilyn Eales for her continuing contribution of the Pacific Way section of the Journal.

SAFETY COMMITTEE

Members of the Committee are J. Parker (Convener) and B. Cornere.

With so much occurring in the industrial arena there has been little time to consider safety matters. With the Union forming it is hoped that more attention can be paid to this very important area in the coming year.

AWARDS COMMITTEE

J. Parker (Convener).

Our thanks once again to all those firms who have so generously supported us in the past year. They were as follows:

MLTB Examination Award Donors — Certificate Level

Roche Products (NZ) Ltd
Intermed Scientific Ltd
Amersham Australia Pty Ltd
Kempthorne Medical Supplies Ltd
Hoechst NZ Ltd
Gibco NZ Ltd
Biotek Supplies
Sci Med (NZ) Ltd

MLTB Examination Award Donors — Specialist Level

Watson Victor Ltd
Sci Med (NZ) Ltd
Wilton Instruments
Biotek Supplies
Amersham Australia Pty Ltd
Organon Technica/ General Diagnostics
Medic DDS Ltd
Gibco NZ Ltd

QTA Examination Award Donor
NZ Blood Foundation

Journal Awards
Roche Products (NZ) Ltd
Travenol Laboratories (NZ) Ltd

Travel Award
Wellcome NZ Ltd.

Our thanks also to the membership whose contributions make possible a number of NZIMLT Examination and Journal Awards and two Scholarships.

EDUCATION COMMITTEE

Members of the Committee are J. Parker (Convener), B.T. Edwards and W. Wilson.

This year has been a time of great uncertainty with regards the present and future education of Medical Laboratory Technologists. The decision of the last annual general meeting that the NZIMLT should take responsibility for the Part III examinations was superseded by the decision of the MLTB to request that changing of the regulations be deferred to take effect in the 1990 year. Polytechnics have effectively replaced the NZCS paramedical with a broader based NZCS in module format. At the same time the Auckland Technical Institute has introduced a Diploma course to replace both NZCS and the post NZCS component of the current course. The Hawke Report and the proposals for graduate tax are having effects on both Universities and Polytechnics in terms of funding and provision of new and existing courses. The Under-Secretary of Health undertook to express to the Otago University and to the Education Department ministerial support for our degree proposals, in the Cabinet reshuffle both the Minister and Mr Dunne have moved on and the support of the new Minister is less certain. The Otago University has now been named as the sole school for training Pharmacists in New Zealand from 1990 and this can only strengthen our own case. At present our proposals are 'on the table' with the Otago University who fully intend to pursue the initiative now that the fate of the School of Pharmacy is decided.

PUBLICATIONS COMMITTEE

Members of the Committee are D. Dixon-McIver (Convener), D. Reilly, W. Wilson and P. Reilly (Advertising Manager).

There were 17 papers proffered for publication (3 Auckland, 3 Dunedin, 3 Wellington, 3 Christchurch, 1 Palmerston North, 1 Blenheim, 1 Rotorua and 1 U.S.A.) of which 15 have been accepted for publication and published. This compares with 14 in 1987, 13 in 1986 and 28 in 1985.

The slight upturn in material being proffered is pleasing but it is important that it is maintained.

The editor wishes to record his thanks to Trish Reilly, Maurice Sheppard and the Royal NZ Foundation for the Blind for their continued assistance and support.

MEMBERSHIP COMMITTEE

Members of the Committee are G. Rimmer (Convener), D. Dixon-McIver and D. Reilly.

Total membership of the Institute has risen and has almost reached the peak of 1985/86. Most of the new members are in the 'Member' category possibly the result of the new legislation which meant that the Institute was deemed to be the Union for this year.

Approximately 2/3 of the membership are taking advantage of the automatic salary deduction to pay their annual subscription. As with any new system there has been a few problems, most of which have now been resolved.

This years 43 Complimentary Members were the last as there were no new additions to this category after March 1988.

With the formation of the N.Z. Medical Laboratory Workers' Union, resulting in probably the biggest change in the Institutes history, the retention of membership is an important challenge for the coming year. Please think carefully about the role of and direction of your Institute and remain a part of this new era. Your continued membership is essential to the future of Medical Laboratory Technology as a strong profession.

	1988/89	87/88	86/87	85/86	84/85
Membership from previous year	1465	1536	1792	1352	1369
Less deletions	87	340	454	58	190
	1378	1196	1338	1294	1179
Plus application	331	269	198	498	173
Membership as at 31st March	1709	1465	1536	1792	1352
Membership composition:					
Life Members	17	16	14	15	15
Fellows	29	30	39	42	40
Associates	781	752	785	732	610
Members	741	579	625	956	595
Complimentary Members	43	123	168	235	110
Non-practising Members	68	58	55	32	77
Honorary Members	30	30	18	15	15

FELLOWSHIP COMMITTEE

Members of the Committee are J. LeGrice (Convener) K. McLoughlin and H. Potter.

A discussion document on the proposed revision of the N.Z.I.M.L.T. Fellowship Regulations was presented a the 1988 Conference in Rotorua. The proposals were based on the assumption that the Specialist qualification would no longer be available. However, this assumption proved incorrect and as a result there were no changes to the Fellowship regulations. Some alterations to the regulations are required, not the least of them being an increase in application fee so that the Fellowship system pays its way.

This year the Committee had three applications for Fellowship which covered all three avenues of attainment: thesis, examination and exemption. One of these has been resolved. Mr Rob Seibers was granted Fellowship by exemption on the basis of a higher qualification and the merit of supporting published scientific material.

INDUSTRIAL RELATIONS COMMITTEE

Members of the Committee are P. McLeod (Convener), W. Wilson, D. Dixon-McIver and S. Gainsford.

The major efforts for the committee during the last year have centered on the establishment of the Medical Laboratory Workers Union and the negotiation of our award.

N.Z. Medical Laboratory Workers Union

At the Special General Meeting of the Institute at Rotorua in 1988, the members supported the concept of the establishment of a separate union to handle the industrial matters which the State Sector Act now demands must be available to all State workers. The Institute, it was felt, was not an appropriate body to deal with industrial matters and in future should concentrate on the professional aspects of the organisation.

In March 1989, the inaugural general meeting of the union was held. At this meeting, the draft rules, subscription rate and interim executive were approved. Until one thousand people have joined the union and an application for registration of the union is sought, the Institute will continue to represent its members in all industrial matters.

Settlement of Award

The first negotiation round got under way in November 1988, however it became obvious very quickly that it was not the "right time" to settle our award. At that time there had been only a few award settlements in the private sector and the State Services Commission was offering a counterclaim that was verging on the ridiculous.

