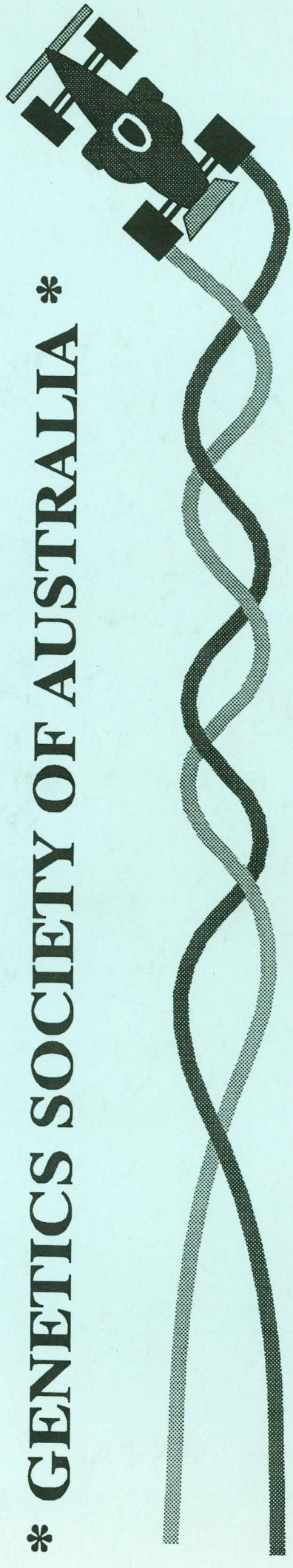
CHRIS MORAN

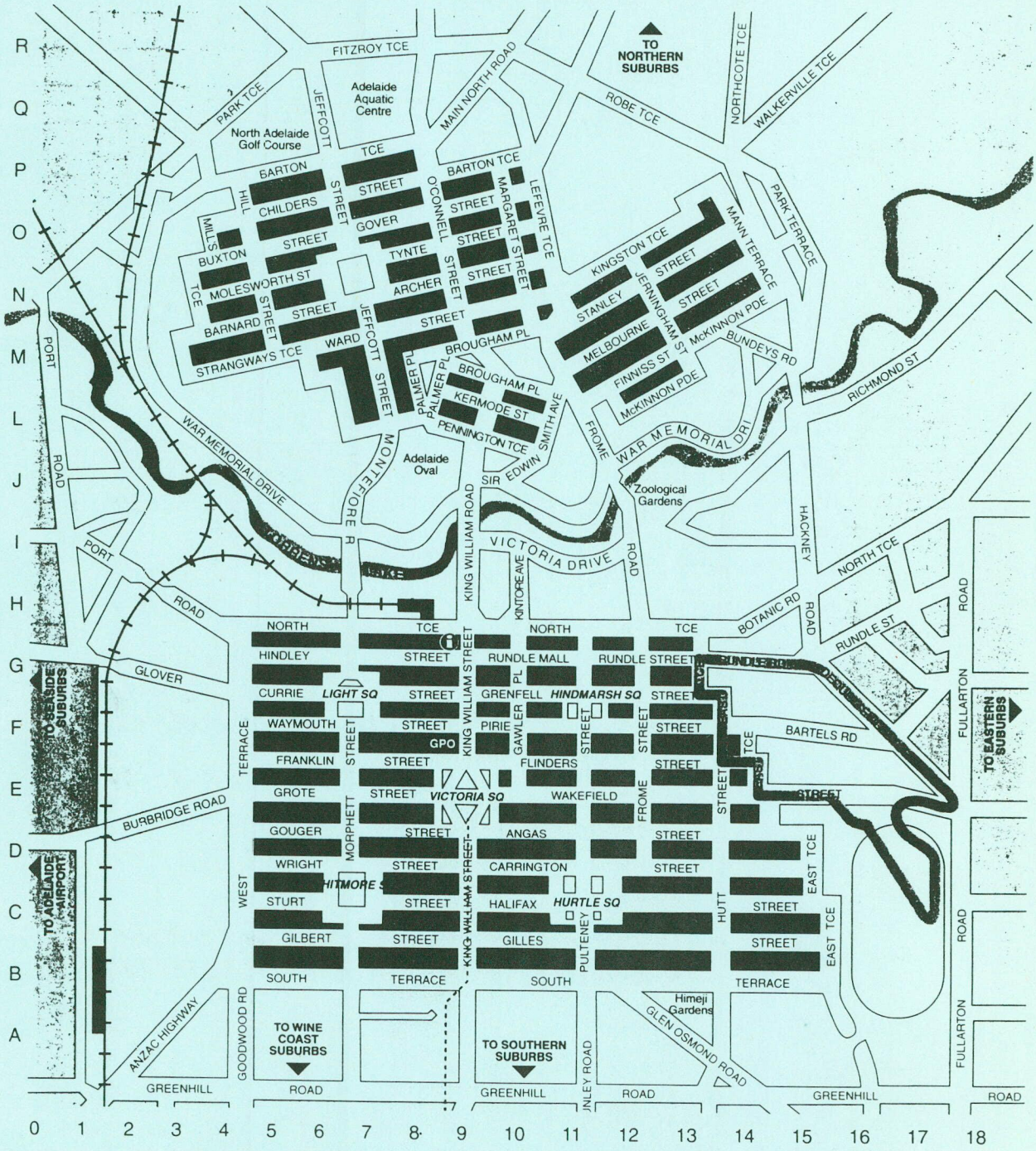


GSA 40th ANNUAL CONFERENCE

ADELAIDE
SOUTH AUSTRALIA
JULY 7TH-10TH
1993

* GENETICS SOCIETY OF AUSTRALIA *

MAP OF ADELAIDE AND NORTH ADELAIDE



Services	Grid Ref.
Central Bus Terminal	E 7
Interstate Rail Terminal	B 1
Glenelg Tram Terminal	E 9
Metropolitan Bus & Information Office	G 9
Metropolitan Railway Station	H 8
St Mark's College (near Anglican Cathedral)	L 9
Medical School, Univ of Adelaide	I/H 12.5

Facilities	Grid Ref.
Tourism South Australia Travel Centre	H 9
Adelaide Casino	H 8
Adelaide Convention Centre	H 8
Grand Prix circuit	

This map does not show the location of all visitor attractions.
Please refer to the Adelaide & Suburbs Map and the Adelaide Visitors Guide available from Tourism South Australia.

GENETICS SOCIETY OF AUSTRALIA

40TH ANNUAL CONFERENCE AND WORKSHOP

GENERAL ORGANISATION

The GSA local committee welcomes you to Adelaide for the 40th Annual Meeting.

A mixer will be held in St. Mark's College Senior Common Room on the evening of Wednesday 7th July from 7.00 pm. Drinks and finger foods will be provided.

The 40th GSA meeting will be very busy and varied. The joint meeting with ASBMB will be held in the Adelaide Convention Centre, North Terrace on July 8th finishing in the afternoon. This will be followed by a workshop and a session in the Medical School, Frome Road, devoted to presentations associated with applications for the Smith-White Travel Awards. July 8th will therefore contain new and exciting ventures for the GSA in addition to the joint meeting with ASBMB. July 9th and 10th will be in the regular GSA format. Members are reminded that the *Gene Mapping Workshop* is being organised by Cynthia Bottema of the Waite Agricultural Research Institute. This meeting is scheduled to start immediately after our conference but there is no formal connection between the two society meetings this year.

REGISTRATION

Registration will be from 5.00-8.00 pm on July 7th at St Mark's College. Delegates arriving later than this will have an opportunity to register at times and venues specified in the programme. Entrance to **all** sessions will be by official GSA name/affiliation badge.

Delegates of the meeting who are not GSA members are required to pay an increased registration charge.

WORKSHOP

A workshop will feature a number of computer software packages which are useful in teaching Genetics and in developing computer images and slides for lectures.

MEALS

The Annual Conference Dinner will be held at St. Mark's College on Friday 9th July and the cost will include bottled wines and mineral water. Other meals may be obtained in the Adelaide and North Adelaide restaurants, at the University Staff Club or at the Students' Union Bistro. Delegates are requested to wear their official GSA conference name badge at these venues. A barbecue will be held at Woodstock Winery, McLaren Vale at the end of the conference. Buses will transport delegates to the winery and return to the city calling at the airport at 5.30 pm in time for most interstate flights.

Members are reminded that the *Australian Gene Mapping Workshop* starts in the Florey Lecture Theatre at 2.00 pm. Those who wish to attend the AGMW may prefer to have a barbecue with the Gene Mappers at a more local venue which is yet to be finalised by the organisers.

LOCAL COMMITTEE:

David Catcheside, Joan Kelly, Carolyn Leach,
Rob Saint, Rob Shroff and Jeremy Timmis.

LIFE SCIENCE

Special Issue: DNA Research

Millipore NEWS

Now You Can Eliminate Radioactivity from Southern and Northern Blotting Procedures

The Millipore PolarPlex™ Blotting Kit is the sensitive and fast way to eliminate radioactivity from Southern and Northern blotting procedures. Based on the proven chemiluminescent detection chemistry of the Plex® DNA Sequencing Kits, PolarPlex Kits offer the following features:

Speed - Only 40 minutes are required for the entire detection procedure. Exposure times of 1 - 10 minutes are typical.

Sensitivity - 0.1 pg of DNA can be detected in a slot-blot format and single copy genes can be identified from as little as 1 µg of human genomic DNA.

Multiple Exposures - Light is emitted at a constant rate for days, so you can perform multiple exposures to optimize signal intensity. And re-exposure at a future date is achieved by simply adding more Lumigen™-PPD reagent.

Simple Reprobing - If you want to search the same membrane for a different set of fragments, simply strip the membrane and rehybridize with a different probe.

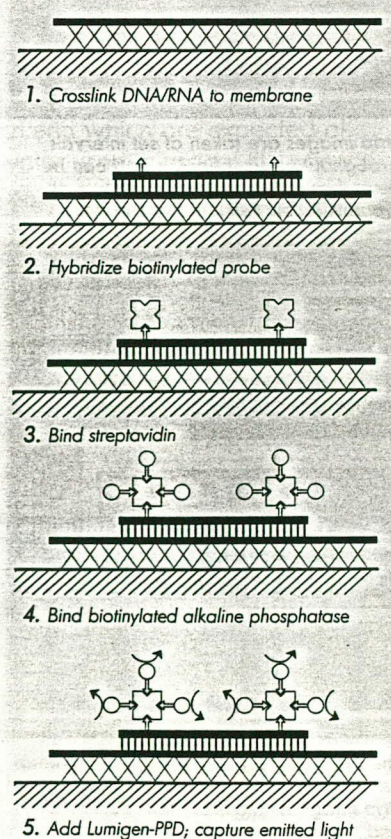
Stability - Biotinylated probes and reagents are stable for extended periods.

To obtain full information on PolarPlex Kits, please circle No. 1 on the reply-card.

■

The Chemiluminescent Detection Procedure

Biotin is incorporated into hybridization probes through a random primer reaction. The biotinylated probes are hybridized to the target nucleic acids immobilized on the membrane. A streptavidin/biotin interaction is used to anchor the enzyme (alkaline phosphatase) to the hybrid. The chemiluminescent substrate, Lumigen-PPD, is added in the final step. The substrate is destabilized when dephosphorylated by the enzyme and, subsequently, decomposes, emitting light in the process. The light is captured on X-ray film for analysis. The key to the high sensitivity of this chemistry is the specific activity of the labelled probe and the selection of reaction conditions.



1. *Crosslink DNA/RNA to membrane*
2. *Hybridize biotinylated probe*
3. *Bind streptavidin*
4. *Bind biotinylated alkaline phosphatase*
5. *Add Lumigen-PPD; capture emitted light on X-ray film*

New Instruments and Systems for Electrophoresis and Gel Analysis

The Millipore BioImage® range of systems has been designed to improve the accuracy and speed of electrophoresis analysis. The systems are based on the new Sun Sparc® computers. These computers are ten to twenty times more powerful than PC's - yet the cost is becoming increasingly competitive. The model 60 is the laboratory workhorse. It provides highly accurate analysis of 1D gels, RFLP's, DNA Sequencing and 2D experiments. The model 110 is designed for advanced research applications and high sample throughput. The systems are highly modular allowing expansion as the needs of the laboratory change.

Accuracy and ease-of-use were primary design considerations, and Millipore offers a complete range of peripheral devices such as high resolution film readers and laser printers to compliment the applications.

Restriction Fragment Length Polymorphisms

The analysis of RFLP samples presents a complex analytical problem. The BioImage system is calibrated using optical density wedges and then scans the sample with a high resolution CCD camera. A complete image is captured in less than one second.

The computer compensates for lane distortion; the bands are automatically located and quantified. The quantification of the bands is also automatic and uses the whole band as the basis for measurement. Measurement of the band background is also taken into account. Dedicated data basing and reporting software provides the user with band and lane match reports. Asymmetric band migration properties are measured and highly accurate matching criteria are reported.

The RFLP data base can compare thousands of gels in a few seconds and report on band similarities and frequencies. Pattern recognition software allows the user to program and compare non-standard patterns quickly and easily.

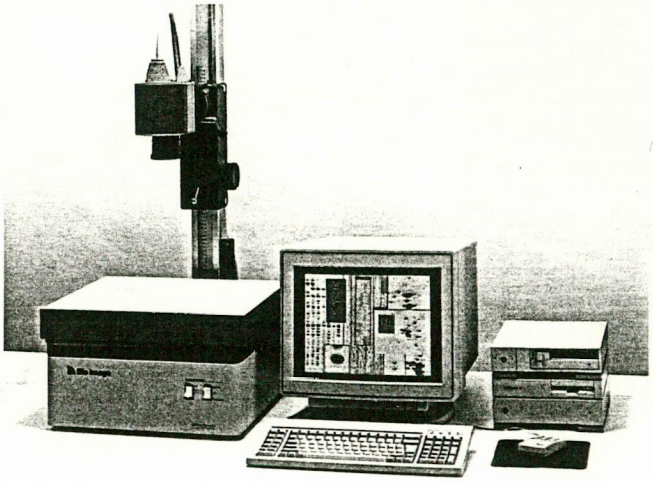
DNA Sequencing Gel Analysis

Highly advanced algorithms incorporated in the DNA Sequencing software help to achieve both high accuracy and high speed analysis. Films of any length are fed into a flat bed film scanner and digitized in a few seconds. All or part of the image is displayed and interactive zoom, pan and colour coding keys allow the user to examine the image in detail.

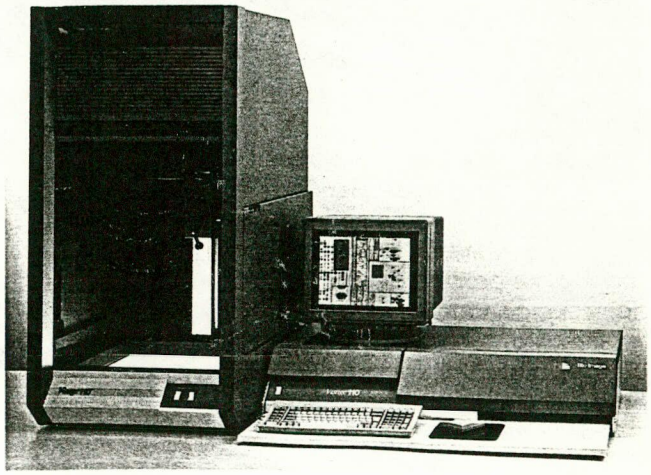
By showing the computer the general area of analysis, a series of automatic procedures are applied to locate the lane edges. The computer follows the lanes vertically to compensate for any "smiling" or distortion. When all of the lanes have been defined, the system analyses the horizontal distortions to correct for geometric inconsistencies. The sequence is then read automatically. Using a series of self-learning algorithms, the computer indicates ambiguous and unambiguous bands and the operator can correct and verify the analysis before reporting the data to the screen, the printer or on-line data bases.

The whole analytical process takes only a few seconds; the amount of editing needed is dependent upon the quality of the gel. The overall accuracy is extremely high, even in areas of compression and band competition.

BioImage model 60 electrophoresis analysis system



BioImage model 110 electrophoresis analysis system



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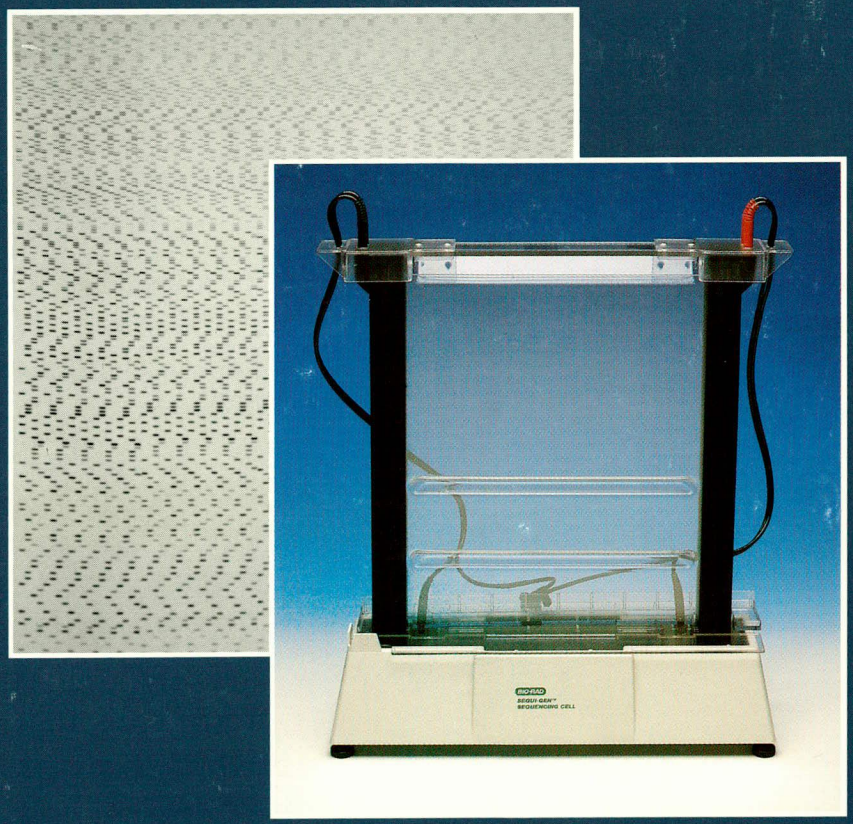
Correct at 6.6.93

Sequi-Gen® II Nucleic Acid Sequencing System



Designed for High Resolution and Now Improved for Increased Durability and Ease of Use

BIO-RAD

The Best Gets Better

High Resolution

New features make the Sequi-Gen cell even more user friendly—without compromising resolution or safety. The Sequi-Gen cell remains the best high-resolution sequencing unit available. The key is our patented integral plate chamber (IPC). The IPC uses the upper buffer chamber as a heat sink for maintaining uniform heat distribution over the entire gel plate—it eliminates smile patterns.

Multiple Uses

The long, thin gel format of the Sequi-Gen cell allows good separation of DNA and RNA fragments that differ by only a single nucleotide. In addition to sequencing, this feature is useful for the following applications:

- *PCR*-amplified tandem repeat studies*
- *DNA footprinting*
- *RNase protection assays*
- *S1 nuclease mapping*
- *Primer extension analysis*
- *Mutation screening*

PCR amplification of human polymorphic CA repeat sequence electrophoresed on the Sequi-Gen cell. Photo is courtesy of Catriona MacGeoch, David Kelsell, and Nigel Spurr of the Imperial Cancer Research Fund, U.K.

Easy to Use

It is simple to put the Sequi-Gen cell together or to take it apart. The one piece clamps snap into place quickly and the stabilizer bar holds the IPC snugly in the universal base.

Space Saving

You asked for it, you got it: The new smaller and lighter 16 x 48 cm universal base saves valuable bench space. It also includes a convenient spout for draining buffer.

Safe

Chemically tempered glass plates resist breaking or chipping. Interlocking safety covers maximize operator protection. The silicone sealed upper buffer chamber eliminates the need for a gasket. New molded cables add durability and increase safety.

*PCR is covered by U.S. patent number 4,683,202, issued to Cetus Corporation.

Ordering Information

Catalog Number	Description
165-3600	**Sequi-Gen Cell,** 21 x 40 cm
165-3601	**Sequi-Gen Cell,** 21 x 50 cm
165-3604	**Sequi-Gen Cell,** 38 x 50 cm
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GENETICS SOCIETY OF AUSTRALIA
40TH ANNUAL CONFERENCE AND WORKSHOP

JOINT MEETING WITH THE AUSTRALIAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY

PROGRAMME: 7TH - 10TH JULY 1993

Wednesday July 7th

5.00-8.00 pm GSA Registration, St Mark's College, 46 Pennington Terrace, North Adelaide
7.00-10.00 pm GSA Mixer, St Mark's College, 46 Pennington Terrace, North Adelaide

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Thursday July 8th: Joint Meeting of ASBMB and GSA

Venue: Adelaide Convention Centre, North Terrace.

N.B. Abstracts of papers (indicated in parentheses [eg. SYM-13-1]) are provided in the ASBMB book of abstracts supplied at registration

8.30-9.00 am GSA Registration (8.45-9.00 am: ASBMB Student Poster Awards)

ENTRANCE TO SESSIONS WILL BE STRICTLY BY OFFICIAL GSA OR ASBMB NAME BADGE ONLY

****

9.00-10.00 am **Plenary Lecture (PLE-8):** Chair: P Nagley
John Shoffner: **Mitochondrial DNA mutations and degenerative diseases: an overview**

****

10.00-10.30 am Morning tea/coffee & exhibition

****

10.30-12.00 noon **Thematic symposia (concurrent sessions)**

Human molecular genetics Chair: R Cotton
Grant Sutherland (SYM-13-1): Mapping human chromosome 16
Julian Mercer (SYM-13-2): Identification of the gene for Menke's disease by positional cloning
Brandon Wainwright (SYM-13-3): Gene knockout mouse for the study of cystic fibrosis

Environmental regulation of plant gene expression Chair: W Taylor
Elaine Tobin (SYM-14-1): Genes negatively regulated by phytochrome action in *Lemna*
Masumi Robertson (SYM-14-2): Regulation of gene expression by dehydration stress and hormones
Alan Neale (SYM-14-3): Molecular approaches to cloning genes conferring dessication tolerance in resurrection plants
Rudi Dolfeus (SYM-14-4): Characterisation of *Adh* promoter elements involved in environmental stress responses

Lipid metabolism and heart disease Chair: N Fidge
Noel Fidge (SYM-15-1): The role of receptors in the metabolism of high density lipoproteins
K-A Rye (SYM-15-2): The relationship between the composition, structure and function of reconstituted HDL
Alana Mitchell (SYM-15-3): HDL metabolism - the role of hepatic lipase
Roger Dean (SYM-15-4): Lipoprotein metabolism - the effect of oxidation

Thursday July 8th: Joint Meeting of ASBMB and GSA (continued)

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12.00-1.00 pm Lunch

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1.00-2.30 pm **Thematic symposia (concurrent sessions)**

Manipulation of plant development and physiology Chair: G Fincher

Don Grierson (SYM-17-1): Possible mechanisms for the inhibition of plant gene expression with sense and antisense genes

David Smyth (SYM-17-2): Genes which make cauliflowers from *Arabidopsis*

J Varghese (SYM-17-3): Crystal structures of two related beta glucanases with distinct substrate specificities

Abed Chaudhury (SYM-17-4): *Ampl* - a mutant with high cytokinin levels and altered embryonic pattern, faster vegetative growth, constituitive photomorphogenesis and precocious flowering

Molecular biology of mitochondria: disease and aging Chair: P Nagley

John Shoffner (SYM-18-1): Mitochondrial DNA mutation associated with Alzheimer's and Parkinson's disease

Phillip Nagley (SYM-18-2): Mitochondria, bioenergy diseases and human aging: molecular biology and cellular bioenergetics

Roger Dean (SYM-18-3): Mitochondria and protein oxidation in aging

Gene targeting Chairman: L Williams

Ian Lyons (SYM-19-1): A gene targeting approach to the study of early murine development: disruption of a homeobox gene expressed in early heart progenitors

Anna Michalska (SYM-19-2): Targeted disruption of the metallothionein I and II genes in the mouse

Brandon Wainwright (SYM-19-3): Targeted modification of the cystic fibrosis transmembrane conductance regulator gene in ES cells by homologous recombination

Kerry Fowler (SYM-19-4): *Wa-1* and *wa-2*: mouse mutants with dysfunctional TGF alpha and EGFR loci

Intracellular targeting and protein transport Chair: G Both

M Clarke (SYM-20-1): Recycling of modified cathepsin D molecules to the ER occures from a compartment which is separate from the compartment containing phosphotransferase

Keith Stanley (SYM-20-2) Targeting and trafficking signals of intracellular membrane proteins

David James (SYM-20-3): Intracellular sorting signals of the insulin-regulatable glucose transporter

Paul Luzio (SYM-20-4): Transport of proteins from endosomes to lysosomes

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2.30-3.30 pm **Plenary Lecture (PLE-9):** Chair: Tony Howells

Spiros Artavanis-Tsakonas (Joint GSA/ASBMB invited speaker):
The involvement of the Notch group proteins in Drosophila in cell fate choices

3.30-3.45 pm Closing ceremony of the joint ASBMB/GSA meeting

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Genetics Society of Australia Annual Meeting (continued)

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PLEASE VISIT THE POSTER AND TRADE DISPLAYS

4.30-6.00 pm **Set up posters:** Room S126, Medical School North
Sustaining members set up trade displays

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5.00-6.30 pm **GSA Workshop:** Stirling Theatre, Medical School South
Computer software for research and teaching
Computers will be available for individual use until 10.00 pm in the Science Faculty Suite (Mawson Laboratories) University of Adelaide

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8.00-10.00 pm **Smith-White Student Travel Award Applicants**
(Chair: Judy Ford)
Abstracts: A(1-6)

Talks:

8.00-8.20 *Lars S Jermiin, Ross Crozier:* Patterns of evolutionary change in insect mtDNA AT content

8.20--8.40 *Ross Howden, Chris Cobbett:* Cadmium sensitive mutants of *Arabidopsis thaliana*

8.40-9.00 *B Patterson, P Wigley, R Saint:* Regulation of the *string* gene during embryonic cell proliferation

9.00-9.20 *Darryl Reed, John Gibson:* TATA box and amino acid mutations in the *sn*-glycerol-3-phosphate gene in *Drosophila melanogaster*

Poster Presentations:

Andrew Lonie, Stanley Robert, Richard D'Andrea, Robert Saint: The *Polycomblike* gene of *Drosophila melanogaster* is required for the correct expression of the homeotic genes of the Antennapedia and Bithorax complexes

Lynn Jones, Helena Richardson, Robert Saint: Genomic and genetic characterization of the cyclin E gene of *Drosophila melanogaster*

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Friday July 9th

Venue: Florey Theatre, Medical School North, University of Adelaide

8.50-9.00 am **Welcome to The University of Adelaide:**
Gavin Brown, Deputy Vice-Chancellor (Research), Vice-Chancellor Designate, University of Adelaide

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Friday July 9th (continued)

9.00-10.00 am **Plenary Lecture:** Chair: Joan Kelly
Abstract: B1

Lorna Casselton: **Homeotic genes and self/non-self recognition in the mushroom *Coprinus***

****

10.00-11.00 am **Concurrent Sessions:** Medical School South

Evolution: Stirling Theatre (Chair: Doug Anderson)
Abstracts: C(1-3)

10.00-10.20 *Charles Robin, Kerrie Medveczky, John Oakeshott, Robyn Russell*: Evolution of an esterase gene cluster implicated in organophosphate resistance in insects (1) Organisation and sequence of the cluster in *Drosophila melenogaster*

10.20-10.40 *Leon Court, John Oakeshott, Robyn Russell*: Evolution of an esterase gene cluster implicated in organophosphate resistance in insects (2) Correlation of the cloned genes with biochemical phenotypes in *Drosophila melanogaster*

10.40-11.00 *Richard D Newcomb, Robyn Russell, John Oakeshott*: Evolution of an esterase gene cluster implicated in organophosphate resistance in insects (3) The molecular basis of diazinon resistance in the Australian sheep blowfly, *Lucilia cuprina*

Population Genetics I: Hone Theatre (Chair: Alan Wilton)
Abstracts: D(1-3)

10.00-10.20 *Y M Parsons, D W Cooper, L R Piper*: Genetic variation in Merino sheep

10.20-10.40 *Stella Koulianos: Molecular characterisation of Australian commercial and feral honeybee strains*

10.40-11.00 Ben Oldroyd*: Genetic variance in honey bees for preferred foraging distance*

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11.00-12.00 noon Tea/coffee, Posters and Trade Displays

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12.00-1.00 pm **Concurrent Sessions**

Gene Expression: Stirling Theatre (Chair: Rob Shroff)
Abstracts: E(1-3)

12.00-12.20 *Donna Cohen, Jill McGovern, Andrew Sinclair*: The testis-determining protein SRY enhances transcription of Fos-related antigen-1 (Fra-1) promoter constructs

12.20-12.40 *Nicole Chamalaun, Joan Kelly*: The CREA protein from *Aspergillus nidulans* is a DNA binding protein

12.40-1.00 *Michael Hynes, Julie Sharp, Meryl Davis*: Carbon and nitrogen metabolite repression of the *amdS* gene of *Aspergillus nidulans*

Friday July 9th (continued)

Population genetics II: Hone Theatre (Chair: Jeremy Timmis)
Abstracts: F(1-3)

12.00-12.20 *Michelle Waycott:* Genetic variation in the seagrass *Posidonia australis*: an investigation into clonal biology using RAPDs

12.20-12.40 *M Byrne, G F Moran*: Chloroplast DNA diversity in *Eucalyptus nitens*

12.40-1.00 *Carey Krajewski, Amy Driskell, Jodie Painter, Michael Westerman, David Edwards*: Molecular systematics of New Guinean Dasyurids

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1.00-2.00 pm Lunch: Committee to meet to decide Smith-White awards
****

2.00-3.00 pm **Concurrent Sessions**

Molecular Genetics I: Stirling Theatre (Chair: David Catcheside)
Abstracts: G(1-3)

2.00-2.20 *Saovanee Dharmsthiti, Viji Krishnapillai*: DNA sequence conservation at the gene level in a chromosomally conserved segment of two related *Pseudomonas* bacterial species

2.20-2.40 *J Williams, A Morgan, S Barett, N Brown, D Rouch, B Lee:*Copper resistance in enteric bacteria isolated from pigs in the U.K and Australia

2.40-3.00 *Thomaz Wilanowski, John Gibson*: The effects of a retrotransposon insertion on the expression of the *Gpdh* gene in *Drosophila melanogaster*

Genomic variation: Hone Theatre (Chair: Angela van Daal)
Abstracts: H(1-3)

2.00-2.20 *Chris Moran*: Survey of microsatellites repeats in the pig (*Sus domestica*) and the chicken (*Gallus domesticus*)

2.20-2.40 *Alan N Wilton, Judy Kaye, Guo Guanglan, V Michael Holers*: A CA repeat in an intron of CR2 used to examine linkage disequilibrium

2.40-3.00 *L M McKenzie, D W Cooper*: Low levels of restriction fragment length polymorphism in class II major histocompatibility complex genes in the tammar wallaby

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3.00-4.00 pm Tea/coffee, **Posters and Trade Displays**
****

4.00-5.00 **Concurrent Sessions**

Molecular Genetics II: Stirling Theatre (Chair: Rob Saint)
Abstracts: I(1-4)

4.00-4.20 *Michael O'Neill*: Enhanced germ cell specific transcription of *Tctex-1* due to deletion of repressor sequences

4.20-4.40 *D Catcheside, P Yeadon, F Bowring*: Molecular analysis of the genetic controls of meiotic recombination in Neurospora

4.40-5.00 *S Goode, M Morgan, A Mahowald*: Cloning of *brainiac*: a neurogenic gene of *Drosophila* which is required for multiple follicle cell functions during oogenesis

5.00-5.20 *Xinmin Li, Jan Nield, David Hayman, Peter Langridge*: Characterization of a gene associated with expression of self incompatibility in *Phalaris coerulescens*

Friday July 9th (continued)

Regulation & Selection: Hone Theatre (Chair: John MacKenzie)
Abstracts: J(1-4)

4.00-4.20 *Marion Healy, Mira Dumancic, Anh Cao, John Oakeshott*: Evolution of regulatory sequences conferring sex specific expression on *D. melanogaster EST6*

4.20-4.40 *B C Morrish, M J Healy, J G Oakeshott*: Complexities in *Est6* regulation uncovered using *D. melanogaster* and *D. yakuba Est6* promoters

4.40-5.00 *Susan Lawler*: Artificial selection for development time and the correlated response in the ribosomal genes of *Drosophila hydei*

5.00-5.20 *A G Davies, A Y Game, S Goodall, J Yen, J A McKenzie, P Batterham*: Evidence that an allele of the *Notch* homologue of the Australian sheep blowfly is an asymmetry modifier

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5.30 pm Annual General Meeting: Stirling Lecture Theatre

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7.30 pm Dinner: St Mark's College

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Saturday July 10th

9.00-10.00 am **Concurrent Sessions**

Human genetics: Stirling Theatre (Chair: Rory Hope)
Abstracts: K(1-3)

9.00-9.20 *G Peters, H Eyre, D Callen, P Allen, J Ford:* Phenotypically "neutral" translocations which predispose to oncogenic mutation in human malignancy

9.20-9.40 *Judith Ford, Tie Lan Han, Graham Webb:* Observed and implied aneuploidy in human germ cells

9.40-10.00 *Helen MacLean, Wai-Ting Choi, Simon Chu, Garry Warne, Jeffrey Zajac:* Triplet repeat mutations in the androgen receptor gene cause Kennedy's disease

Conservation genetics: Hone Theatre (Chair: Ross Crozier)
Abstracts: L(1-3)

9.00-9.20 *Craig Moritz, Shane Lavery, Michael Cunningham*: Problems in applying genetics in conservation: the need for a non-equilibrium approach

9.20-9.40 *N Kirk:* The population dynamics of the intertidal Ascidian *Pyura stolonifera* - a genetic analysis

9.40-10.00 *B A Houlden, W D Greville, W B Sherwin*: Molecular biology and conservation genetics of koala populations

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10.00-10.30 am Tea/coffee

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10.30-11.30 am **Concurrent Sessions**

Genetical concepts: Stirling Theatre (Chair: Carolyn Leach)
Abstract: M(1-3)

10.30-10.50 *Ian Franklin*: Multi-locus theory and intragenic evolution

10.50-11.10 *Oliver Mayo*: The evolution of dominance: a theory whose time is past?

11.10-11.30 *Richard Frankham*: Inbreeding and extinction

Gene organization, expression and evolution: Hone Theatre
(Chair: David Hayman)
Abstracts: N(1-3)

10.30-10.50 *Steven Cooper, Rachael Murphy, Gaynor Dolman, Guy Webber, Rory Hope:* Marsupial *beta*-globin gene organization and expression

10.50-11.10 *Charles Claudianos, Hugh Campbell*: The *Caenorhabditis elegans* homologue of the novel *Drosophila flightless-I* gene: a study in the evolution of the gelsolin gene family

11.10-11.30 *Alexander Dobrovic, Denise O'Keefe, R Sage, Elaine Batchelder:* Imprinting and loss of ABO antigens in leukaemia

****

11.30-12.30 pm **M J D White Address:** Florey Theatre
(Chair: Ross Crozier)
Abstract: O(1)

John MacKenzie: **Fundamental approaches, applied outcomes: Insecticide resistance in the Australian sheep blowfly**

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12.30 pm Buses leave for Woodstock Winery, pick up cases at St Mark's

1.30-4.30 pm Barbecue Lunch

4.30 pm Buses leave for airport and city, ETA airport 5.30 pm

****

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POSTERS

Abstracts: Posters (1-45)

1. *Greg Adcock, Thomas Loy:* Ancient DNA and Australian prehistory: Preliminary analysis of DNA sequences derived from sub-fossil Australian Aboriginal bone
2. *Deryn Alpers, Bill Sherwin*: Molecular genetics and demographic modelling in management of an endangered species: the Northern hairy-nosed wombat
3. *Z Baxter, S Thomas:* Characterisation of DNA polymerase 1 in the marine bacterium *Vibrio natriegens*
4. *F Bowring, D Catcheside*: The search for *rec-2*: a gene that modulates the level of meiotic recombination in *Neurospora crassa*
5. *H Brereton, D Turner*: Origins of polymorphism at a polypurine hypervariable locus
6. *Michael Cancilla, Alan Hillier, Barrie Davidson, Gaetan Limsowtin, Ian Powell*: Strain identification of lactic acid bacteria using genetic fingerprints generated by arbitrarily-primed PCR
7. *David Clancy*: Cytoplasmic incompatibility and sex ratio distortion in insects infested with *Wolbachia pipientis*: mode of spread, host mtDNA evolution and work towards pest and disease control
8. *Chris Collett, Ros Joseph*: Characterization of "expressed sequence tags" from a marsupial mammary gland library
9. *K Davies, L Christidis, M Westerman*: A molecular assessment of the taxonomic status of Cox's sandpiper (*Calidris paramelanotos*)
10. *S Downie, S Donnellan, D Anderson*: Molecular phylogeny of the Galliform birds - evidence from mitochondrial DNA sequence
11. *Mark Dowton, Andrew Austin*: PCR products can be directly sequenced without intermediate purification
12. *Jennie Finch, Angela van Daal, Rory Hope*: Species identification and sex determination using PCR
13. *P Flynn, B Cheetham, M Katz:* The characterisation of an *Aspergillus nidulans* strain, defective in the production of extracellular proteases under certain growth conditions
14. *C Fowler, V Barrett, G Baker, C Garbett, D Burzacott, A Dyer:* Molecular characterisation of earthworm species and strains in southern Australian soils
15. *A Game, A Davies, S Goodall, T Williams, J Yen, B Yen, P Batterham, J MacKenzie*: Molecular and genetic analysis of the *Scalloped wings* gene of *Lucilia cuprina*
16. *David Hayman and Meredith Smith:* Further evidence for differences in recombination frequency between the sexes of marsupial species
17. *David Hayward, Peter Atkinson, Antony Howells:* Isolation of the *sex-lethal* gene of the Australian sheep blowfly, *Lucilia cuprina*
18. *Lynn Jones, Helena Richardson, Robert Saint*: Genomic and genetic characterization of the cyclin E gene of *Drosophila melanogaster*
19. *Roslyn Joseph, Chris Collett*: A two-colour *in situ* hybridization study of mRNAs in Tammar wallaby mammary gland
20. *P Leeton, L Christidis, M Westerman*: The avian order Pelicaniformes: monophyletic or polyphyletic?
21. *Robyn Lints, Meryl Davis, Michael Hynes*: Putting a finger on *amdS* regulation by the *amdA* gene of *Aspergillus nidulans*
22. *Andrew Lonie, Stanley Robert, Richard D'Andrea, Robert Saint*: The *Polycomblike* gene of *Drosophila melanogaster* is required for the correct expression of the homeotic genes of the Antennapedia and Bithorax complexes
23. *Amir Masoumi, Margaret Katz*: Genetic and molecular study of a putative regulatory gene in *Aspergillus nidulans*
24. *Muladno, Paul Le Tissier, Chris Moran*: Mapping polymorphic microsatellites in the pig (*Sus domestica*)

POSTERS (continued)

25. *Phillip Murphy, Peter Langridge, Sally Smith*: Generation and identification of putative mycorrhizal mutants of *Hordeum vulgare* cv. "Galleon"
26. *Rachael Murphy, Helene Martin, Richard Todd, Meryl Davis, Michael Hynes*: An important factor in *amdS* gene regulation
27. *D O'Keefe, A Dobrovic:* Allelic silencing is responsible for the loss of ABO blood group antigens in malignancy
28. *Anthony Pietsch, Doug Anderson, Carolyn Leach*: Computer simulations for population genetics
29. *D Rowell, A Higgins, D Briscoe, N Tait*: Chromosomal evolution in Onychophorans from Australia (Onychophora: Peripatopsidae)
30. *G Sargent, C Fowler, R Wells, S Donnellan, T Reardon:* Phylogenetic analysis of extinct and extant Diprodontian Marsupials using DNA sequencing
31. *Ian Scott, L Christidis, M Westerman*: Molecular phylogenic relationships of the Pachycephalinae (Passeriformes: Aves)
32. *Sarah Sherson, Jonathan Medd, Chris Cobbett*: Mapping a gene involved in arabinose metabolism in *Arabidopsis thaliana*
33. *Robert Shroff, Joan Kelly*: Analysis of mutations affecting carbon catabolite repression in *Aspergillus nidulans*
34. *L Silva, J Kirton, I Martin, C Moran, F Nicholas*: Prolific inbred lines of mice
35. *M Spackman, S McKechnie*: Assessing population structure in the cotton bollworm, *Helicoverpa armigera (Lepidoptera: Noctuidae)*, using variation in the AT-rich region of mitochondrial DNA
36. *J Sved, L Blackman, R Colless, Y Svaboda*: The mechanism of P element-induced recombination in *Drosophila melanogaster*
37. *M Tan, P Wong, M Holley*: Identification of varieties of *Gaeumannomyces graminis* and their isolates by analysis of nuclear ribosomal DNA
38. *Francois Tardif, Michael Burnet, Stephen Powles*: Inheritance of resistance to herbicides that inhibit ACCase in annual ryegrass
39. *Andrea Taylor, Bill Sherwin, Bob Wayne*: Simple sequence polymorphisms as a measure of genetic variability in the endangered Northern hairy-nosed wombat
40. *Richard Todd Rachael Murphy, Helene Martin, Margaret Katz, Meryl Davis, Michael Hynes*: Analysis of an *Aspergillus* transcription Fac-tor
41. *Angela van Daal, Carolyn Harrington, Kim Williams, Tony Focareta, Nicholas Sly, Rhett Swanson*: The use of highly polymorphic DNA markers using PCR in forensic science
42. *C Warr, A Phillips, L Kelly*: Investigation of the *trp* and *trpl* phototransduction genes of *Drosophila melanogaster*
43. *Margaret Wexler, David Gordon, Peter Murphy*: The distribution of Rhizopine genes in *Rhizobium* populations
44. *Dara Whisson, Herdina*: Molecular evidence for the generation of variability in *Rhynchosporium secalis*
45. *P Yeadon, D Catcheside*: Sequence differences between *cog* and cog^{+}, two alleles of a recombinator in the *histidine-3* region of *Neurospora crassa*

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Chromosome 11 (D11Z1)
Chromosome 12 (D12Z3)
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Chromosome 14/22 (D14Z1/D22Z1)
Chromosome 15 (D15Z)
Chromosome 16 (D16Z2)
Chromosome 17 (D17Z1)
Chromosome 18 (D18Z1)
Chromosome 20 (D20Z1)
Chromosome X (DXZ1)
Chromosome Y (DYZ3)
Chromosome Y Cocktail (DYZ1, DYZ3)
All Human Centromeres

Beta Satellite Probes
Chromosome 9 (D9Z5)
Acrocentric Chromosomes

Classical Satellite Probes
Chromosome 9 (D9Z1)
Chromosome 15 (D15Z1)
Chromosome Y (DYZ1)

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Chromosomes 3, 4, 7, 8, 9, 10, 11, 13, 14, 15, 16, 17, 18, 19, 21, 22, X
Chromosomes 2, 5, 6, 12, 20, and Y to be released by the end of 1992

COSMID PROBES

Cosmid DNA Probes for Microdeletion/ Microduplication Syndrome Research
Charcot-Marie-Tooth (CMT1/Type 1A) Cosmid
Cri Du Chat Cosmid
Miller-Dieker Cosmid/Chromosome 17 α-satellite (D17Z1)
Prader-Willi/Angelman Syndrome Cosmids
Wolf-Hirschhorn Cosmid/Chromosome 4 α-satellite (D4Z1)

Chromosome-Specific Cosmid DNA Probes
Chromosome 13-Specific Cosmid
Chromosome 21/Down Syndrome Critical Region Cosmid

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Human Tel 4p Cosmid/Chromosome 4 α-satellite (D4Z1)
Human Tel 7q Cosmid/Chromosome 7 α-satellite (D7Z1)
All Human Telomeres

ONCOLOGY/PATHOLOGY:

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p53 Cosmid
p53 Cosmid/Chromosome 17 α-satellite (D17Z1)
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RES001.3

Patterns of evolutionary change in insect mtDNA AT content

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A tendency of related organisms to have similar base content (adenine and thymine = AT%) in the 5S rRNA gene was noted in Eubacteria only 6 years ago. While this result suggests that a similar phenomenon also might occur in Metazoa, supporting data has been made available only recently.
Three mitochondrial genes (16S rRNA, cytochrome oxidase sub-unit II and cytochrome b) from 26 species of insects have been analysed. The results show that the AT% of these genes fluctuates between 53.3% and 80.7% and that species with approximately the same AT% often are related. For the cytochrome oxidase sub-unit II and cytochrome b genes the data furthermore that the bias in the AT% is accompanied by an acceleration of the amino acid sequence divergence. A simple model to explain this phenomenon is presented.

CADMIUM-SENSITIVE MUTANTS OF *ARABIDOPSIS THALIANA*

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A genetic approach using the small crucifer *Arabidopsis thaliana*, was adopted to investigate the mechanisms by which plants respond to heavy metals. A Cd-sensitive mutant, *cad1*, was isolated and genetic analysis has shown that the sensitive phenotype is recessive to the wild type and segregates as a single Mendelian locus. Using genetic crosses, the *CAD1* locus was mapped to the *tt3* region of chromosome five and from RFLP analysis the *CAD1* locus is located less than 0.5 cM from the 12S seed storage protein gene, *cra1*. The isolation and charaterisation of the *cad1* mutant has been published, (Howden and Cobbett, 1992).

From a screen of x-ray mutagenised (M2) seeds another cadmium sensitive mutant, *cad2* was isolated. Genetic analysis shows that *cad2* is not allelic to *cad1*. The *cad2* mutant is also phenotypically different from *cad1*, appearing less sensitive to low levels of Cd. A phenotypic analysis of the *cad2* mutant, using the *cad1* mutant and wild-type as comparisons, was undertaken.

Using Gel filtration columns, Cd-binding proteins have been detected in *Arabidopsis* and they show similar features to the Cd-binding peptides (phytochelatins) present in plant cell cultures and yeast. Striking differences have been observed in the profiles of the Cd-binding proteins of the *cad1* and *cad2* mutants, indicating possible mutations in the phytochelatin pathway.

Howden,R. and Cobbett,C. (1992) Cadmium-sensitive mutants of *Arabidopsis thaliana*, Plant Physiology, 100: 100 -107

Regulation of the *string* gene during embryonic cell proliferation. B.Patterson, P.Wigley, R.Saint. