

chemistry

in new zealand

Vol 53 No 1 February 1989

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Contact Tony McDonagh, General Manager, ATA, by letter or fax (address overleaf). Please provide the usual personal background information and the reason why you believe that you can succeed in this enterprise.



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Front Cover Story





in recent statements and articles the New Zealand Chemical Safety Council has pointed out that "fear of chemicals stems from ignorance". From other quarters we hear that "Chemistry has become mysterious, and that's a pity". Nobody in his right senses would quarrel with these sentiments. However, it is timely to point out that the hazards arising from the use of many chemicals can be minimised by the installation of the right furnecupboard. This certainly applies to many laboratory situations. What is the right fumecupboard, and how does one go about selecting it?

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Advertising Features

February:

In this issue we feature products for use in biochemistry. Also, in todays environment of heightened awareness of the potential hazards in handling biological materials there is a timely Cover Story on fumecupboards and laminar flow cabinets - essential supporting equipment in biochemical laboratories.

April:

The features products will be the full range of chromatography equipment - GC, HPLC, and a special note on ion chromatography.

Guest Editorial

Innovation in Teaching Chemistry

J.Gavin Fletcher MSc FNZIC

During my younger years I had the opportunity of sharing (teaching) the joys of chemistry with students at secondary and tertiary levels. More recently, I have assisted my two sons through their secondary chemistry/science curricula, and as chairman of the Science Fair Board - been keenly interested in science projects at intermediate and secondary levels.

From these experiences, from my experience as the Director of a highly successful Research Association, and as the owner of a food manufacturing company, I would like to urge our chemistry educators to take a really fresh took at their influence over our future scientists. In the future, New Zealanders will have to live by their wits and knowledge to a far greater extent than before. This will require much more emphasis on science technology, engineering and marketing than in the past. I have had the opportunity to visit several Japanese heavy engineering research establishments as well as metallurgical engineering and science centres of excellence around the world. Lately I spent some time at the Singapore Science Centre. Clearly we - as chemists (for science educated people) must make sudden and dramatic changes, if we are to maintain any kind of world class science base. As our applied farming and manufacturing bases shrink, fewer science educated people are needed, with the result that fewer educators are needed etc. With the noises for economic union with Australia becoming stronger, coupled with the present "user pays etc" policies of the NZ Government, it is likely that our tertiary science teaching/research resources will be dissipated until New Zealanders will have to go to Australia or Japan to complete their education.

In part I believe this trend can be reversed equally dramatically by innovation in the teaching of chemistry. What the country needs is innovative entreprenuers who have a sound education in chemistry (and for that matter other economic disciplines) and who are enthused and encouraged to "have a go". I believe there is huge potential for increased wealth for New Zealand if only we had bright people adequately trained in chemistry and then in business techniques.

But this will not happen if all our bright young people continue to favour medicine and other new productive disciplines. In order to attract the brighter students to chemistry, the subject and the multitude of industries based on it, must be presented in an attractive way - that is once again where the teacher comes in.

I have been greatly disheartened to witness the almost nil
progress in this process since I
sat School Certificate in 1955
and then we were taught exactly
the same information using the
same methods as those in 1940!
The presentation of Chemistry in
our schools has not changed with
the times. It is not good enough
for the next generation if they are
to enjoy a reasonable standard
of living in New Zealand (unless
we wish our science and Technology to be done by expats).

Where are the videos of New Zealand Chemistry based products being shown to schools? Where are the travelling road shows of how exciting chemistry

When do the clever, innovative chemistry teachers show their colleagues their ideas, charts, experiments, demonstrations, computer programs etc?

Where are the participative exhibitions to involve the public in chemistry?

Where are the brightly coloured textbooks, videos, slides and other visual aids?

Where is kitchen chemistry, laundry and motorcar chemistry being demonstrated or experienced in our schools?

Who is making chemistry relevant and challenging to the 17 year old?

Where are the prizes and recognitions for New Zealand chemists who have done well? (Do school pupils ever hear of them?)

Who is marketing chemistry as the basis of a career - or a career in itself?

Why isn't the Tearaway magazine full of the joys of chemistry?

Those who write syllabii, and teach chemistry, should have a jolly good look at their product and change it for a 1990's version.

Comment

Research Associations Provide Model For Research Funding

In the current debate on research funding, Research Associations provide a good example of how joint funding by Government and industry can support both long and short term research, says the Director of the Coal Research Association of New Zealand, Dr. Rob Whitney.

Speaking on the work of the Association at a recent DSIR seminar, Dr Whitney said that there are good reasons why the Government continues to support funding for research into coal.

It is New Zealand's largest energy resource by far and will continue to be used long after other resources have been depleted.

Coal reserves are regional assets in terms of providing jobs and opportunities for export and industrial development.

Coal is a complex material whose properties still need investigation and, as deposits vary from country to country, we need to know what can be done with

the particular coals that we have.

Commenting on the movement towards "market-driven" research, Dr Whitney said that he thought that there were a number of advantages in this for the scientific community.

Research which was produced in response to demand had much more chance of being put to use.

Time and money would not be spent endlessly refining technology for which there was no demand.

Research projects, which at present tended to be limited by the resources of individual organisations, could become the subject of co-ordinated efforts, aimed at solving particular problems.

Dr Whitney said that he believed science and technology were essential to the future wellbeing of New Zealand. Scientists needed to convince the community of this and those carrying out basic research should, he said, be sensitive to national priorities. 1989 NZIC/NZBS CONFERENCE
22-24 August, University of Waikato, Hamilton

The overall format of the annual combined NZIC/NZBS conference, to be held this year in Hamilton, has been worked out by the organising committee under the chairmanship of Prof. Ken Mackay, with Dr Lyndsay Main as secretary.

The theme for the conference is to be "Chemistry in New Zealand". It is hoped that the conference will act as a window for New Zealand science, at a time when far reaching decisions are being made regarding its future. Emphasis will be placed on local speakers, who are being invited to give plenary review lectures on various topics of expanding interest in New Zealand. Student participation at the conference is to be strongly encouraged.

The local organisers of the specialist group fecture and poster sessions would be pleased

Conferences And Courses Third Coal Research Conference, Wellington, 9-11 October 1989

The purpose of the Conference is to provide a forum for exchange of information and ideas between research workers and practitioners in the field.

to receive suggestions for topics and speakers. They are: Analytical (Dr Max Sutton, MAFRuakura, P.B., Hamilton), Chemical Education (Dr Malcom Carr, University of Waikato, P.B. Hamilton), Chromatography (Dr Peter Robinson, Waikato Polytechnic, P.B., Hamilton), Industrial/Applied (Dr Nath Pritchard, Electricorp, Huntly and Dr Chris Kirk, University of Waikato, P.B. Hamilton), Inorganic (Prof. Ken Mackay, University of Waikato, P.B.Hamilton). Organic (Dr Lyndsay Main, University of Waikato, P.B. Hamilton) and Physical (Dr Peter Morris, University of Waikato, P.B. Hamilton).

The active participation of Institute members would be very welcome, because we want to make attendance at this conference a valuable and enjoyable experience.

Topics of interest for the Conference include: Coal Quality, Marketing, Environment, Utilisation, Coal Science, Resources.

For further information contact: The Conference Secretary, Third Coal Research Conference, P.O. Box 31-244, Lower Hutt, New Zealand.

People

NZIC Chemical Education Award

The 1988 recipient of this award is Mrs May Croucher.

After graduating from Otago University, she worked for a year in the Food section of Chemistry Division, DSIR, before taking up teaching at Rotorua Girls' High School in 1969. There, she taught Chemistry, Science and Mathematics until July 1988. In the earlier years of teaching she also worked in a laboratory at the Forest Research Institute during the Christmas vacations.

While teaching, she participated in and helped to organise in-service Chemistry courses for Rotorua and Bay of Plenty regions. She has been involved in various writing sessions, the latest being a section of teachers' Guide Notes for the new Form 7 Chemistry syllabus.

May is co-author of three books used in secondary schools; Chemistry Guidebook (published 1975 and now in its 3rd edition), Chemistry (published 1981 for 3rd and 4th forms) and Science Guidebook (published 1988).

In 1986 and 1987 she was tutor at the Rotorua Outpost of Hamilton Teachers' Training College. Since July 1988, she has been working as a Scientist at the Forest Research Institute. In her own words "Although I have very much enjoyed teaching, I succumbed to the lure of indulging my self in Chemistry again... especially now that my family is growing up."

Les Boulton of the Auckland Industrial Development Divison, DSIR, recently attended the 28th Annual Conference of the Australasian Corrosion Association held at Observation City Hotel in Perth, Western Australia. He presented a keynote address at the conference entitled "Corrosion of Austentic Stainless Steel Weldments", in the symposium on "materials performance". The conference was attended by 200 delegates from all states of Australia and other countries.

A.F. Wilson

Dr A.F.(Ashley) Wilson has been appointed to the new position of Director Quality Programmes at TMI New Zealand Ltd. Dr Wilson is responsible for TMI's new quality programmes. He had previously been with the NZ Forest Products group for 23 years holding positions as General Manager, Kinleith Industries and Managing Director of NZFP Technology Limited.

The author of 11 papers on technical, quality, planning and training subjects, Dr Wilson has had practical experience with a number of quality improvement programmes.

The TMI quality programme has been developed in Denmark. It embodies a new approach to quality improvement by emphasising personal quality as a starting point for product, service and organisation quality improvement.

CIT Appoints Head Of Science

Dr Martin Grinsted has been appointed to the position of Head of the School of Science at the Central Institute of Technology, Heretaunga.



Dr Grinsted gained a DPhil from the University of Waikato in 1977, having previously obtained a BSc(Hons) from Bristol University in 1971 and a Postgraduate Certificate of Education from Brunel University in 1972. He returned to the United Kingdom in 1978, spending 16 months undertaking postdoctoral research at Oxford University.

Or Grinsted has been on the staff of CIR since 1980, being Acting Head of the School of Science and Chairman of the Institute's Admissions Committee from 1987.

Living in Whiteman's Valley, he is married with four children and is a keen gardener, tramper and spelaeologist.

A.C. Kennett Award

The 1988 recipient of this award is Mr Dennis Hills of the Southern Industrial Development Division, DSIR.

Dennis has been associated with the plastics and rubber industries for 31 years, has published widely in the field, and is New Zealand's only Fellow of the Plastics and Rubber Institute. Thirteen years in the manufacture of industrial rubber goods were followed by three years in marketing and technical service (mainly related to adhesive products). He has been in his present

position of Scientist, Southern Industrial Development Division, with DSIR since 1974, involved in research development and trouble-shooting with all nonmetalic materials for South Island industry and has a national



responsibility for technical service to the rubber industry. An interest in community education and industrial training saw him receive the University of Canterbury's new Certificate in Continuing Education in 1983.

The A.C. Kennett Award is presented jointly by the Australasion Corrosion Association and the NZIC for work in the area of non-metallic corosion.

ANZAC Fellowship

Linda Boyd of Chemistry Divn, DSIR, in Auckland, has been awarded an ANZAC fellowship.

The fellowship enables New Zealanders to study and train in Australia - in order to benefit themselves, New Zealand and the furtherance of good relations between Australia and New Zealand.

Linda will be working for 6 months in Melbourne at the State Forensic Science Laboratory. She will be attached to the Fire Investigation Unit which works with the Victoria State Police. The unit attends all suspicious

and fatal fires with the police, and determines the cause of the fire.

This training period will cover all aspects of fire investigation including attendance at postmortems of fire victims, travelling all over Victoria, attending bush fires, and any mass disasters such as plane crashes, should they occur, laboratory analysis of fire debris, reconstruction of incendiary devices and giving evidence in court.

Hon. Gen. Sec.

And last, but by no means least, we note the passing of an era.

Towards the end of last year Dr John Rogers handed over the reins of office to his successor, Alan Turner. In mid-January the process was completed when the Carriers arrived to haul away the masses of material associated with the office. After 8 years in the job, John described the occassion as a momentous one, for both himself and his wife Molly (who has had almost as much involvement in the job as John himself).

John took over the office of General Secretary from Gavin Fletcher at the end of 1980, and has striven mightily on behalf of the Institute ever since. A full appreciation of his work was published in the December 1986 issue of Chemistry in New Zealand, on the occasion of his election to Honorary Fellowship, Of course, John's involvement with the Institute is by no means over - in August he was elected to the office of 2nd Vice President, so he will be with us for a few years yet.

Members will be concerned to learn that John has been quite ill lately and spent most of December in Auckland Hospital. He Is at home now however, and appears to be recuperating well. I am sure all members would join me in wishing him a speedy recovery.

Conference

2nd EurAsia Conference on Chemistry; Seoul, Korea, 16-20 April, 1990. The scientific programme will include coverage of the following areas of chemistry: bioinorganic, computational, coordination, environmental, natural products, organometallic, polymer, solid state, and solution chemistry.

Further information and registration forms are available from Prof C.J. O'Connor, Chemistry

Dept, Unviersity of Auckland, P/B, Auckland.

Working With Chemicals - an industrial safety symposium organised by the Auckland Branch, NZIC. 24 May 1989. Further information will be published in the next issue of Chemistry in New Zealand. Advance details may be obtained from Lester Stonyer, Continuing Education, University of Auckland.

Obituary

John Aggett FNZIC, 1936-1989

It is with much sadness that the N.Z.I.C. records the death of Associate Professor John Aggett, after a short illness, on January 12, 1989.

John entered Auckland University College in 1954 and among his classmates were Professor Barry Welch, (the late) Dr Michael Kingsford (Government Analyst), Dr Ray Golding (Vice Chancellor of James Cook University) and the author. He graduated MSc with First Class Honours in Chemistry in 1958 and PhD in 1961. After spending the next two years as a Research Officer at the Australian Atomic Energy Commission, where he was mainly concerned with the development of processes for recovering essential materials from spent reactor fuels, using solvent-extraction processes, John returned as a Lecturer in Chemistry to the University of Auckland in 1963 and became an Associate Professor in 1973. In 1969 he was awarded the prestigious Nuffield Foundation Travelling Fellowship in Science and Humanities and spent his leave at Imperial College, London with Professor West. There began his long-standing interest in mechanisms of atomisation and interference in non-flame atomisers and in atomic absorption analysis. He was also awarded a Fulbright Scholarship in 1984, for the furthering of his studies on trace elements in the environment.

In 1967, the Chemistry Department created an Analytical Section and from its inception John was responsible for directing the Departmental teaching of analytical chemistry. In that role he worked to build a teaching facility which adequateley prepared students both for professional careers in the wider community, and at the same time assisted the development of those who intended to further their studies in the academic environment. Given the difficulties which confront all programmes within the University which are dependent on expensive equipment, satisfactory progress required dedication and persistence. John had both qualities to a high degree and the department now offers a teaching programme in analytical chemistry at the third year level which provides graduates with the degree of sophistication necessary to enable them to supply industry with backgrounds suitable for their professional employment.

The popular departmental course on Environmental Chem-

istry was original developed jointly by Associate Professors John Spedding and John Aggett and after the former's death in 1984, John Aggett assumed full responsibility for this course.



In 1976, John was a co-founder of the New Zealand Trace Element Research Group and in 1977 he became an assessor for the Testing Laboratory Registration Council. In 1983 he was appointed to the Registration Advisory Committee for Chemistry and in these positions with TELARC he was able to contribute to the improvement of professional standards in industrial laboratories. Last year the Royal Society of New Zealand appointed him to the National Committee on Chemistry.

His research was innovative and flourishing and at the time of his death he was undertaking or discussing collaborative projects with Electricorp, NZ Steel, Manukau Harbour Authority, mining groups in the Coromandel and others.

For many years, John had surrered from painful arthritis and was unable to continue his active participation in sport. Nevertheless, he maintained a keen interest, especially in rugby. He also contributed tirelessly to community activities, particularly to the Intellectually Handicapped Children's Society and to his Church.

He is survived by his wife, 5 children and 3 grandchildren and to them we extend our sincerest condolences.

Charmian J. O'Connor John Vaughan 1920-88

John Vaughan, emeritus professor of chemistry at Canterbury University from his retirement in 1985, died in a car accident near Murchison on November 14th.

Vaughan came to Canterbury in 1949 having graduated from Cardiff and served in the Armaments Research Establishment and the Crookes Laboratories, London. After two years as assistant lecturer in general chemistry at Swansea, he came to Canterbury to collaborate with the late John Packer, who had developed over two decades, a course in organic chemistry with special reference to reaction mechanisms. The collaboration bore early fruit. Vaughan's enthusiasm gave the needed stimulus to the coversion of Packer's lecture notes into 'A Modern Approach to Organic Chemistry'. (1985). A four year delay in publication took something away from the view expressed in the foreword by C K Ingold - the book 'blazes a trail in the written presentation of organic chemistry'! Other authors came forward who were also realising the way in which the presentation of organic chemistry had to change. But 'Packer and Vaughan' was a notable achievement, and would have been so in any part of the

world of chemistry.

Vaughan's research interests
and influence were wide and
varied. Of special significance
was a short paper early in the

1960s which upset the current orthodoxy in the field of electrophilic, aromatic substitution. He took full advantage of the improved possibilities for research funding following the Hughes-Parry report on university education in New Zealand, and first, as a professor (1963) and then as head of department (1965) he created a strong group of investigators in his own field, and equally encouraged other groups in the varied aspects of chemistry available in his department.

Devoted as Vaughan was to chemistry, he was equally devoted to university affairs in general. His influence in his University and beyond it, was pervasive. His enquiring mind sought out what was happening, and produced an acute sense of what was possible and desirable academically and even politically. For more than one Vice Chancellor he was an eminence grise. He was a member of the founding Professorial Board of Waikato University, was active on the Council of the Cawthron Institute and in the management of Research Associations, concerned with wheat, ceramics and leather, and chaired the governing body of Christchurch Boys High School in its centennial year. (1981). He was President of the Institute in 1969 and it was during his chairmanship of the Canterbury Branch that the successful 'Chemistry in Action' series was initiated.

John Vaughan's contributions to teaching and research in chemistry were matched by his administrative talents, and the wide interests, including literature, history and rugby (both Welsh and New Zealand), which earned the respect and affection of his many friends.

H N Parton

Two-Day Course on Cathodic Protection

ACA Inc. is pleased to present a two-day intensive course on the theory and paractice of Cathodic Protection (C.P.) as a corrosion control strategy for subsea and sub-soil structures.

The Course Lecturer is Mr Ike Solomon, MSc (UMIST), Ml-CorrST, MIEAust., M.Cons. E.Aust, Principal of Solomon Corrosion Consulting Services P/L, of Melbourne, Australia, who has had 17 years experience in C.P. and corrosion consulting. He is an experienced lecturer and is a specialist in C.P. on structures

such as pipelines, well casings, offshore platforms, building services, LPG service station tanks, wharves, boats and concrete structures.

The course contents will include: Introductory Corrosion Theory, Cathodic Protection Fundamentals, Marine CP, CP of Underground Structures, Practical Aspects and Demonstrations, Discussion forums.

This is a unique opportunity to cut your corrosion bills by learning how to correctly utilize the C.P. technique. It is the first course of its kind held in New Zealand. More details of the course follow:

The course is being held twice: Course 1, Auckland, 3-4 April, 1989. At AIDD, DSIR. Brooke House, 24 Balfour Road, Parnell.

Course 2, New Plymouth 6-7 April, 1989. At Taranaki Country Lodge, Bell Block, New Plymouth. Cost ACA members \$350 (GST inct)

Non-ACA members \$385 (GST incl)

Write phone or fax for a Brochure, to the Course Convenor, as soon as possible: Les Boulton, AIDD, DSIR, P.O. Box 2225, Auckland. Telephone (09) 34116, Fax (09) 370618.

The Batt Symposium

Palmerston North, 26 August 1988

At the 1988 NZIC/NZBS Annual Conference in Palmerston North special symposia were held to mark the retirements of Professor R.D.Batt from the Department of Chemistry and Biochemistry at Massey University and Professor A.D.Campbell from the Department of Chemistry, University of Otago. The Conference Organising Committee felt that it would be fitting to have a written record of these symposia and is grateful to the editor for agreeing to publish edited versions in *Chemistry in New Zealand*.

The present issue is dedicated to the symposium held in honour of Professor Dick Batt. One of the lasting satisfactions upon retirement from a lifetime in the academic world must surely come from reflecting on the subsequent careers of graduate students whom one supervised at a formative stage of their lives. The symposium brought together six of Dick's former research students who were asked to comment briefly on their early association with him and then to present a short review of their own research, either current or looking back over a period of years. The result was an interesting spectrum of some significant contributions to biochemistry in New Zealand. Professor Batt's plenary address tracing his own lengthy journey from chemistry into biochemistry provided a very appropriate conclusion to a Conference which served to high-



PROF. R. D. BATT

light the strong and fruitful relationship between chemistry and biochemistry in New Zealand.

The following papers are, in most cases, considerably abbreviated versions of the addresses given but it is hoped that they preserve the essence of each contribution.

G.G.Pritchard

Becoming a Biochemist

Richard D. Batt

Department of Chemistry and Biochemistry, Massey University, Palmerston North

The Dictionary defines a reminiscence as 'an account of something remembered: the recurrence to the mind of the past'. Well, this is what I have been told to do - 'to reminisce'.

You will be relieved to know that I will be selective in what I recount - and although I want to name people with whom I have worked, omissions just mean that I will not have time to fit them all in.

How did it all begin? Well, I had a friend at secondary school whose mother was a diabetic having at least two injections of insulin a day. They lived near us and I sometimes was there when my friend or his brother gave their mother her injection. It seemed to me that medical research should be able to discover some way of avoiding this treatment which had the whole family under a degree of tension. I was already taking science subjects at school and in a vague sort of way I wondered whether I could do medical research some day - perhaps even on diabetes. I had no ambitions to be a doctor but when eventually I went to the University of Otago to do a science degree in chemistry, I realised that almost all medical research was being carried out by medical graduates. At about the time I was completing my BSc degree, the possibility of going into second-year medicine was mentioned to Sir Horace Smirk - the Professor of Medicine at the Otago Medical School and he strongly advised me to do a MSc in Chemistry before applying for admission to second-year medical classes. The MSc degree was a one year degree - and in those days you did your degree in minimum time because you just could not afford to take longer. As finals approached, the time came when I had

to put in my application to enter second-year medicine. At the last minute, I decided I had had enough of examinations and kept the application form. Eventually, the MSc examination results came out and Professor Soper wrote to offer me the John Edmond Fellowship in Chemistry - to do a non-existent PhD degree in Chemistry at the University of Otago. The degree was 'non-existent' in that the degree had been removed from the statutes of the then University of New Zealand early in the century but Professor Soper, who was on the Senate of the University of New Zealand, knew that the regulations to reintroduce the degree were with the Senate - and likely to go through. I was enrolled at Otago to do the prospective PhD (with Stanley Slater). Two years later, the thesis was in and not long after, Dr Briggs arrived in the department to conduct the oral examination. They passed me and Professor Soper was very pleased with the complimentary comments made by Sir John Simonsen - the external examiner. A PhD in Organic Chemistry was not getting me much closer to the sort of work I had thought I would want to do. At the beginning of 1948, I was appointed to a Junior Lectureship in Chemistry at the University of Otago and speculated about my future.

For my masterate thesis I had analysed the essential oil of the mountain cedar, Libocedrus bidwillii, under the supervision of Cedric Hassall (now FRS). We - i.e. Vernon Tyrrell, the glassblower - made a Lecky and Ewell fractionating column which had 40 theoretical plates. It gave marvellous separations of the oil constituents and I think they were as good as the first fractionations by gas-chromatography but, of course, much

slower. My PhD thesis included the structure of a new sesquiterpene, which we called gamma-curcumene. In 1948, I went back to get some more gamma-curcumene from *Libocedrus bidwillii* - but there was not a trace. My explanation is that gamma-curcumene was never there - we made it by the quaint chemical step we included in essential oil analyses in those days of extracting phenolic substances from the oil. I had a litre of oil for my masterate thesis and I was told to extract it with a dilute solution of NaOH to pull out the 'phenols'. Well, I got about 2 litres of the most resistant emulsion - it took weeks of techniques which were really 'blind magic' to finish up with about 750ml of oil - and no phenolic substances! Cedric was very cross.

Alot has happened since then. In those days, if it was water-soluble, it went down the drain. Our basic purification techniques were distillation and crystallisation. I can remember somebody coming into our basement laboratory for a short stay and he was separating the components of cow's urine on blotting paper. I think we all thought he was a bit 'whacky'.

What came out of all of that? Well, I will pick up the thread, but, years later, I was back at the Medical School and I heard that someone was going to give a talk on the mass spectrometry of sesquiterpenes in the Department of Chemistry. I went and heard how you could solve the structures of many sesquiterpenes in a few months with only a few milligrams of each compound. I had had 100g of my compound and it took me 2 years - and all the material - to get its structure. I knew, at that time, that I was going to Massey University and I was sure in my own mind that mass-spectrometry was a highly desirable acquisition for any up-and-coming department. I applied to the Golden Kiwi Committee for £50,000 to purchase a high resolution mass-spectrometer and the full range of Packard scintillation counting equipment. I first knew of the fate of my application when, on the bottom of a letter from Alan Stewart in 1964, he had written 'The Golden Kiwi has laid you a 50,000 pound golden egg'. To buy today what we bought with that grant would now cost nearly a million dollars. More important to morale was the recognition that Massey chemistry was more advanced - in instruments - than any other New Zealand Chemistry Department. Working on sesquiterpenes had its spinoff. We got our machine - we got Dick Hodges - the dothistromin project: became a saga - John Shaw and Stephen Kent are there to carry on the line - and there is Rex Gallagher, perhaps the closest of all of them to the direct line from sporidesmin.

To return to my first job - Junior Lecturer in the Otago Chemistry Department - in 1948. While I was on the staff that year, Professor Edson from the Medical School Department of Physiology gave a lecture on the Tricarboxylic Acid Cycle in the Chemistry Department and Lassume it was to the local branch of the Institute of Chemistry. I was very impressed, not only by Professor Edson who was an outstanding lecturer (matched only in my student years by Stanley Slater) but also by the chemistry of which we had heard nothing during our years of undergraduate and graduate study. I made an appointment to see him some time later, really to find out how you got into that sort of chemistry. He was very encouraging and his initial proposal was that I should join their staff in 1949 with the intention that I would be sent, in 1950, to the United States to learn about radioisotopes and their applications in medical research. Long before that possibility could have happened, the Medical School was asked to nominate someone for a Nuffield Demonstratorship in Biochemistry at Oxford University. It was to be in competition with a nominee from South Africa. I had no hesitation in saying I was prepared to be nominated but I did wonder about the paucity of people who knew about the Tricarboxylic Acid Cycle - it seemed to me that a minimum requirement for nomination was a basic knowledge of what Professor Edson had talked about in his lecture. Time went by

and I was giving a paper on gamma-curcumene to the Institute of Chemistry Conference which was held that year in Dunedin' when I saw Professor Edson come in and sit in the front row of the main Chemistry Lecture Theatre. The Chairman was Professor Briggs who had been my NZ examiner for my PhD. After I finished, Professor Edson asked Professor Briggs if he could make an announcement and he then informed the conference that I had been awarded the Nuffield Demonstratorship in Biochemistry. Well, clearly, there was just as much ignorance in South Africa about the Tricarboxylic Acid Cycle and they did not have anyone as close to metabolism as a PhD in natural product chemistry. I went off to Oxford and for the first of two times in my scientific career, I was ushered into a bare laboratory with the professor saying 'you have arrived at a very busy time - we are in the middle of the Michaelmas term'. When the term finished, Sir Rudolph Peters found me in the departmental library and he said 'Batt, you don't know anything about Biochemistry.' Well, of course I agreed. Then he put his proposals for my time at Oxford - 3 years. Firstly, since there was no Honours course in Biochemistry, I should do the Honours course in Human Physiology. He commiserated with me about having to become an undergraduate again but cheerfully claimed that we remain students for our whole lives. He said I should sit finals in 18 months time which was pushing things, considering it was a 3 year degree. I was put into Wadham College, given Denis Parsons as my tutor, and launched into becoming something a little closer to a biochemist. In fact, we had some very uninteresting lectures based on Baldwin's book and I think there may have been some biochemical questions in the final papers. Dr Midwinter, who was in one of the first classes I lectured to after I returned to NZ, claims that I was barely one step ahead of the class as I read the next chapter of Baldwin for my next lecture. The Oxford decision to make me an undergraduate again was not well received back home. That is an understatement. When I was well on my way to finals, Professor Edson arrived in England on leave and reached a compromise with Sir Rudolph which was, in essence, that I should finish the Honours degree and then do a DPhil degree with D.D.Woods - the discoverer of the mechanism by which sulphonamides worked.

At the beginning of 1952, I returned to NZ via the United States on a Carnegie Travel Grant and met some of the great names of Biochemistry. I was returning to a lectureship in the Department of Biochemistry at the Otago Medical School and for the second time I was ushered into an empty laboratory—well, not quite, there was a burette stand. I should have kept it. This time I was not being sent back to do yet more undergraduate courses, but after 10 years of study, I was no biochemist. These days we train a biochemist through to a PhD in 6-7 years—but we could be skimping. Biochemistry is the basic biological science and to be a well-trained biochemist it is necessary to have competence as a chemist and competence as a physiologist.

For 12 years, I taught 2nd and 3rd year classes of medical students and all levels of the science offerings. At the end of all that, I think I was beginning to know what I was talking about. Sir Rudolph was right - you never cease being a student. Before I left Otago to go to Massey, Sir Rudolph came to NZ and when I met him, he was keen to be reassured that what he had decided for me was right. I have always been pleased that I came into Biochemistry through that protracted pathway and it was certainly a help in establishing the Massey combined Department of Chemistry and Biochemistry after I was appointed to the Chair of Biochemistry in 1963. Being somewhat provocative, perhaps all NZ universities should have combined departments of chemistry and biochemistry - considering the very strong biological base to our economy. To head such departments, you would, ideally, want people with feet in the two camps of chemistry and modern experimental biology.

Secretion of Mucus Glycoproteins in the Digestive Tract and their Importance in Cytoprotection

A. M. Roberton

Department of Biochemistry, University of Auckland, Auckland

I believe I was Professor Batt's first B.Sc. honours student, and in 1961 carried out a research project in his laboratory studying metabolism of phenyl-substituted fatty acids in *Nocardia corallina*. Thinking back to the third year biochemistry class of 1960 I believe all of us in that class were stimulated by Dick Batt's enthusiasm. He also emphasised the importance of experimental research and the potential of Biochemistry. And in large part, Dick influenced me to pursue a career in Biochemistry, and encouraged me to take up an opportunity to do a D.Phil. at Oxford, rather than going farming.

Since those early days in Dick Batt's lab, my research efforts have followed interests in energy metabolism, microbial biochemistry, and in the last ten years the intriguing relationship between the mucus layer lining the digestive tract of the host animal, and the bacteria which live in and on the digestive tract. This has led in turn to studies on the mucus glycoprotein itself, and how its secretion is controlled, which is the subject of my talk today.

There is a layer of mucus covering the surface of the digestive tract from the mouth to the rectum. It is thought that this mucus layer protects the delicate underlying cells from damage by external agents. The mucus is believed to act in a number of ways which are summarised in Table 1. Incredibly we know very little about how mucus carries out these roles, or the factors which control mucus secretion to maintain this barrier function.

Protection from abrasive damage
Protection from dehydration
Selective filter
Protection from pH extremes
Protection from digestive proteinases
Factor in colonisation by pathogenic bacteria
Secretion during inflammatory response
Food source for some bacteria
Factor in disease - altered mucus glycoprotein

Table 1. Roles of mucus

The main structural constituents of the mucus layer are the mucus glycoproteins (Fig.1). These molecules have a huge molecular size. They are composed of approximately 15% protein and 85% carbohydrate: the carbohydrate is composed of oligosaccharide chains joined to the peptide chains at every 3rd or 4th amino acid, by an O-glycosidic linkage to serine or threonine. The oligosaccharides are so numerous that proteinases can not cleave the peptide chains. Subunits of mucus glycoprotein are linked together to give a long coiled thread. The individual subunits are thought to be linked by disulphide bonds. And near these disulphide bonds are stretches of naked peptide chain which can be cleaved by proteinases. So native mucus glycoproteins can be cleaved into subunits either by proteinases or by reducing agents such as mercaptoethanol.

It is not easy to study mucus secretion for many reasons, the main ones being difficulty of quantitation, and the inaccessibility of mucus-secreting tissue for sampling. Some of the experimental approaches that have been used are:

Electron microscopy
Measurements of mucus gel thickness

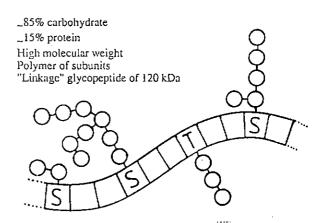


Fig. 1. Composition and characteristics of mucus glycoprotein. S = serine residue and T = threonine residue.

Studies on animals by sacrifice or intubation Studies on isolated mucus-secreting cells Tissue culture

Studies on cancer cell lines - e.g. T84 secretes mucus.

Many factors may control mucus secretion such as calcium, acetylcholine, prostaglandins, histamine and bacterial toxins. So far, progress in understanding the control of mucus secretion by these and possibly other factors has not been very satisfactory. In collaboration with Professor Cliff Tasman-Jones at Auckland, several colleagues in my laboratory have been developing a human model for studying control of mucus glycoprotein secretion. It is a live model, and thus gets over the problem of working with mucus secreting cells that have been separated from the normal background stimulation by neuro-transmitters and blood-borne hormones. Also it gets around the inaccessibility of the digestive tract for sampling purposes.

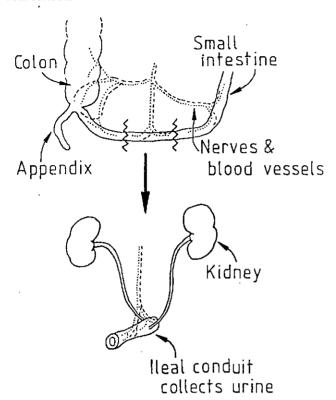
Patients with ileal conduits (Fig.2) have usually had bladder cancer, which necessitated surgical removal of the bladder. To give them a reservoir for holding urine, a section of small intestine is surgically relocated to form an artificial bladder, connected to the ureters and emptying into a bag strapped to the belly. The blood and nerve supplies to the ileal conduit are left intact. So the ileal conduit is an isolated pouch, and all the mucus secreted into the pouch is voided with the urine. Thus a 24 h collection of the urine contains the mucus secreted by the ileal conduit that day.

We have used these patients to study mucus secretion from small intestinal tissue. A very sensitive Elisa assay for human small intestinal mucus glycoprotein has been developed to measure the secreted mucus glycoprotein in the unne. This was developed by Howard Dalzell and Carol Harding in my laboratory. It measures down to fractions of a nanogram protein of mucus glycoprotein. Bandule Rabel, a Ph.D. student in my laboratory has recently shown that the mucus glycoprotein secreted in the ileal conduit urine is very high molecular weight and therefore undegraded.

Using the ilead conduit model we have measured control by drugs of mucus glycoprotein secretion in trials with patients. We tested the effect of 900 mg aspirin per day on mucus glycoprotein secretion. Since aspirin is a prostaglandin antagonist, we expected this drug to inhibit mucus glycoprotein

secretion in small intestinal tissue. In the experiments performed, in every case we found that aspirin caused an increase of 45-55% in secretion. We interpret these results as showing that aspirin causes a small and generalised irritation to the mucosa, and this causes an inflammatory response which stimulates mucus secretion.

Fig. 2. Translocation of a length of small intestine to form an ileal conduit.



Disadvantages:

- (i) Luminal nutrition affects structure
- (ii) Effects on secretion of luminal food versus urine
- (iii) Flora on ileal conduit surface different

Advantages:

- (i) Sampling easy. Can be done repetitively
- (ii) Use same subject repetitively without destruction
- (iii) Cells not deteriorating. Nerve and blood supply intact
- (iv) Self flushing
- (v) No contamination with other mucus
- (vi) No pancreatic protease digestion
- (vii) Can preserve collected mucus at 4°C, with azide &
 - (viii) No Elisa reactive material in normal urine
 - (ix) Patient is own control
- (x) Can characterise mucus glycoprotein
- (xi) Results on human subjects. Results reproducible

Table 2. Advantages and disadvantages of the ileal conduit

We have also seen a marked effect by the cortisol analogue drug, prednisone. This caused an increased secretion of mucus glycoprotein, and the response was dose-dependent. At the highest doses, mucus glycoprotein secretion was stimulated about 200% above baseline secretion rates.

These preliminary results suggest that the ileal conduit model will be a useful and effective means of studying the factors which control mucus glycoprotein secretion. The advantages and disadvantages of this novel method for studying mucus glycoprotein secretion are outlined in Table 2.

In conclusion I'd like to wish Professor Batt well in his retirement; but in particular I'd like to thank him for his enthusiasm and his encouragement which helped to launch many of us here today on a career in experimental biochemistry.

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Biochemistry of Developing Nitrogen-Fixing Nodules in Legumes

John G. Robertson

Biotechnology Division, DSIR, Palmerston North

It is a pleasure to express my thanks to Professor Batt for the role he has played in establishing my work in science. As supervisor of both my Masterate and Doctoral theses on the organism *Nocardia corallina*, he developed my understanding of the scientific method especially as applied to biochemical research. He also encouraged my interest in research on biological membranes which led to postdoctoral studies in the USA on the electron microscopy of biological membranes and biochemical studies of membrane proteins.

These foundations in biochemistry and cell biology led to a research programme on biological membranes in legume nodules as part of a programme on biological nitrogen fixation, in DSIR in the early 1970s. Emphasis was being placed on developing pastures and grain crops requiring a low-input of fertilizer nitrogen using the Legume-Rhizobiumsymbiotic system of nitrogen-fixation. The aimwasto understand and control the processes of inoculation, infection and development of nitrogen-fixing bacteroids (the symbiotic form of rhizobium) in legume nodules, in order to achieve maximum levels of plant production. Work in various laboratories throughout the world has had an enormous impact on increasing pasture production and legume grain yields in various nations, over a period of the last fifty years.

My own research centred on the function and biogenesis of the peribacteroid membrane in legume nodules. The peribacteroid membrane is the membrane which surrounds the bacteroids in nodules and separates the bacteria from the plant cytoplasm. The initial objectives of the work were to isolate and purify this membrane and to define what key metabolites of the nitrogen fixation process might be passing across the membrane. Lupin was chosen as the model plant because it could be grown under highly controlled conditions in a manner which provided uniform development of nodules of up to 100 g/ harvest. Such amounts of tissue in turn allowed for the isolation of subfractions of nodule tissue with characteristic properties and enzymic activities relative to a particular stage of the development of the nitrogen-fixing process (Figure 1). During the course of these studies it was discovered that much could be learned about what metabolites were being transported across the peribacteroid membrane by following the pattern of induction of key enzymes in the plant and bacteroid cytoplasmic fractions (Robertson et al., 1975). Identification of the ammonia assimilation enzymes glutamine synthetase and glutamate synthase, as the primary enzymes involved in ammonia assimilation in the plant cytoplasm, led to a series of studies which resulted in the establishment of the pathway for assimilation of ammonia in legume nodules (Robertson and Farnden, 1980).

Purification of the peribacteroid membrane was achieved by first isolating the bacteroids still surrounded by the membrane, which was then released by osmotic shock and purified by centrifugation on a sucrose gradient (Robertson *et al.*, 1978). Isolation and purification of various proteins and membrane fractions from nodules opened up the opportunity for raising antibodies to these components and for using these antibodies to determine the location of the component within the nodules, using immunogold labelling of thin sections. Using this technique it was possible to localise the site of leghaemoglobin, over which there had been much debate, within the ground cytoplasm of infected cells of pea nodules (Robertson *et al.*,

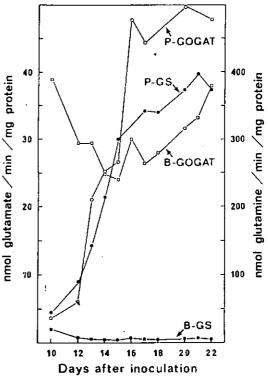


Fig. 1. Induction of glutamine synthetase and glutamate synthase during nodule development in lupin. Nodules were collected from plants harvested at intervals following inoculation and the plant and bacteroid soluble protein fractions were prepared and assayed. (•) glutamine synthetase activity in the bacteroid soluble protein fraction (B-GS). (a) glutamate synthase activity in the bacteroid soluble protein fraction (B-GOGAT). (•) glutamine synthetase activity in the plant soluble protein fraction (P-GS). (o) glutamate synthase activity in the plant soluble protein fraction (P-GOGAT).

1984). Further studies using monoclonal antibodies to the peribacteroid membrane, gave support to the hypothesis that the peribacteroid membrane was a modified form of plasma membrane and that biosynthesis of the peribacteroid membrane and the plant plasma membrane, involved the endomembrane system of membrane biogenesis (Brewin *et al.*, 1985).

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Continued on page 12

Free Radical Reactions of The Blood

Christine C. Winterbourn

Department of Pathology, Christchurch School of Medicine, Christchurch Hospital, Christchurch

The title for my presentation to this symposium marking Dick Batt's retirement serves to link what I am doing now to what I did for my PhD under Dick's supervision. Blood is an obvious connection, since then and now my work is with blood cells. Free radicals, because my main interest now is in the involvement of free radical reactions in pathological processes, and maybe because they hold the key to answering the question I was posed for my PhD, "Why does the red cell die?"

I came to Massey after completing a degree in chemistry at Auckland and a year at Auckland Hospital measuring lipoprotein turnover. I went to see Dick looking for a job. He had money and a project and offered me both. So there I was as a green biochemist confronted with the problem of how the red cell grows old.

The mature red cell contains no nucleus and no protein synthetic ability. Its function in life is to maintain its haemoglobin in a form that can transport oxygen around the body. This it does for 120 days (in humans) and then it dies. Dick had a theory that bacteria, and perhaps red cells, have an endogenous energy reserve the consumption of which leads to cell death. It quickly became apparent with red cells that this was not the case. Nevertheless, I learned to separate young from old cells (on the basis of density), and found out how the membrane lipid composition changes with age and the extent to which the phospholipids turnover. I did not solve the aging mystery; but at least I have the consolation that until very recently nobody else knew much about it either.

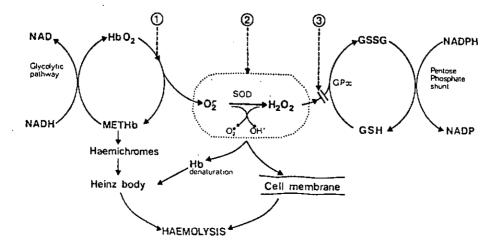
After two years in Canada, I returned to Christchurch where I began working with Robin Carrell, again on red cells but this time not on the membranes but on the haemoglobin inside. Our interest was in a class of mutant haemoglobins, the unstable haemoglobins, that precipitate inside the cells to give inclusions called Heinz bodies. We showed that the inclusions consisted of oxidized denatured haemoglobin, and proceeded to study the mechanism of oxidation. This is where my interest in free radicals began, since it had recently been shown that when normal oxyhaemoglobin oxidizes to methaemoglobin, the superoxide radical (0 $_{\rm 2}$) is also produced (Fig. 1.). We found that the unstable haemoglobins underwent this reaction more

rapidly than normal. We also showed that whereas normal methaemoglobin is quite stable, the unstable methaemoglobins undergo a further conformational change to give a haemichrome, the formthat precipitates as Heinz bodies. Thus the unstable haemoglobins stress the red cell in two ways, by giving an increased flux of superoxide, and readily precipitating in their ferric form.

At this time we expected superoxide to be highly damaging to cell constituents and were disappointed, therefore, when we looked at its reactions with haemoglobin. It reacted only with the haemiron, either oxidizing the ferrous or reducing the ferric form. This is just one example leading to the general conclusion that superoxide itself is relatively benign. Nevertheless superoxide and its dismutation product, hydrogen peroxide, provide oxidizing power, and the continuous production of these species could be a source of oxidant stress. If the red cell is considered to be in a state of balance of oxidant production and removal by antioxidant enzymes (as in Fig. 1), then excess production, as occurs with an unstable haemoglobin, could upset this balance and affect the function of the cell.

Through the study of haemoglobin I developed a more general interest in free radical biology and shifted some of my attention to neutrophils, the white blood cells that provide our primary defence against invading micro-organisms. When neutrophils ingest their targets, an oxidase on the cell membrane is activated to produce a burst of superoxide into the phagosomes, at the expense of intracellular NADPH. Other cell constituents stored within granules are released at the same time. Although some of these are bactericidal, the burst of superoxide is essential for efficient killing. Cells that lack the oxidase cannot kill some organisms, and individuals with this defect generally die of infection at a young age. Again it appears that it is not superoxide itself that is toxic. Rather, the best candidate is hypochlorous acid (chlorine bleach) produced from hydrogen peroxide and chloride by myeloperoxidase, a haem enzyme that is released by the stimulated neutrophils. A major emphasis of our current research is on how hypochlorite is produced by the cells and what it does to its targets.

Figure 1: Production and removal of activated oxygen species in the red cell. Positions 1 and 2 are where pathological conditions can lead to increased production and position 3, decreased removal of oxidants. Abbreviations: SOD, superoxide dismutase; GSH, GSSG, reduced and oxidized glutathione; OH hydroxyl radical; 0₂*, other activated oxygen species.



Neutrophils can also damage host tissue in inflammatory diseases like arthritis. The cells respond not only to ingestible particles, but also to a variety of inflammatory mediators such as complement components and immune complexes, releasing superoxide and granule constituents into their surroundings. Others have shown resultant killing or alterations in function of host cells, and Margret Vissers in my laboratory has measured damage to extracellular matrix proteins. Using isolated glomerular basement membrane, we showed that immune complexes cause neutrophils to adhere and gradually digest the membrane collagen. The cells also release superoxide at sites of adherence. Digestion of the membrane is due to proteinases released from the neutrophils, but oxidants (primarily hypochlorite) modify the substrate to enhance its proteolytic susceptibility. Our recent studies indicate that they also modify functional properties of the matrix. The role of oxidants is complex, however, as through lack of discrimination they can also inactivate the proteinases - one example of the kamakasi behaviour of neutrophils. One useful purpose for defining the role of neutrophils and their oxidants in inflammatory tissue injury is to provide a rationale for planning interventionist treatment. For example, there are reports documenting the anti-inflammatory properties of superoxide dismutase, which is available in N.Z. as a drug for veterinary use. We have found that a number of more conventional anti-inflammatory drugs inhibit neutrophil function in our basement membrane model.

With regard to free radical reactions of the blood, I have described how haemoglobin oxidation is a constant source of superoxide that is enhanced in some pathologies. Neutrophils produce large amounts of superoxide which is converted to hydrogen peroxide and hypochlorous acid and these contribute to bacterial killing and inflammatory tissue damage. And the link with red cell aging? As far as is currently known, when red cells grow old they develop a surface antigen that makes them immunologically recognisable. As a result they are phagocytosed, although not by neutrophils, but by macrophages resident in the liver and spleen. The origin of the altered antigenicity is not clear. However, one of the more seriously considered proposals is that continuous oxidant production throughout its lifespan finally overwhelms the defences of the cell, so that there is a recognisable oxidative change to a membrane constituent that targets it for destruction.

The Acetaldehyde Story

K. E. Crow

Department of Chemistry and Biochemistry Massey University, Palmerston North

In the late 1960's, Professor Batt initiated a research programme on the metabolism of alcohol in mammalian systems. My involvement in this area began in 1972, with a Ph.D. research project involving purification of sheep liver aldehyde dehydrogenase. At this time acetaldehyde (the first product of ethanol oxidation in the liver - see Fig. 1) and its metabolism appeared to be one of the most exciting areas of alcohol-related research. There had been very little work carried out on mammalian aldehyde dehydrogenases. Also, it had been proposed that either acetaldehyde itself, or other endogenous aldehydes accumulating as a result of preferential acetaldehyde metabolism by aldehyde dehydrogenase, could react with biogenic amines to form addictive compounds. Thus it was thought that the cause of alcoholism might be revealed by studies on acetaldehyde metabolism.

The first part of my Ph.D project involved purification and physical characterisation of cytosolic aldehyde dehydrogenase, but kinetic studies on this enzyme were to be carried out by another PhD student, Alistair MacGibbon and the mitochondrial enzyme was being purified and characterised by Dr T.M. Kitson. For the second part of my PhD, therefore, I moved from characterisation of cytosolic aldehyde dehydrogenase to the use of this enzyme in measurement of acetaldehyde.

Another researcher with the group, Ken Couchman, was measuring blood acetaldehyde concentrations in human volunteers after alcohol ingestion, and was using a gas chromatographic method. The range of blood acetaldehyde values quoted in the literature up to this time was huge (from 1.0 - 681 M) and we decided that the use of two independent methods to cross-check our values would be advisable.

An enzymic assay for acetaldehyde was developed, based on the fluorescence produced when acetaldehyde and NAD+ were converted to acetate and NADH by cytoplasmic aldehyde dehydrogenase. Previous work suggested that yeast aldehyde dehydrogenase, although commercially available, was not ideal for use in this assay as a rapid end-point was not reached, but we later discovered that the yeast enzyme did work well provided pyrophosphate buffer, and not Tris buffer, was used.

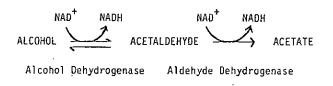


Fig. 1. The pathway of alcohol metabolism.

To avoid high background fluorescence from blood samples, acetaldehyde was diffused from filter paper strips into the assay mix, and with this method we measured concentrations of 10-60 M acetaldehyde in blood from volunteers who had consumed alcohol. This agreed with concentrations found by gas chromatography but, by both methods, plasma acetaldehyde concentrations were found to be much lower than those for whole blood. This observation became the subject of another PhD research project, carried out by Allan Stowell. Allan developed an automated enzymic assay for acetaldehyde, and showed that much of the acetaldehyde measured in whole blood in our earlier studies was an artefact, being produced from ethanol during deproteinisation of the blood with perchloric acid. With appropriate correction for the artefact, Allan found concentrations of about 5°M acetaldehyde in blood from volunteers.

During subsequent studies at the Alko laboratories in Helsinki, however, Allan discovered that the artefact correction method he had used was not completely reliable, and he developed another method in which blood was pre-treated with semicarbazide to stabilise the acetaldehyde, plasma was separated, and acetaldehyde concentrations were measured using head space gas liquid chromatography. With this method, peripheral venous acetaldehyde concentrations were shown to be in the range of 0-1,4M for normal individuals given a moderate ethanol dose. A number of groups, using the semicarbazide method, or other methods which did not produce artefactual

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Box 37-583 Parnell. Telephone (09) 770-392 Fax (09) 398-514 acetaldehyde, also confirmed that peripheral venous acetaldehyde concentrations were close to zero in normal individuals.

With the finding of very low peripheral blood acetaldehyde concentrations, interest in the idea of acetaldehyde binding reactions waned to some extent, particularly as research in the area had produced little progress. An earlier observation of elevated blood acetaldehyde concentrations in alcoholics had been cast into doubt as it became apparent that the methods used would have caused significant generation of artefactual acetaldehyde.

More recently, however, interest in the area of acetaldehyde binding reactions has redeveloped. Results using reliable methods for acetaldehyde measurement have shown that in some circumstances alcholics do have elevated acetaldehyde concentrations in peripheral blood. It has also been recognised that hepatic acetaldehyde concentrations may be considerably higher than those in peripheral blood, since the liver is the site where acetaldehyde forms, and once it enters the blood it is subject to metabolism by aldehyde dehydrogen ase in the red blood cells. In rats, in vivo, we have demonstrated that acetaldehyde concentrations in hepatic venous blood (blood directly leaving the liver) are very variable and range from 10-160-M. In Scandinavia, it has been possible to do the same type of experiments in humans, and hepatic venous acetaldehyde concentrations were shown to be, usually, much higher than those in peripheral blood. The concentrations ranged from 1-70_MM in non-alcoholics, and 1-160_MM in alcoholics.

Recent progress in the area of acetaldehyde binding reactions is summarised in a volume entitled 'Aldehyde Adducts in Alcoholism'. There is a range of compounds with which acetaldehyde has been shown to form adducts under physiological conditions, for example: (1) Hemoglobin - acetaldehydemodified hemoglobin has been suggested as a marker for assessing chronic alcohol intake; (2) Hepatic proteins - acetaldehyde binding to these has been suggested as a possible initiator of liver damage due to alcohol; (3) Opioid peptides - still undiscovered when the original acetaldehyde-amine binding theory was proposed, these have now been shown to bind acetaldehyde with a consequent decrease in their receptor binding ability; (4) Biogenic amines - work still continues on confirming the original theory of binding to biogenic amines, and assessment of the possible changes in neurotransmission and other functions resulting from the modified compounds is an important research area.

Thus, during the 18 years since the original proposal that binding products of acetaldehyde could lead to alcohol addiction, it has become accepted that peripheral blood, and therefore probably also brain, acetaldehyde concentrations are much lower than initially believed. The theory has been kept alive by the observation of significant binding reactions under physiological conditions, by reliable findings of elevated acetaldehyde in some alcoholics, and by evidence of higher acetaldehyde concentrations in liver than in peripheral blood. It remains to be demonstrated that there is a definite link between any acetaldehyde binding reaction and the onset of alcohol addiction, but this still appears to be a valid possibility. Overall, Professor Batt's decision of 18 years ago that acetaldehyde metabolism was the field to enter proved to be a very worth-while move.

Continued from page 9

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Metabolism of Lactic Acids Bacteria (20 years on)

T. D. Thomas

New Zealand Dairy Research Institute, Palmerston North

Like several other contributors to this symposium my links with Dick Batt go back to the time when he first came to Massey University as Professor of Biochemistry. After doing an M.Sc. in organic chemistry at Otago University, I took a job as an industrial chemist in Auckland. While there my attention was drawn to an advertisement for a job at the New Zealand Dairy Research Institute. The job was to carry out fundamental research on the metabolism of lactic streptococci which are used as starter bacteria in cheesemaking. In spite of having almost no background in either microbiology or biochemistry, I was given the job and told that I could work on anything at all related to this subject. So I asked Dick Batt, who had just come to Massey if my research in this area could be the basis for a PhD. The answer was yes, no problem, even with my background. So along with Christine Winterbourn, Grattan Roughan and John Robertson away we went - 24 years ago. Christine had her red blood cells, Grattan his chloroplasts, John his soil bugs and I had lactic streptococci. We all started with the same question - why do cells die (in biochemical terms)?

I remember that Dick was keen on manometry so I started with respiration studies and, after what seemed like an age, I discovered that lactic streptococci hardly respire at all. In fact they did not seem to do anything that my colleagues' organisms could do. I thought what useless uninteresting bugs they were. After a slow start we decided to look at the endogenous metabolism of lactic streptococci in relation to their survival. Thankfully these studies turned out to be both interesting and productive. One of the lasting reminders of my association with Dick is the series of papers on endogenous metabolism of lactic streptococci that we published in the Journal of General Microbiology. I was fortunate to be in the U.K. doing post-doctoral studies at Cambridge on membrane biogenesis when our papers on survival came out, because they generated a lot of interest there.

Since that time most of my research has concerned the two key metabolic properties of lactic streptococci - proteolysis, whereby milk protein is converted to amino acids to supply nitrogen for starter growth, and fermentation of lactose to lactic acid which supplies the energy needed for growth.

The lactic streptococci are the comerstone of our cheese and casein industries. For economic manufacture of these products the milk fermentations must be rapid and off-flavours must be absent in the product since it is a food. Rapid fermentation means that the starter must grow fast in milk. This requires an efficient proteolytic system since the concentration of low melecular weight nitrogen in milk limits growth. The starter must also have systems for rapid lactose fermentation. To avoid off-flavour in products such as cheese, the proteolytic system must minimise the level of bitter peptides generated from casein and the lactose fermentation must produce minimal products other than lactic acid.

Our research at D.R.I. has helped establish some of the important details of the proteolytic system. Fig. 1 shows how lactic streptococci use casein in milk for the synthesis of their own protein. Proteinase activity, located at the surface of the cell wall, allows hydrolysis of large molecules such as casein. Some of the resulting oligopeptides, possibly after further degradation by cell wall peptidases, are small enough to diffuse into the cell wall to the cell membrane. We know that size restrictions only allow transport of peptides with up to 4 to 5 amino acid residues through the cell membrane. Several distinct systems exist for peptide transport. Once inside the cell, peptides are confronted with a range of peptidases. These complete the obligatory hydrolysis of peptides to free amino acids which are then available for protein biosynthesis inside the cell. This proteolytic system is very efficient. It operates fast enough for lactic streptococci to reproduce in about 60 minutes in milk at 30°C, yet their proteolytic activity is very low compared to other bacteria. Indeed one of the beauties of these starters is their rapid growth without excessive proteolysis.

Turning to the other vital starter property, which is lactose fermentation, it is here that most of our research has been focussed. In the lactic streptococci, lactose enters the cell via a specific phosphotransferase (PTS) system and appears

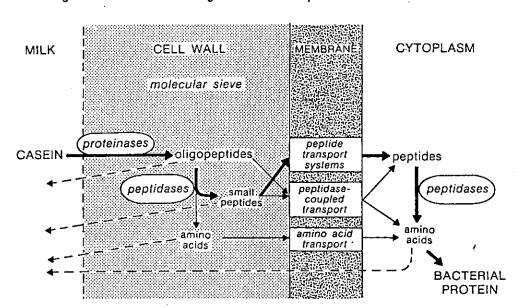


Fig. 1. Utilisation of casein for growth of lactic streptococci in milk.

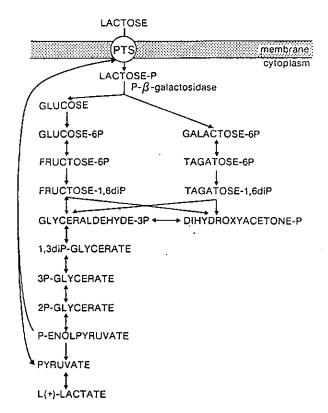


Fig. 2. Pathway of lactose metabolism in lactic streptococci.

inside as lactose phosphate (Fig. 2). This is cleaved to glucose and galactose phosphate. The glucose goes through the glycolytic pathway and the galactose phosphate through the tagatose 6-phosphate pathway, the two pathways joining at the triose phosphates. Lactose breakdown is fast, with cells producing their own weight of lactic acid in just 10 minutes. Our work has established many of the factors involved in the regulation of these pathways.

We have focussed on the last two enzymes in this process -pyruvate kinase and lactate dehydrogenase. Pyruvate kinase is pivotal because its substrate is also required for lactose transport into the cell. A single allosteric pyruvate kinase is present which is markedly activated by all eight intermediates from the hexose phosphates through to the triose phosphates. This control mechanism regulates the intracellular phosphoenoi pyruvate (PEP) concentration and provides a coupling between lactose transport and subsequent metabolism. When lactose is exhausted, the pyruvate kinase activators are quickly depleted inside the cell. This results in a massive build up of PEP and its two immediate precursors leaving the cell primed for sugar transport via the PTS once the sugar supply is restored.

The last enzyme, lactate dehydrogenase determines the fate of pyruvate in lactic streptococci. We require pyruvate to go to lactate with no side reaction products. As Fig. 3 (top line) shows that is what does happen when lactose or glucose is present in excess. However, these organisms have the hidden potential for much more than that. By limiting glucose or lactose supply, or by using free galactose, we get formate, acetate and ethanol instead of lactate. By supplying oxygen as well we found a homoacetic fermentation - an amazing turnaround from our homolatic situation.

It was clearly important to understand the regulation of this apparently latent potential since it would play havoc with the flavour of fermented milk products if it was expressed. The lactate dehydrogenase in lactic streptococci is completely dependent for its activity on either fructose 1, 6-bisphosphate or tagatose 1,6 bisphosphate. With lactose excess the levels of these Intermediates inside the cell are high so that lactate dehydrogenase is fully activated (Fig. 4). At the same time the level of triose phosphates is also high and these intermediates are potent inhibitors of a second enzyme that can compete for

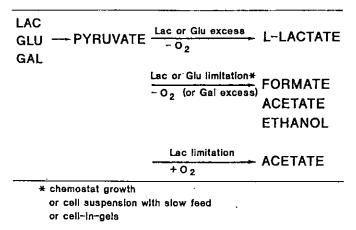
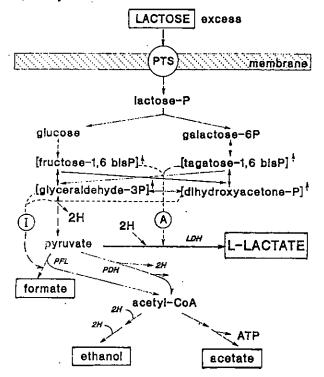


Fig. 3. Possible fates of pyruvate produced during carbohydrate fermentation by lactic streptococci.

available pyruvate, namely pyruvate-formate lyase (PFL in Fig. 4). With limiting supply of lactose the intracellular levels of the glycolytic intermediates drop. Without sufficient activator (fructose or tagatose bisphosphate) the activity of lactate dehydrogenase is low while with low levels of triose phosphates the inhibition of PFL is lifted. The result is a very unattractive heterolactic fermentation with formate, acetate and ethanol formed as well as lactate. With lactose limiting and oxygen present, the highly oxygen-sensitive PFL is knocked out and the pyruvate dehydrogenase complex (PDH in fig. 4) takes over. The result is a very undesirable homoacetic fermentation. We now know that this nasty deviant behaviour is not normally expressed in milk fermentations mainly because the supply of lactose does not limit its rate of fermentation and oxygen is largely excluded.

In my PhD studies with Professor Batt I was initially frustated because these lactic streptococci did not seem to do anything metabolically. I now realise that therein lies the secret of their success. It is in fact their comparative inactivity which makes these organisms so important to the dairy industry. What they have to do, they do extremely well with their beautifully designed systems for proteolysis and lactose fermentation. They do not really do anything else - and we would not want them as starters in the dairy industry if they did.

Fig. 4. Regulation of lactose metabolism under conditions of lactose excess. Lactate is the sole product. A: enzyme activation, I: enzyme inhibition.



Unravelling Glyceroliped Biosynthesis in Plants; A Personal Saga

Grattan Roughan

Division of Horticulture and Processing, DSIR, Auckland

At about the same time as Dick Batt was due to move from Otago University to the Chair at Massey, I was recruited by the relatively new Plant Physiology Divison (PPD) of DSIR at Palmerston North. My new employer was delighted to have me to study toward a PhD in biochemistry under Professor Batt and in 1965, after one false start, it was decided that the topic of my PhD thesis would be "Lipid metabolism in plants". In 1965 very little was known about plant lipid biochemistry. The usual phospholipids found in animal tissues had earlier been identified in leaves and were considered to reside predominantly outside the chloroplasts. In addition, monogalactosyl diglyceride (MGD), digalactosyl diglyceride, sulphoquinovosyl diacylglycerol and phosphatidylglycerol were recognised as characteristic of green plant parts and were, therefore, probably involved somehow with chloroplasts.

The fatty acid constituents of these plant lipids were mostly polyunsaturated with linolenate (18:3)being by far the major fatty acid of green leaves. Newly developed methods, permitting confident analyses of the positional distributions of fatty acids within glycerolipids, introduced even greater complexity into the picture. The same glycerolipid could have different distributions of fatty acids depending upon its source, and different phospholipids from the same source could have different fatty acid distributions. Although completely absent from some plants (18:3-plants), hexadecatrienoic acid (16:3) was found to be localised at the sn-2 position of MGD in other plants (16:3-plants) (Fig. 1.). It was a confusing scene.

By the early 1960s it was known that isotopically labelled CO₂, acetate and inorganic phosphate could all be incorporated into the lipids of detached leaves. Isolated chloroplasts, however, appeared to incorporate labelled acetate only into cleate (18:1) and paimitate (16:0), and not into polyunsaturated fatty acids. The site and mechanism of desaturation thus remained a major problem.

Having spent a good part of my PhD work developing methods for routinely separating all of the glycerolipids from small samples of leaf, we were poised to investigate the turnover of individual glycerolipids within leaves. It became evident in preliminary experiments that, whilst incorporation of acetate into long-chain fatty acids was very rapid, the subsequent turnover of glycerolipids was relatively slow and that the leaves would have to be kept alive for several days. It would therefore be inappropriate to work with detached leaves. In the back garden of our home was a pumpkin patch. There were huge leaves on the vines. If some way could be found to apply labelled acetate evenly over the surface of a leaf, and if that acetate was incorporated into cellular glycerolipids, then by repeatedly sub-sampling that leaf we should be able to perform a pseudo pulse-chase experiment lasting several days. It turned out to be an astonishingly successful experiment which established that phosphatidylcholine (PC) was intimately involved in the desaturation of 18:1 to linoleate (18:2) and 18:3. It was also notable that fatty acids originally esterified to PC were subsequently transferred to MGD. Being unconstrained by preconceived ideas, I had readily accepted the simplest interpretation of the data which was that 18:1 esterified to PC, rather than to CoA, was the true substrate for the desaturation of 18:1 to 18:2 and possibly of 18:2 to 18:3 in leaves. This explained the inability of isolated chloroplasts to desaturate 18:1 since fatty acid incorporation into PC occurred outside the

chloroplast in the endoplasmic reticulum. However, the idea was given, at best, an indifferent reception. Another difficulty in most people's minds must have been the idea of fatty acids being synthesised from acetate in chloroplasts then being transported to the endoplasmic reticulum and finally making their way back to the chloroplast to be incorporated into galactolipids. It seemed too bizarre altogether, particularly as chloroplast autonomy was quite fashionable at the time.

Fig. 1. Monogalactosyl diacylglycerol (MGD), quantitatively the most important glycerolipid in leaves. In eucaryotic MGD, both R1 and R2 are linolenate (18:3) whereas in procaryotic MGD, R1 is linolenate and R2 is hexadecatrienoate (16:3).

When, after a long break, I resumed work on plant lipid metabolism at PPD in collaboration with Dr Roger Slack, we confirmed the pivotal role for PC in cleate desaturation for developing maize leaves. Other evidence strongly suggested that 18:2 was transferred from PC to MGD where it was further desaturated to 18:3. Using expanding pea leaves, the labelled PC was shown to be localised primarily within the microsomal (endoplasmic reticulum) fraction of the cell, and a virtual quantitative movement of label from microsomal 18:1-PC to chloroplast 18:3-MGD over 48 hr was demonstrated in spinach leaves. We subsequently showed that the whole diacylglycerol (DAG) moiety of PC was being utilised for MGD synthesis.

The most influential labs in plant lipid metabolism, those of Douce at Grenoble and of Heinz at Cologne, were not sympathetic to our ideas. Douce contended that the chloroplast envelope was capable of synthesising all of the MGD required by the developing organelle and could, therefore, see no need to involve a second pathway in the e.r. for generating MGD precursors. Heinz protested, correctly, that in his experiments, the different distributions of fatty acids within PC and MGD were incompatible with the former being the precursor of the latter. These differences were eventually resolved when Heinz visited our lab in 1982 and we discovered the genetic difference between 16:3 and 18:3 plants (Fig. 2). Chloroplasts from the latter, such as maize and pumpkin, were deficient in phosphatidate phosphatase activity and therefore could not

Fig. 2. Illuminated chloroplasts isolated from 16:3 plants incorporate labelled acetate into fatty acids, phosphatidate, and diacylglycerol. Fatty acids are moved out of chloroplasts as CoA esters. Chloroplasts from 18:3 plants cannot dephosphorylate the phosphatidate.

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generate the appropriate DAG which would result in the synthesis of MGD containing 16:3. Heinz then realised that our differences of opinion in the past arose simply because we worked with different types of plants. He had worked mainly with a 16:3 plant, which synthesised the bulk of its MGD without the intervention of PC, whereas we had done almost all of our work on 18:3-plants where all of the MGD is derived from PC (Fig 3).

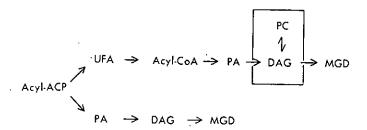


Fig. 3. Flow of newly-synthesised acyl chains through the eucaryotic pathway (upper) into eucaryotic MGD, and procaryotic pathway (lower) into procaryotic MGD. The latter is synthesised entirely within the chloroplast. Desaturation occurs after fatty acids are incorporated into PC and MGD.

Ideas as to how the fatty acid compositions of plant lipids might be controlled suddenly began to gel. We tested the idea that fatty acid distributions might be impressed upon glycerolipids at the very earliest stages of their synthesis, i.e. at the acylation of G3P. In chloroplasts isolated from spinach and pea, the soluble G3P acyltransferase showed a marked preference for, but was not absolutely specific for, 18:1-ACP whereas the 1-acyl G3P acyltransferase was absolutely specific for 16:0-ACP. Hence, procaryotic glycerolipids synthesised wholly within chloroplasts could initially have either 18:1 or 16:0 at sn-1 but could have 16:0 alone at sn-2. In the endoplasmic reticulum, on the other hand, it could be inferred from careful positional analyses of PC fatty acids that the membrane-bound G3P acyltransferase similarly transferred either 16:0 or 18:1 from CoA to the sn-1 position of G3P, but that the 1-acyl G3P acyltransferase was absolutely specific for 18:1-CoA. Only C18 unsaturated fatty acids could occur at the sn-2 position of eucaryotic glycerolipids synthesised in the endoplasmic reticulum. Hence, the chloroplast and the endoplasmic reticulum would synthesise characteristically different molecular species of phosphatidate from which the typical membrane glycerolipids would be formed. The MGD of 16:3plants could be synthesised from DAG originating both in the chloroplast and in the endoplasmic reticulum and was comprised of a mixture of two molecular types, whereas the MGD of 18:3-plants was synthesised from DAG originating in the endoplasmic reticulum alone and was almost entirely of a single molecular type (see Fig. 1).

This then was the genesis of our "two pathway hypothesis" of glycerolipid synthesis in plants. The idea of the two pathways ("procaryotic" and "eucaryotic") was first introduced in the Biochemical Journal in 1980, but we had not yet recognised a relationship between pathway of synthesis and glycerolipid structure. By 1982 we were prepared to speculate how different molecular species of glycerolipid would be synthesised through the two pathways, and in 1984 we presented a more detailed explanation of the hypothesis. Since then we have plugged away and gradually wom down the opposition so that in 1988 I can confidently claim that almost all of the active researchers in the field of plant lipids are interpreting their results in relation to the two pathways and that rapid progress is now being made on several fronts.

Reference

Roughan, P.G. and Slack, C.R. (1984) Glycerolipid synthesis in leaves. Trends in Biochemical Sciences, 9 (1), 383-386.

1989 Branch Chairs (Part 2)

Manawatu - J.Lee

Dr Julian Lee received his early education at Reporoa College in the Bay of Plenty. He graduated from Massey University in 1974 with M.Sc (Hons) in chemistry and completed his PhD at Massey in 1977. Studies for his thesis dealt with the chemistry and biochemistry of metal accumulation in plants. He was particularly concerned with nickel



uptake by unusual endemic ultramafic plants from New Caledonia. Several publications arose from this work, describing aspects of the nickel chemistry in these intriguing and newly discovered nickel accumulators.

Julian then spent two years on a Canadian National Research Council fellowship in Ottawa at the Geological Survey and at Carlton University. He worked on trace metal speciation in surface waters relating to aspects of environmental and geochemical dispersion of metals.

In 1980 Julian joined DSIR's Biotechnology Division (then Applied Biochemistry Division) in Palmerston North. His current research interests are in analytical chemistry and instrumentation, and in trace element metabolism in plants and animals. He is studying zinc metabolism in animal and mamalian systems and in the interaction of environmental factors in nutrition.

Julian has been a member of NZIC since 1980 and active on the Manawatu Branch Committee since 1981. For the past 4 years he has been Branch Treasurer. His interests outside work include road and cross-country running, horticulture and wine.

Waikato - N. Pritchard



Nath Pritchard is a Station Chemist at Electricorp's Huntly Power Station, where he has provided chemical services for that high pressure power utility for the past five years. Nath has been associated with water treatment for over twenty-five years: he has spent over half that time in power utilities in the UK, South Africa and New Zealand, three years on a copper mine in Zambia, and the remaining time with two major international water treatment engineering companies based in England.

Nath trained as a power station chemist with the Central Electricity Generating Board, near Manchester, then studied for professional qualifications at Salford Technical College and John Dalton College of Technology (Manchester). He is a Chartered Chemist, a Member of the Royal Society of Chemistry and a Member of the New Zealand Institute of Chemistry, His chemical interests are in inorganic and especially water chemistry, and he has a particular interest in the work of Michel Pourbaix.

A Welshman by birth, he has more than just a passing interest in rugby: although he is past the playing stage, he is secretary of a local club.

Otago - Stan Winter

Stan has been employed by the Southland Co-op Phosphate Co. Ltd in Invercargill since leaving schoolin 1966. He was Works Chemist from 1975 and appointed Technical Manager in



1983. He became a member of the Institute of Chemistry in 1981 and was elected a Fellow in 1986.

The main thrust of his work in recent years has been based on research into the interactions in the Soil-Plant-Animal-Climate-Fertiliser System. The resulting data base is being used to define and remedy problems for farmers relating to soil fertility and physics. A service which they find very valuable.

Stan also operates an X-Ray Fluorescence Laboratory performing consulting work for farmers on plant and animal tissues and for industrial users, particularly geochemical analyses. He serves on several Specialist Technical Committees and represents the Fertiliser Industry on one Government Interdepartmental Committee.

Stan is married with two children and enjoys photography, tramping, music and resting in the sun.

Branch News

Manawatu

The 1988 Branch AGM, was held at the Manawatu Polytechnic on 19 November. A barbecue followed the formal meeting, topped off by the Chairmans address.

Dr Julian Lee, a research scientist at the Biotechnology Divison of DSIR, was elected chairman for the coming year. Our secretary and treasurer are Drs Alastair MacGibbon and Mark Brimble respectively. Rev. Ted Fletcher is the Hawkes Bay representative and the Taranaki representative is Mr Dave Wills. Other committee members elected were Drs David Husbands, David Newstead, Darryl Rowan, Cecil Johnson (branch editor), Joyce Waters and Alan Furness (immediate past chairman) and Ms Cathy McArdle (student representative). Dr MacGibbon is our Council Dele-

Dr Alan Furness, in his Chair-

man's Address entitled "Images of Chemistry", concentrated his talk on students' perceptions of chemistry. Alan said that for many students chemistry is thought of as being difficult, boring and no fun. However, after his talk Alan gave us a visual display of chemicals which made pretty colours or loud noises. Judicious use of these demonstrations can make the subject more enjoyable. The attendance at this meeting was small but enthusiastic.

BOP - Sub branch

In the first year of operation we have had 3 existing speakers and a visit by the President. Speakers were Prof. Richard Neidleim (Heidelbeg), Prof. David Doddrell (Queensland, Aust) and Prof Roger Powell (Madison, USA). Roger spoke at the start of November on chemical modification of wood and research being done in the US, China, Sweden and UK. Although much

of the chemistry was simple in concept, the challenges were great, e.g. use of solid liquid phase transfer catalysis, chemical diffusion problems, and byproduct removal from wood. The Japanese have one commercial enterprise producing per-acetylated wood (highly stable and decorative) for use in loudspeaker enclosures and musical instruments. Radiatar pine wood is proving itself to be an excellent biopolymer for chemical modification at FRI, now part of Powell's international team researching the modified wood topic.

Overall, the BOP sub branch committee (comprising initially Terry Lomax, Nicola Deacon and Robert Franich, now joined by Roger Medor) felt the inaugural year went well, much though, of the credit to the BOP members from Rotorua, Taupo. Whakatane, Kawerau, Tuaranga and Mt. Maunganui who have supported meetings. (There is always room for improvement)! Meetings have attracted 18-33 people, in the very comfortable and amiable surroundings of the FRI Rimu Room.

Corosion Technology A Short Certificate Course in Two Parts

The Australasian Corrosion Association (Inc.), Auckland Division, in association with Auckland Technical Institute will present a short Certificate Level course in corrosion science, corrosion engineering and corrosion control, to be conducted in two parts, in 1989, at ATI. The course is part-time only.

The course is aimed at the Technologist level, and although there is no rigid entry qualification, a good knowledge of basic science and engineering principles will be assumed. This is the sixth time the course has been offered at ATI. The course aims to provide a basic understanding of corrosion principles and corrosion mitigation practices.

To obtain a Brochure which contains enrolment information, or to enquire further about the course, please contact: Keith Ufton, ATI, tel. (9) 773570 or Les Boulton, DSIR, tel (9) 34116.

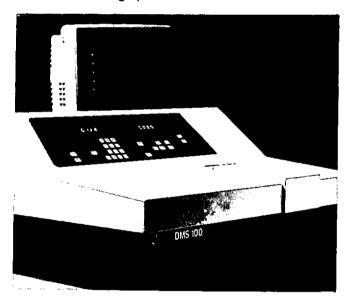
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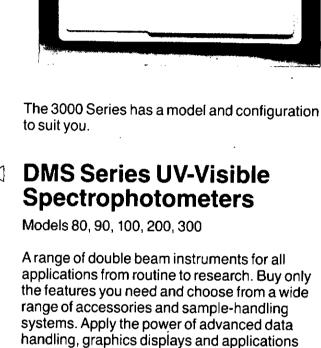
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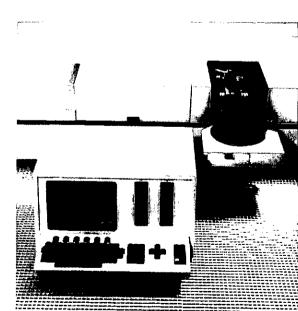


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Labsupply Pierce the sole NZ Agent carries the enzyme and kits ex stock Auckland for immediate delivery.

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New Brochure From HP Describes Enzyme Kinetics And Reaction Model-Fitting

The brochure (Publication 5956-4182) describes how more reliable results can be obtained from fewer experiments. It shows, for example, that all the data required for optimizing complete experimental procedures can be obtained from a few initial measurements.

The system featured in the brochure has three main components: a software package dedicated solely to enzyme kinetics; the latest HP diode-array UV/vis ChemStation. The system automates the study of enzyme mechanisms from data acquisition, through evaluation and model-fitting, to the printing of results.

For determining rate data, the brochure shows how users can save time and minimize sample consumption. Up to six assays can be carried out in parallel. The brochure lists four curve-fit models for calculating rate data; alternatively, users can enter their own equations.

For reaction model-fitting, the brochure describes how a rate-data table can be compiled using data from previous experiments, or from other sources. Nine enzyme-reaction models are listed: they include models with single substrate with or without inhibition, and with two substrates.

Again, users may enter their own equations. Other examples include showing how the fit of the experimental data with the models can be interpreted from graphical and numerical reports.

This brochure is available through Medtec Products Limited.

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New DNA Amplification System

Perkin-Elmer Cetus have released a new amplification technique allowing microgram quantities of DNA to be obtained from picogram amounts of starting material. This patented procedure called the Polymerase Chain Reaction (PCR) technique amplilies, in vitro, a target DNA sequence at least 100,000-fold in just hours instead of days. Developed by Cetus scientists, PCR represents a major breakthrough in molecular biology.

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The system comprises:

DNATHERMAL CYCLER-for automation of the PCR technique and GENEAMP KIT.

The thermal cycler is a sophisticated yet easy-to-use programmable temperature cycling instrument. Just place the sample in the instrument, program the cycle profile and walk away.

All the necessary reagents, optimised for PCR including the thermostable enzyme Taq Polymerase are contained within the Geneamp kit. Each kit will perform 100 individual amplifications quickly and efficiently.

The above system is available through SCI-MED, the exclusive distributor for Perkin-Elmer Cetus products in New Zealand.

For further information please circle no. 3 on reader reply card.

Autophoresis

Molecular Dynamics, who recently introduced a Densitometer capable of scanning large gels in less than 3 minutes and manipulating the data in many ways, has just announced a new technique called Autophoresis. Using laser optics, High Resolution Dynamic Imaging is an optical technique that permits viewing of bands directly as they move through electrophoresis gels. Results can be recorded on film at any time without stopping electrophoretic separation. HRDI does not use stains and does not require radioactive or fluorescent

For further information please circle no. 4 on reader reply card.

Sequence Analysis Software For DNA, RNA & Proteins

Restriction enzyme mapping with calculation and graphic representation of circular restriction maps is one of the features of the latest version of the Micro Genie® Sequence Analysis software available from Beckman Instruments. Also new to the system is the ability to predict and plot protein hydrophobicity. Other features include 'shotgun' DNA sequencing where sequences can be entered from the keyboard or directly from a gel with the Gel Mate™ 1000 Sonic Digitzer, homology search and alignment facility, sequence translation, transformation and much more.

For further information please circle no. 5 on reader reply

Laminar Flow Equipment

Gelman Sciences Pty Ltd have specialised in the design and manufacture of what is believed to be the most comprehensive range available world wide of laminar flow equipment and safety cabinets using HEPA (high efficiency particulate air) filters or alternatively ULPA (ultra low particulate air) assessed at about 0.1 micron retention.

Typical Cabinets and systems have been available in New Zealand for the past 25 years, and this experience is reflected in product performance, design and assurance.

The Trace Metal Analytic Cabinets are designed to not only provide a particle free work zone, but for the cabinet construction to be resistant to typical acid

For further information please circle no. 6 on reader reply card.

New Literature Available From Watson Victor

DuPont

DuPont have prepared an international guide to the Sorvall range of Centrifuges. Sorvall centrifuges enjoy an outstanding reputation for high quality and reliability among research and clinical laboratories worldwide.

New Brunswick

The ML-4100 is a high-powered Multi-Loop Process Control System for Fermentation and Cell Culture. Full specifications and product information are included in this new brochure.

Schott

We have available from Schott

a new leaflet outlining the applications of the KL1500 Cold-Light source. 'Cold-Light source' is the term used to describe a light source in which the thermal component of the energy from the lampis filtered out and the visible light is passed through a flexible fibre-optic light guide to the area requiring illumination.

Drott

For those customers involved in the manufacture of beer, mineral water, fruit-juice, wine, cider and other beverages, the Drott Haze Meter TMB1 is most suitable for the routine measurement of hazen. We have just received a supply of literature on this new product.

Radiometer

Radiometer have produced a booklet entitled 'The Operational Lifetime of Radiometer Glass and reference Electrodes', and suggested maintenance. This publication is intended to answer customers' questions about the lifetime of electrodes and the best method of maintaining them.

Developments at John Morris

As of 1 April 1989, John Morris Scientific Ltd become the exclusive agents for Metrohm, and New Brunswick Scientific. This will further complement the recent addition of the Hermle range of centrifuges.

The Metrohm range covers ph, titration, ion chromatography, and

polarography.

New Brunswick Scientific cover fermentors, cell and culture growth, media preparation, incubators and shakers.

Product Seminars

A series of hands on seminars on pH, titrators, Carl Fischer, and ion chromatography will be held in May, followed in October by a similar series on chromatography and polarography.

Final dates and venues to be confirmed.

For further details contact: John Morris Scientific Ltd, 1/29 Ashfield St, Glenfield. PO Box 6348, Wellesley Street, Auckland 1. Phone (09) 444-5836, Fax (09) 444-0974.

THERMOPLASTIC ENGINEERING LTD.

In some laboratories dangerous situations seldom arise. It would be true to say however that few do not have to deal with fumes either irritant or unpleasant. Many installations have to be sufficiently versatile to handle a range of applications covering a fume spectrum ranging from harmless irritants to highly lethal fumes.

It was to cover situations involving a wide variety of hazards that Thermoplastic Engineering Ltd developed their Laminair Safety Fumecupboard. Like all Thermoplastic fumecupboards it is fully ducted to the open air. The company, dedicated to providing optimum safe working conditions for the operator and other laboratory personnel regard this as an essential principle. It is certainly in line with modern thinking which does not favour ductless fumecupboards which recirculate air for general use.

The Laminair Safety fumecupboard provides an example of the meticulous attention to detail which Thermoplastic bring to all their products. It has been tested by DSIR and incorporates the following features:

· an aerodynamic base beam

and side columns to reduce air turbulance within the fumecupboard.

- a laminar slotted baffle for greater fume containment (removable for ease of cleaning)
- freeflow roof outlet which reduces air resistance and improves fan performance
- fully closing door for greater operator safety
- Corotex flooring system incorporating a low heat transmission underbase and chemical heat resistant top surface.

In common with other types, Thermoplastic Laminair Safety fumecupboards are constructed of high quality calender pressed and polished PVC (Options are 316 Stainless Steel or Polypropylene). They are fire resistant with a vertical sliding clear front door of 6mm acrylic sheet. Armour plated glass or polycarbonate can be fitted if required. Cabinet roofs are sloped for more efficient airflow and to prevent dead spots. The roof outlet is a 245mm PVC spigot socketed to allow easy connection to further ducting. Aesthetically pleasing, the cupboard is available in rich italic cream with brown door trim or in light industrial grey.

Thermoplastic Engineering have probably the widest range of fumecupboards available in New Zealand. In addition to the Laminair Safety they manufacture the Laminair Perchloric for use in high hazard situations or those involving Perchloric Acid. It incorporates the features of the Safety ranges plus double hand welded corner joints for ease of cleaning. A wall mounted hand shower simplifies washing down. The worktop of tinted armour toughened glass is raised above a shallow sink to act as a safety drain should acids be spilled.

The long standing and highly popular Safety fumecupboard designed for less hazardous purposes is nevertheless a highly sophisticated product. It incorporates the Corotex system, considered one of the best of its kind and providing a low heat transmission underbase and chemical and heat resistant top surface.

Econocare, as its name implies is an economy model but has most of the features of other ranges. It has a single opening inlet design in which the air velocity increases. The vertical side columns are sloped to assist in

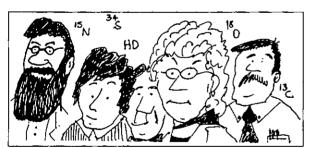
reducing turbulence within the cupboard.

A heat and chemical resistant flooring system is provided as well as heat protection on the side and rear walls.

All Thermoplastic fumecupboardranges have four standard models. Uniformly of a height of 1220mm and a front to back width of 760mm they vary in length from 915mm to 1830mm. They can also be provided in 316 Stainless Steel and Polypropylene. Recognising too that standard types and sizes do not meet all situations, special dimensions are available on request.

Very much part of the Thermoplastic fully ducted systems are fume handling fans. Centrifugal fans for interior and exterior installation in conjunction with all types of fumecupboards and axial flow fans specially made for use with the Econocare model TEFC3 with a short ducting run. And as sole New Zealand agents for Brinkmann GmbH of Frankfurt, Germany, the company can supply a whole range of extra high performance fans to cover a wide variety of needs.

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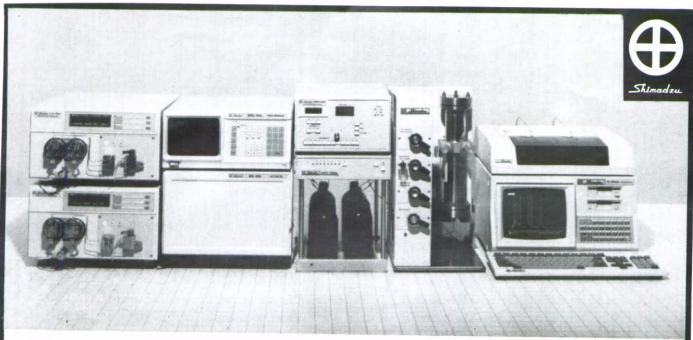
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