On March 1 and 2, 1989 we again met with the Commission and proceeded to settle our award. What was achieved is now history and comparing the settlement with other health groups, we feel very pleased with the outcome.This settlement does not expire until July 31st, 1990. In the meantime, all efforts are to be concentrated on establishing the new union and settling an award for the private medical laboratory workers.

I would like to thank all the committee for their untiring efforts and support and to all the members of the Institute who have given us encouragement and support in what is at times an extremely exasperating task. However, I hasten to add, that occasionally this committee does achieve some victories which somehow seem to make it all worthwhile.

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY TECHNOLOGY INC. STATEMENT OF FINANCIAL POSITION AS AT 31 MARCH 1989

	1989 $	1988 $
ACCUMULATED FUNDS		
Balance 1 April 1988	10,538	35,422
Surplus (deficit) for the year	25,957	(24,884)
Balance at 31 March 1989	36,495	10,538
Clinical Laboratory Special Fund (Note 5)	—	641
TOTAL FUNDS AS AT 31 MARCH 1989	$36,495	$11,179
Represented by:		
CURRENT ASSETS		
Cash at bank	20,268	2,852
Stock on hand (Note 3)	856	920
Sundry debtors	16,142	24,947
Subscriptions outstanding	—	2,007
GST	3,408	4,593
TOTAL CURRENT ASSETS	40,674	35,319
LESS CURRENT LIABILITIES		
Sundry Creditors	20,994	36,741
Subscriptions in Advance	—	6,660
Examination Fees in Advance	3,841	2,272
TOTAL CURRENT LIABILITIES	24,835	45,673
NET CURRENT ASSETS (LIABILITIES)	15,839	(10,354)
INVESTMENTS (Note 2)	20,000	20,000
FIXED ASSETS		
Typewriters (Note 4)	656	1,533
	$36,495	$11,179

Treasurer — D.M. Reilly

President — W. Wilson

The attached notes form part of this statement.

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY TECHNOLOGY INC. STATEMENT OF INCOME AND EXPENDITURE FOR THE YEAR ENDED 31 MARCH 1989

	1989 $	1988 $
INCOME FOR THE YEAR WAS DERIVED FROM:		
Conference surplus (as per statement)	8,960	4,288
Examination surplus	1,481	521
Interest received	3,359	6,119
Miscellaneous income	6,096	2,695
Subscriptions and levy	90,085	51,186
Refunds	2,400	—
Donations	1,200	—
TOTAL INCOME	113,581	64,809
FROM THIS INCOME THE FOLLOWING EXPENDITURE WAS MET:		
Accommodation, etc	10,414	9,715
Accountancy and audit fee	1,524	1,652
Computer services	9,176	8,404
Fees — D.S.U., LAMLT and NCCLS	2,455	3,453
Honoraria, gratuities and prizes	2,050	2,200
Journal cost (as per statement)	10,277	3,560
Legal expenses	14,336	2,250
Post Graduate Education and Pacific Training	1,058	2,551
Postage and tolls	4,877	5,759
Printing, stationery and typing	2,203	8,811
Sundry expenses	1,049	4,349
Travelling expenses	19,328	22,513
	78,747	75,217
Consultancy Fees	—	10,000
— Medical Laboratory Trust	—	3,599
Depreciation of typewriters	877	877
TOTAL EXPENDITURE FOR YEAR	79,624	89,693
Excess of Income Over Expenditure	33,957	24,884
Donation to New Zealand Medical Laboratory Science Trust (Note 5)	8,000	—
Surplus (deficit) for the year	$25,957	$(24,884)

The attached notes form part of this Statement.

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY TECHNOLOGY INC. CONFERENCE ACCOUNT FOR THE YEAR ENDED 31 MARCH 1989

	1989	1988
INCOME FOR THE YEAR WAS DERIVED FROM:	$	$
Registration	16,245	8,957
Trade rentals and advertising	13,471	17,158
Donations	5,953	3,325
Social functions and lunches	16,424	10,148
Bank interest and other income	481	35
Accommodation	—	3,845
Other income	1,234	—
	53,808	43,468
FROM THIS INCOME THE FOLLOWING EXPENDITURE WAS MET:		
Travel, accommodation and meals	32,967	29,284
Social function costs	3,261	3,833
Rentals	1,160	494
Postage, stationery and administration	3,876	3,889
Other expenditure	2,396	1,680
TOTAL EXPENDITURE	43,660	39,180
NZACB Share of Profit	1,188	—
Which leaves an excess of income over expenditure transferred to the Statement of Income and Expenditure	$8,960	$4,288

The attached notes form part of this Statement.

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY TECHNOLOGY INC. JOURNAL ACCOUNT FOR THE YEAR ENDED 31 MARCH 1989

	1989	1988
INCOME FOR THE YEAR WAS DERIVED FROM:	$	$
Advertising revenue	38,429	40,381
Subscriptions	1,041	1,426
TOTAL INCOME	39,470	41,807
FROM THIS INCOME THE FOLLOWING EXPENDITURE WAS MET:		
Printing — journal and newsletter	39,370	40,515
Postage and stationery	4,619	3,722
Sundry expenses	5,758	1,130
TOTAL EXPENDITURE	49,747	45,367
Which leaves an excess of expenditure over income transferred to the Statement of Income and Expenditure	$10,277	$3,560

The attached notes form part of this Statement.

NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY TECHNOLOGY INC. NOTES TO THE 1989 FINANCIAL STATEMENTS

1. **STATEMENT OF ACCOUNTING POLICIES**

The historical cost basis of accounting has been used in the preparation of the financial statements. Reliance is placed on the fact that the Institute is a going concern. Accrual accounting is used to match expenses and revenues.

Particular accounting policies:

(a) Fixed assets and depreciation.
Depreciation is calculated on a straight line basis to write off the typewriters over their estimated useful lives of 5 years.

(b) Stock is valued at actual cost.

There have been no changes in accounting policies. All policies have been applied on bases consistent with those used in previous years.

2. **INVESTMENTS**

Debenture stock

General Finance Ltd $20,000 @ 16.5% mature on 21/08/90.

3. **STOCK**

	1989 $	1988 $
Ties/badges, etc	$856	$920

4. **TYPEWRITERS**

	1989 $	1988 $
Cost	4,385	4,385
Accumulated depreciation	3,729	2,852
NET BOOK VALUE	$656	$1,533

5. **NEW ZEALAND MEDICAL LABORATORY SCIENCE TRUST**

The Council of the New Zealand Institute of Medical Laboratory Technology Inc. approved a donation of $8,000 together with the $641 held in the clinical Laboratory Special Fund to the NZ Medical Laboratory Science Trust.

TREASURER'S REPORT

June 1989

Dennis M. Reilly

The 1988/1989 financial year has ended with a surplus of $25,957. The Institute's income has increased mainly through subscriptions and a significant conference surplus. Council approved that $8,000 from this surplus be passed to the Trust.

A large proportion of members have elected to have their subscriptions deducted from their salaries. Initially there was a few hiccups but now it has settled into a smooth operation, although a significant amount of Treasurer's time is required in processing fortnightly computer print outs and banking cheques. Most Boards have been helpful while others deduct a percentage for their effort.

On the expenditure side the Journal and legal costs stand out as the major increases. The legal costs are virtually all due to the formation of the New Zealand Medical Laboratory Workers Union. This is now separated from the Institute and will not be a continuing expense.

The total expenditure of $79,624 reflects the wide range of activities that we have and if the Institute is to be more involved in examinations such as the 'Specialist' level we will need to maintain our income at a similar level. Consequently Council has suggested that subscriptions be maintained at their present level.

AUDITORS' REPORT TO THE MEMBERS OF THE NEW ZEALAND INSTITUTE OF MEDICAL LABORATORY TECHNOLOGY INC.

Deloitte Haskins+Sells

We have audited the financial statements on pages 1 to 5 in accordance with accepted auditing standards and have carried out such procedures as we considered necessary.

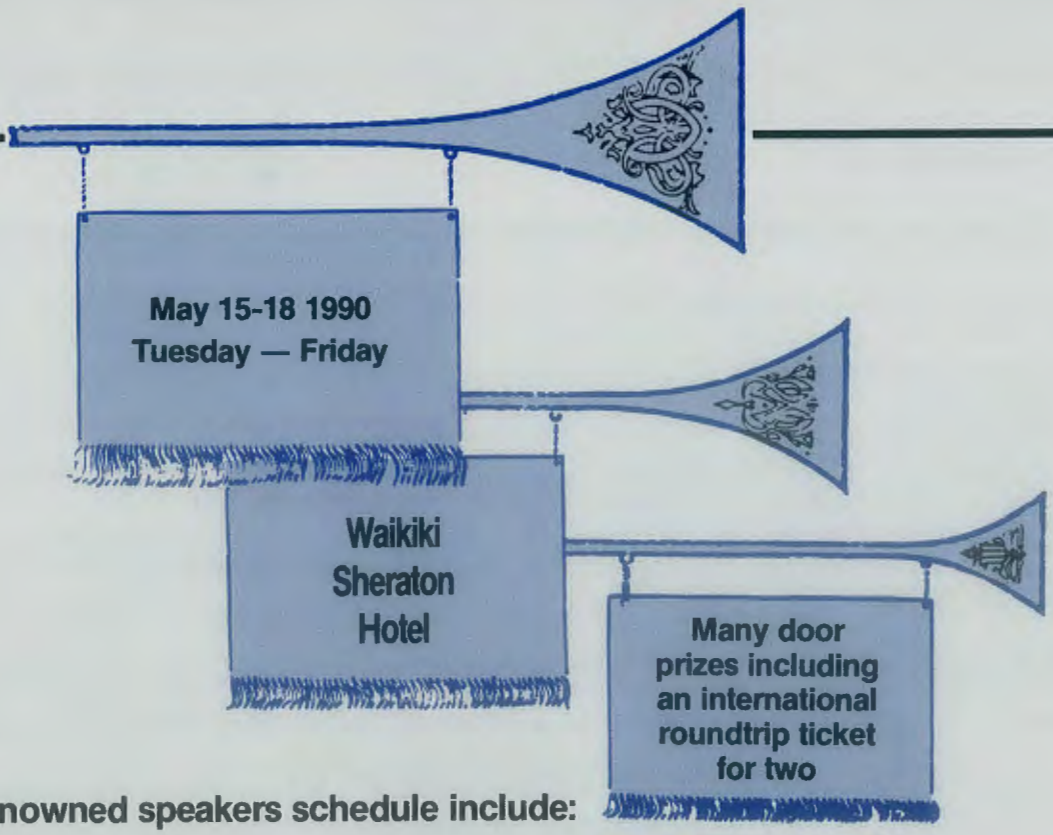
In common with other organisations of a similar nature, control over income prior to its being recorded is limited, and there are no practical audit procedures to determine the effect of this limited control.

Subject to the possible effect of the limited control over income referred to in the preceding paragraph, in our opinion the financial statements give, using the historical cost method, a true and fair view of the financial position of the Institute as at 31 March 1989 and the results of its activities for the year ended on that date.

Deloitte Haskins + Sells

11 May 1989
MANUKAU CITY, NZ

CHARTERED ACCOUNTANTS



Renowned speakers schedule include:

Dr Sydney Finegold	Anaerobic microbiology
Dr Ellen Jo Baron	Co-editor of BAILEY & SCOTT'S DIAGNOSTIC MICROBIOLOGY
Dr Lee Hilborne	Recombinant DNA technology
Dr Wayne Grody	Medical genetics/Molecular pathology
Dr Christopher Frings	Laboratory Consultant, Toxicologist
Dr Douglas Triplett	Haematology/Coagulation
Dr Judith Barr	Major contributor in ASMT government activities

Special Topics

Motivation Strategies
Improving Laboratory Utilisation
Government Update and Current Issues

AND MORE . . .

For more information, please complete the following and mail to:

Lucille Holzgang
c/o 98-418 Koanohi St. *3
Aiea, Hawaii 96701

NAME: _____

ADDRESS: _____

CITY, STATE, ZIP CODE: _____

Please send me information on: ☐ PROGRAMME ☐ TRAVEL PACKAGE

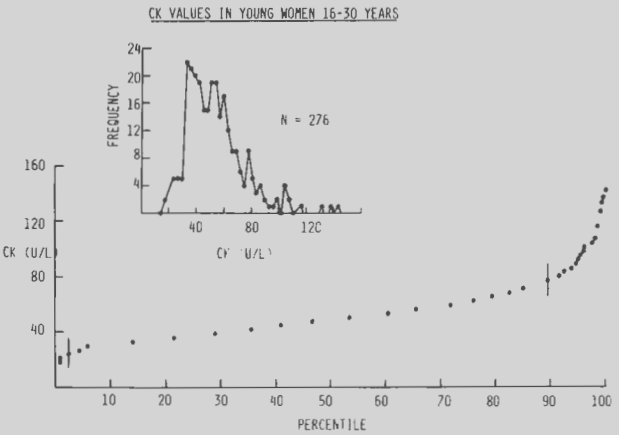


Figure 1: *Value-percentile plot (lower graph) of the distribution (upper graph) of serum creatine kinase (CK, 30°C) in young women. Vertical lines encompass the reference interval obtained.*

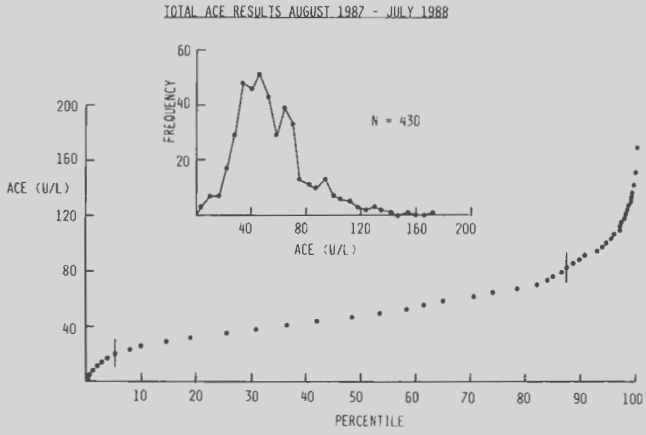


Figure 2: *Value-percentile plot (lower graph) of the distribution (upper graph) of serum angiotensin converting enzyme (ACE, 37°C) in patients for whom ACE was requested during a twelve-month period. Vertical lines encompass the reference interval obtained.*

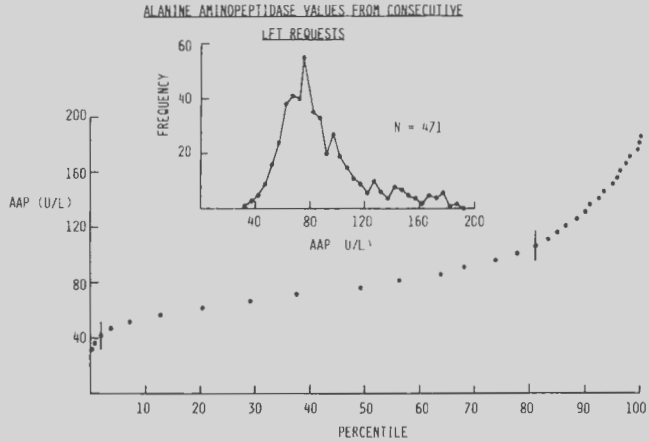


Figure 3: *Value-percentile plot (lower graph) of the distribution (upper graph) of plasma alanine aminopeptidase (AAP, 37°C) in consecutive patients for whom a liver function test profile was requested. Vertical lines encompass the reference interval obtained.*

Beginning near the median (by definition the most typical value), calculate the produce-moment correlation coefficient, *r*, between values and percentiles from some value sightly below the median to some value slightly above the median. Repeat the calculation by progressively and symmetrically extending the range of values above and below the median. As the range of values extends into the tails of the distribution the relationship between values and percentiles will deviate from linearity and the magnitude of *r* will decline. Merkouriou and Dix have chosen 0.9900 as the minimum value of *r* acceptable because it is the minimum *r* that permits accurate calculation of regression lines by the classical least-squares method (7). When symmetrial expansion causes *r* to recede below the critical value it may be possible to further extend the span of linearity by holding the lower or higher limit fixed and expanding the calculation of *r* into the opposing tail. In this way the maximum span of linearity is assured.

Results and Discussion

Table 1 details the calculation of the reference interval from the creatine kinase (CK) distribution in Figure 1. Progressive and symmetrical calculation of *r* around the median gave acceptable linearity up to 24-78 U/L. Further expansion either symmetrical or asymmetrical caused *r* to decline below acceptable limits. Thus maximum linearity was obtained between the 2.5th and 89.5th percentiles. For comparison the reference intervals calculated by conventional methods are also listed. In all cases, the conventional methods yielded wider reference intervals. This was to be expected since it is well documented that the conventionally estimated upper reference limit for CK is excessively influenced by extreme values, not only for men as aforementioned but also for women (8). With the exception of cholesterol, medical decision points lower than the 95th-97.5th percentiles have yet to be accepted.

Whilst the associated regression parameters of slope and intercept have no obvious use in the reference interval calculation, the standard error of the estimate (Sy/x), in my opinion, has. Because it can be considered as an average standard deviation in the relationship between x (percentile) and y (analyte value), we can estimate the 95% confidence intervals of the reference interval endpoints as y ± 2 Sy/x. Thus from Table 2 the CK reference interval expressed with confidence intervals parenthetically is 24 (19, 29) — 79 (73, 83) U/L.

Because in this method values are deemed typical or atypical by statistical rather than clinical criteria, the method would seem to have the most potential in the estimation of reference intervals from patients' results. For years biochemists have been attracted to this readily accessible data base, but early methods proposed to extract reference intervals were criticised because they assumed an underlying gaussian distribution of healthy values within the patient population. No such criticism can be levelled at this present method; all that is assumed is that the population distribution is continuous and essentially unimodal.

The data in Figure 2 and Table 3 shows application of this method to a select patient group, viz. all patients for whom angiotensin converting enzyme (ACE) was requested during a twelve month period. Serum ACE is usually requested for

Table 2: *Reference Interval from CK Distribution.*

CK interval U/L	*r*	Sy/x	CK interval U/L	*r*	Sy/x
48-54	0.9999	0.002	30-72	0.9928	1.67
45-57	0.9986	0.29	27-75	0.9909	2.11
42-60	0.9992	0.28	24-78	0.9901	2.44
39-63	0.9991	0.38	24-81	0.9875	2.87
36-66	0.9976	0.73	21-81	0.9889	2.83
33-69	0.9956	1.14	18-84	0.9870	3.35

Method	CK reference interval, U/L
r	24-78
parametric	19-96
log-parametric	25-104
percentile	25-107

Table 3: *Reference Interval from ACE Distribution.*

ACE interval U/L	r	Sy/x	ACE interval U/L	r	Sy/x
44-56	0.9982	0.29	26-77	0.9935	1.94
41-59	0.9923	0.90	23-80	0.9923	2.33
38-62	0.9953	0.88	20-83	0.9909	2.77
35-65	0.9964	0.93	17-86	0.9895	3.24
32.71	0.9957	1.21	14-89	0.9880	3.73
29-74	0.9950	1.50	11-92	0.9864	4.26

This reference interval: 20-83 U/L

Previously established interval: 24-78 U/L (see text)

Table 4: *Reference Interval from AAP Distribution.*

AAP interval U/L	r	Sy/x	AAP interval U/L	r	Sy/x
72-87	0.9916	1.02	47-112	0.9885	3.29
69-92	0.9897	1.50	47-107	0.9917	2.61
62-97	0.9912	1.75	42-107	0.9914	2.85
57-102	0.9908	2.17	37-107	0.9888	3.47
52-107	0.9900	2.66	42-112	0.9894	3.37

Reference interval from total data (n = 471): 20-83 U/L

Reference interval from culled data (n = 213: 42-104 U/L (see text)

one of two reasons: (a) the diagnosis and monitoring of sarcoidosis — in which an elevated level of ACE indicates active disease, or (b) to assess patients' compliance with antihypertensive ACE inhibitor (e.g. captopril) therapy — whence low levels are expected. In Figure 2 the maximum span of linearity occurred between the 5th and 87.5th percentiles. The corresponding reference interval of 20-83 U/L compares favourably with 24-78 U/L previously established by the nonparametric percentile method from a reference population.

Applying the method to a more heterogeneous patient group was equally successful. Figure 3 shows the frequency distribution of plasma alanine aminopeptidase (AAP) activity in 471 consecutive patients for whom a liver function profile was requested. AAP is an enzyme of diagnostic value similar to gamma-glutamyltransferase. As shown in Table 4, maximum linearity achieved by symmetrical expansion of the calculation of *r* was 52-107 U/L but this could be extended down to 42 U/L by holding the upper limit fixed at 107 U/L. The reference interval thus obtained was 42-107 U/L and corresponded to the 2nd-81st percentiles. This was in remarkable agreement with 42-104 U/L calculated from a subset of the same data base according to the strategy of Sinton et al(9), whereby only those AAP values associated with liver function profiles wholly within reference limits were used.

Now I must point out that for reasons of brevity and clarity, in these examples I have used grouped data taken from the frequency distributions (histograms). Ideally, the value-percentile plot should be constructed from data that has been ranked individually. This requires substantially more work but it does give more points on the value-percentile plot which in turn leads to more accurate demarcation between typical and atypical values. Some consideration of the size of the data base is also appropriate here. Merkouriou and Dix suggested that since the method is based on percentiles a minimum of 120 subjects was required. From my own experience with the method I think that is too few. This is especially so with highly skewed patient populations and I recommend at least twice that number. In any case, 120 is now considered manifestly inadequate for the nonparametric percentile method when dealing with skewed distributions(10) up to 700 may be needed.

A purely statistical concept of the reference interval having no connotations of health or disease is clearly unconventional. Typical values may or may not indicate the absence of disease. Clinical follow-up studies must be carried out to validate the reference intervals obtained and correlations between atypical values and disease must be established. It seems to me that a logical extension of such studies would be to collect data from patients with confirmed diagnoses and thence typical and atypical values for that particular pathology. With the days of computer archiving of results upon us this is now possible. It behoves us all to supply more useful information to clinicians. Indeed, Interpretive Reporting has been identified by the IFCC as one area worthy of new endeavour (11).

Readers may be bemused by the choice of 0.9900 as the minimum acceptable value of *r*. Why not 0.9950 or 0.9880, or some other value? It must be stated that 0.9900 has no special virtue other than that referred to previously. It is purely an arbitrary but objective choice of typical behaviour that is analogous to $P<0.05$ and $P<0.01$ being chosen as arbitrary but objective measures of statistical significance (12). Clinical follow-up studies may require us to alter the critical value of *r*. An increase would restrict the reference interval to values nearer the median thus increasing test sensitivity at the expense of test specificity, whereas a decrease in the critical value of *r* would expand the reference interval thus improving specificity but reducing sensitivity. Different tests may well have different critical values of *r*.

Conclusion

This paper has described a simple, if unconventional, method for estimating reference intervals. Because it is particularly applicable to readily accessible patients' results, every laboratory can use it with their own data.

References

1. West DW., Ash O. Adult reference intervals for 12 chemistry analytes: influences of age and sex. *Am J Clin Pathol* 1984; **81:** 71-76.
2. Expert Panel on Theory of Reference Values: Scientific Committee, Clinical Section (IFCC): The theory of reference values. Part 5. Statistical treatment of collected reference values. Determination of reference limits. *Clin Chim Acta* 1984; **137:** 97F-114F.
3. Riley WJ. Normal values, reference ranges and decision points. *Clin Biochem Revs* 1987; **8:** 5-10.
4. Bais R, Conyers RAJ, Rofe AM, Tormet RI, Geary TD. Creatine kinase reference intervals determined from a multi-centre data pool. *Pathology* 1988; **20:** 367-372.
5. Broughton PMG, Buckley BM. The need for better plasma cholesterol assays. *Ann Clin Biochem* 1985; **22:** 547-549.
6. Merkouriou S, Dix D. Estimating reference ranges in clinical pathology: an objective approach. *Statistics in Medicine* 1988; **7:** 377-385.
7. Wakkers PJM, Hellendoorn HBA, Op De Weegh GJ, Heerspink W. Applications of statistics in clinical chemistry — a critical evaluation of regression lines. *Clin Chim Acta* 1975; **64:** 173-184.
8. Miller WG, Chinchilli VM, Gruemer H-D, Nance WE. Sampling from a skewed population distribution as exemplified by estimation of the creatine kinase upper reference limit. *Clin Chem* 1984; **30:** 18-23.
9. Sinton TJ, Cowley DM, Bryant SJ. Reference intervals for calcium, phosphate, and alkaline phosphatase as derived on the basis of multichannel-analyzer profiles. *Clin Chem* 1986; **32:** 76-79.
10. Linnet K. Two-stage transformation systems for normalization of reference distributions evaluated. *Clin Chem* 1987; **33:** 381-386.
11. Young DS. International Federation of Clinical Chemistry: present and future. *Clin Chem* 1988; **34:** 202-207.
12. Jones, PK. R.A. Fisher and the 0.05 level of significance in medical studies [Editorial]. *J Lab Clin Med* 1988; **111:** 491-492.

The Pacific Way

P.P.T.C. News

Tutor Co-ordinator

Michael Lynch, the Tutor Co-ordinator for the P.P.T.C. has taken up a two year assignment with W.H.O. as Development Technical Officer for HIV Testing Services in the Western Pacific. Mike will be based in Manila but as Field Technical Officer will be visiting many Pacific countries to implement laboratory Testing Methods for HIV.

Gilbert Rose has been appointed Tutor Co-ordinator for two years in Mike's absence. Gilbert, formerly the Charge Technologist, Microbiology Department, Christchurch Hospital, has just completed a three year contract as Technical Research Officer with the Papua New Guinea Institute of Medical Research. Gilbert was based in Tari in the Eastern Highlands. In addition to his involvement with research projects Gilbert worked in the Microbiology Laboratory of Tari Hospital.

1989 Programme P.P.T.C.

The P.P.T.C. has undertaken on behalf of the New Zealand Government to assist Western Samoa with the devopment of a 3 year Technical Training Programme for Medical Laboratory Technologists in Western Samoa.

February — May 1989

The first few months of 1989 will be spent on planning and implementing the first stages of the course in Western Samoa as well as firmly establishing and further developing the Quality Control (QC) Programme for Pacific Island Countries. The QC programme will cover Biochemistry, haematology and Microbiology.

22nd May — 11th August
Immunohaematology Course — P.P.T.C.

11th September — 1st December
Microbiology Course — P.P.T.C.

Annual Report of P.P.T.C. 1988.

The Centre is now into its seventh year of activity and this, like the others, is proving to be one of continued progress and development. New activities have included the successful introduction of a course on the sexually transmitted diseases. This course of six trainees included two medically qualified WHO fellows who attended from the Lao People's Democratic Republic. In addition to this, the Pacific Island Quality Assurance Programme was expanded to include Clinical Chemistry. The Management Committee of the P.P.T.C. would like to take this opportunity to thank Ms. Claire Murphy for her major role in introducing this new component to the Programme.

During the 1987/88 year, twenty eight trainees from a range of Pacific Island countries attended courses at the P.P.T.C. In addition to the courses in Haematology/Blood Bank Technology, Sexually Transmitted Diseases and Medical Microbiology, specialised training was provided in Blood Bank Management and HIV testing for two trainees from Fiji. These two special training projects were made possible by the generosity of the Norman Kirk Memorial Trust.

That the Centre has completed another successful year is due in large measure to many individuals and organisations who have given generously of knowledge, training skills and financial assistance. To all, the Management Committee of the Centre express sincere thanks. Following the decision at the last Annual General Meeting to appoint a part-time technologist to assist Mr Lynch (Tutor Co-ordinator) with the extra work load generated by the introduction of a new course and the expanded Quality Assurance Programme, Mrs Christine Story was appointed in May of this year. Mrs Story's contribution to the work of the Centre has been most valuable and greatly appreciated.

Special thanks are extended to the Wellington Hospital Board for the use of the teaching laboratory area and for the on-campus accommodation facilities for the trainees. It is only through the use of these key facilities that the Training Centre can operate and this major contribution by the board is greatly appreciated not only by the P.P.T.C. but also by the many Pacific Island hospitals who have sent trainees to Wellington over the past seven years.

Thanks are once again extended to the Ministry of Foreign Affairs for the aid grant which enables the Centre to function, and to the Executive and Project Officers of the External Aid Division for valued assistance and guidance.

Central to the activities of the Centre are a continuing flow of suitable trainees, and to achieve this requires the help and co-operation of a number of organisations. Included in these are the Western Pacific Regional Office of the World Health Organisation and the International Divsiion of the New Zealand Department of Health. To the officers of both these organisations for their co-ordinated efforts in assisting the Centre, sincere thanks are extended.

This year, as in the past, we record our indebtedness ot the New Zealand Red Cross Society for the Health Science Scholarships which enable trainees to attend Courses at the Centre, and for the increasing administrative and logistical support which comes so cheerfully from the National Headquarters Staff and assists so greatly in the smooth running of the Centre's affairs.

Gilbert Rose — The new Tutor Co-ordinator, P.P.T.C. Don't they wear lab coats in Tari?

The continued support of the New Zealand Institute of Medical Laboratory Technology has been greatly valued during 1987/88, particularly in respect to continued assistance with the quality assurance programme for the clinical laboratories of the Pacific Islands. Thanks are due also to numberous members of the Institute for the effort and time they have taken in locating and sending useful items of equipment to the Centre for use in the teaching laboratory. As in previous years where items of equipment have been duplicated or not required, the Centre, with the assistance of Red Cross, has sent them on to Pacific Island laboratories where a known need exists.

The Centre would like to take the opportunity in presenting this report to acknowledge with thanks the continued interest and generosity of the Wellington Rotary Club in the work of the Centre. The funding of automated pipettes and a bench top centrifuge for use in the Sexually Transmitted Disease Course was greatly appreciated and made a substantial contribution to the successful introduction of that course. In relation to the above course the Committee of the Centre record sincere thanks to Dr Martin Tobias and Staff of the National Health Institute for their valued input.

We are indebted also the the New Zealand Association of Clinical Biochemists for both financial and technical assistance in getting the clinical chemistry component of the Pacific Island Quality Assurance Programme under way and look forward to receiving their much valued help in the future.

In conclusion, sincere thanks are expressed to Mr Michael Lynch, Tutor Co-ordinator of the Centre. Mr Lynch's efforts within the lecture room and teaching laboratory have once again earned the appreciation of the Committee and trainees for a job well done. To all the staff in the Department of Laboratory Services at Wellington Hospital who gave so generously of their own free time and technical expertise during the past year, the Committee extend sincere thanks.

Trainees Who Completed Courses At The Pacific Paramedical Training Centre, September 1987 — November 1988

Kesler Lakutak (WHO Fellow), Kosrae, Sept-Nov 1987 Medical Microbiology; Tekaibeti Tarataake (WHO Fellow), Kiribati, Sept-Nov 1987 Medical Microbiology; Satis Chand (NZ Red Cross), Fiji, Sept-Nov 1987 Medical Microbiology; Faiatea Latasi (NZ Overseas Development Aid), Tuvalu, Sept-Nov 1987 Medical Microbiology; Geoffrey Wuatai (NZ Overseas Development Aid), Cook Islands, Sept-Nov 1987, Medical Microbiology; Mason Alatala (NZ Overseas Development Aid), Solomon Islands, Sept-Nov 987, Medical Microbiology; Misiona Nicholas (NZ Overseas Development Aid), Niue, Sept-Nov 1987 Medical Microbiology; Mohammed Iqbal (Norman Kirk Memorial Trust), Fiji, Feb-Apr 1988 Haematology/Blood Bank Technology); Peia Beu, (NZ Overseas Development Aid), Cook Islands, Feb-Apr 1988 Haematology/Blood Bank Technology; Foliaki Sosene (NZ Overseas Development Aid), Cook Islands, Feb-Apr 1988 Haematology/Blood Bank Technology; Tala Mauala (NZ Overseas Development Aid), Western Samoa, Feb-Apr 1988 Haemaology/Blood Bank Technology; Bennie Otoa (NZ Overseas Development Aid), Papua New Guinea, Feb-Apr 1988 Haematology/Blood Bank Technology; Dr Traykhouane (WHO Fellow), Lao People's Democratic Republic, June-Jul 1988 Sexually Transmitted Diseases Course; Dr Te Thammawong (WHO Fellow), Lao People's Democratic Republic, Jun-Jul 1988 Sexually Transmitted Diseases Course; Ngatokorua Teariki (NZ Overseas Development Aid), Cook Islands, Jun-Jul 1988 Sexually Transmitted Diseases Course; Bennie Otoa (NZ Overseas Development Aid), Papua New Guinea, Jun-Jul 1988 Sexually Transmitted Diseases Course; Fetuao Tavai (NZ Overseas Development Aid), Western Samoa, Jun-Jul 1988 Sexually Transmitted Diseases Course; Naibuka Nakabuniceva (NZ Red Cross), Fiji, Feb-Mch 1988 AIDS Laboratory Diagnostic Methods; Jane Tyler (Norman Kirk Memorial Fund), Fiji, Apr-May 1988 Blood Bank Management; Areta Aritiera (NZ Overseas Development aid), since May 1982, Extended Training Supervision; Resitiara Mataumomâ (NZ Overseas Development Aid), Western Samoa, Sep-Nov 1988 Medical Microbiology; Fred Fafale (NZ Overseas Development Aid), Solomon Islands, Sep-Nov 1988 Medical Microbiology; Matanornoa Nicholas (NZ Overseas Development Aid), Cook Islands, Sep-Nov 1988 Medical Microbiology; Reikiko James, (WHO Fellow), Truk, Sep-Nov 1988 Medical Microbiology; Lucy Dibay (WHO Fellow) Yap, Sep-Nov 1988, Medical Microbiology; Matlina Whitney (WHO Fellow), Marshall Islands, Sep-Nov 1988 Medical Microbiology; Taukolo Nonu (NZ Overseas Development Aid), Tonga, Sep-Nov 1988 Medical Microbiology.

The Governor General, Sir Paul Reeves, with the students of the Medical Microbiology Course, P.P.T.C. September-November, 1988.

P.P.T.C. — Facilities And Equipment

The major items of equipment in the teaching laboratory remain in good working order with the exception of one of the older model 37°C incubators which has now passed the stage of further repair and will require replacement in the near future. The generous gift of automatic pipettes and a clinical centrifuge by the Rotary Club of Wellington were most welcome and have proved invaluable. Apart from minor maintenance requirements, no problems of any note were encountered during the past year and the facilities and equipment remain very satisfactory overall.

Items of equipment and disposables donated to the Centre and surplus to our requirements were sent to Pacific Island Laboratories who were in need. On this basis, equipment including centrifuges, a flame photometer, petri dishes, culture media and antisera have been sent to Western Samoa, Tuvalu and Fiji. This service by the P.P.T.C. is greatly appreciated by many of the small Pacific Island Medical Laboratories who function on tight budgets and have difficulties in replacing work out equipment.

Visitors To The P.P.T.C.

Visitors were received during the year from the Red Cross Society, the Department of Health and Service Clubs. On occasions, visitors from these organisations have had overseas visitors with them who were involved in technical training programmes for developing countries and for this reason were interested in the teaching strategies developed by the P.P.T.C.

Visitors of note include Ms Elizabeth Williams, Assistant Director Training, Ministry of Foreign Affairs, Mr Alan Oaisa, High Commissioner for Papua New Guinea, Dr John Jewsbury, from the Liverpool School of Tropical Medicine, and external examiner in Parasitology for the Fiji School of Medicine, Dr Helen Bichan, Acting Chief Medical Officer, Wellington Hospital and Sir Paul Reeves, Governor General of New Zealand.

INSTITUTE BUSINESS

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or the Editor, P.O. Box 35-276, Auckland, 10.

Membership Convenor
Geoff Rimmer
P.O. Box 29-115, Greenwoods Cnr, Auckland.

Membership Fees and Enquiries

Membership fees for the year beginning April 1, 1989 are:

For Fellows — $104.00 GST inclusive

For Associates — $104.00 GST inclusive

For Members — $52.00 GST inclusive

For Non-practising Members — $33.00 GST inclusive

All membership fees, changes of address or particulars, applications for membership or changes in status should be sent to the Membership Convenor at the address given above.

Members wishing to receive their publications by airmail should contact the Editor to make the necessary arrangement.

Membership Sub-Committee Report March 1989

Since the October meeting there has been the following changes:

	17.3.89	22.2.89	12.10.88	31.8.88
Membership	1699	1623	1553	1499
less resignations	4	11	3	8
less G.N.A.	—	15	—	14
less deletions	—	—	—	—
less deceased	—	—	—	—
	1695	1597	1550	1476
plus applications	14	102	73	77
plus reinstatements	—	—	—	—
	1709	1699	1623	1553

Applications for Associateship:

EXTON, Katherine, Auckland; McCOMB, Penelope, Auckland; FINDON, Glenne, Auckland; HOSKEN, Leigh, Auckland; POLOAI, James, Auckland; WOODS, Gillian, Auckland; MASON, Elizabeth, Auckland; JOHNSTON, Yvonne, Auckland; BROOKING, Judith, Auckland; TAYLOR, Faith, Auckland; MAYES, Neil, Auckland; HUMPHREY, Myra, Auckland; JONES, John, Auckland; PAPERA, Ina, Kawakawa; HOLLOWAY, Russell, Auckland; CAMPBELL, Anna, Auckland; TOY, Gail, Auckland; MURPHY, Deborah, Auckland; HAINES, David, Auckland; EDWARDS, Philippa, Gisborne; SMITH, Murray, Tauranga; PAYNE, Gordon, Tauranga; PETERS, Jennifer, Gisborne; McPHERSON, Lois, Wellington; WHITE, Glennis, Wellington; ANDERSON, Margaret, Wanganui; MacKAY, John, Wanganui; LOCKYER, Alan, Hastings; KERR, Alistair, Palmerston North; BELL, Robyn, Dunedin; MacDONALD, Elizabeth, Gore.

Applications for Membership:

COWLEY, Norma, Auckland; TETAI, Reremoana, Auckland; PATEL, Mansukh, Auckland; TIPENE, Lillian, Kawakawa; HUNTER, Ian, Auckland; VASS, Alison, Auckland; SEATH, Verena, Kawakawa; TORRIE, Julie, Auckland: WRIGHT, Jackie, Auckland; COUPER, Anne, Auckland; TATE, Gina, Auckland; PRASAD, Kamala, Auckland; MOUNTFORD, Louise, Auckland; ARCHER, Robyn, Auckland; GOUNDER, Pushpa, Auckland; BALLARD, Lynda, Auckland; AIRD, Megan, Auckland; WARDEN, Mark, Auckland; STANLEY, Karen, Auckland; SELMAN, Gillian, Auckland; BAILEY, Alison, Auckland, JULL, Tessa, Auckland; HANRAHAN, Vickie, Whangarei; ADLINGTON, Janet, Auckland, McCONNOCHIE, Evelyn, Auckland; TAUAT, Nicky, Auckland; KIERNAN, Michelle, Auckland; GLAVAS, Christine, Auckland; GILBY, Gaylene, Auckland; SIMPSON, Wendy, Auckland; WALL, Alexandria, Auckland; POWELL, Johnathan, Auckland; McCREADY, Wendy, Auckland; REECE, Tracey-Maree, Auckland; BRADBURN, Nicola, Auckland; COOK, Lynne, Auckland; PRIDDLE, Terrence, Auckland; STIMPSON, Vicki, Gisborne; ROSS, Sarah, Waikato; OAKLEY, Alison, Waikato; ELGAR, Pamela, Rotorua; PAGE, Nicholas, Rotorua; PARKINSON, Christine, Rotorua; FUNNELL, Alison, Gisborne; STOPFORD, Roncevelle, Gisborne; LIBEAU, Delwyn, Hamilton; HAMPTON, Glenis, Mt Maunganui; BROWN, Shirleen, Hamilton; POWELL, Kathryn, Hamilton; McFALL, Julie, Hamilton; FLACK, Christine, Palmerston North; ADAM, Sandra, Waipukurau; PRONK, Marcel, Hastings; STILL, Lee-Ann, Hutt; GOSSE, Michelle, Hutt; McKINNON, Jane, Palmerston North; MAITLAND, Carla, Palmerston North; SMALL, Michael, Wanganui; DORAN, Stephanie, Wanganui; HOLDER, Kathryn, Palmerston North; FITZSIMONS, Glynnis, Feilding; HOLLEY, Maxine, Hutt; FROGGATT, Vivienne, Wellington; ANDERSON, Carmen, Napier; HOLDAWAY, Christina, Palmerston North; WEARNE, Kim, Wellington; BILLETT, Susan, Napier; BLAKE, Taraleigh, Wairoa; WESTNEY, Penny, Christchurch; McKENDRY, Sara, Christchurch; PIKE, Linda, Christchurch; JEFFERY, Nadine, Dunedin; DAVIES, Janet, Dunedin; VOICE, Lee-Ann, Christchurch; GRACEY, Sandra, Christchurch; MURRAY, Elizabeth, Christchurch; DUNLOP, Carol, Christchurch; RICKETTS, Angela, Christchurch; MOIR, Carolyn, Invercargill; AVIS, Karen, Dunedin; CROKER, Deborah, Dunedin; LAU, Siong Kiew Peter, Sarawak, Malaysia.

Applications for Non-Practising Membership:

HALL, Faye, Wellington; MILLER, Euan, Auckland.

Resignations:

ROIGARD, A.; VALLIS, L.; VAUCHELLE, L.K.; GODBY, N.T.;

CALEY, K.J.; BRAAN-STROO, S.; GEORGE, E.A.; KARPIK, A.; NORRIS, D.M.; DUDSON, W.J.; GREENWOOD, S.M.; ROBERTSON, K.L.; SOWDEN, R.C.; HUTTON, S.L.

Gone No Address:

FINCH, R.M.; SHORT, R.J.; CAMPBELL, F.L.; FIELDS, S.A.; WHITTAKER, G.E.; ROSS, D.J.; SMITH, J.G.; DOUGLAS, W.A.; ANDREW, S.; PEARCE, J.L.; GOODYER, C.A.; McPHERSON, D.J.; WRIGHT, N.E.; SHORT, R.J.

LETTERS TO THE EDITOR

Dear Sir,

re: A position in a New Zealand Laboratory

I am a Swedish Laboratory Technician who would like to go overseas and work. I finished the School of Medical Laboratory Sciences in December 1981 and have since then worked as a Lab. Tech. in a Clinical Chemistry Laboratory.

My duties consist of ordinary Lab. Tech. work such as taking blood-tests and analyzing them. The kind of tests we carry out are all the haematological tests, biochemistry tests and some immunological tests such as Hepatitis virus, HIV a.b. and Rubella a.b. During evenings, nights and weekends the lab also takes over the bloodbanking duties.

Last year I spent 7½ months working as a Lab. Tech. for the UN in south Lebanon. Now I'm back in Sweden but I'm dying to go overseas again, and that is why I'm writing to you.

If you have any work for a 26 years old girl from Sweden please contact me and I will send you my personal record.

Yours sincerely,
Inger Pettersson
Badhusvägen 2
460 20 Sjuntorp, Sweden.

Tel. 0520-40798.

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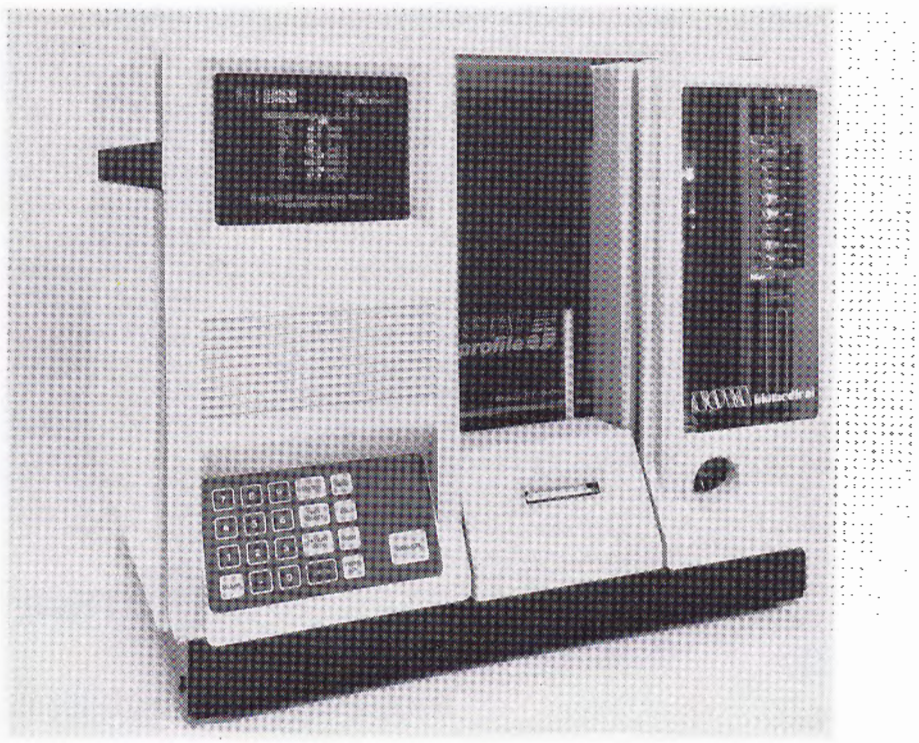

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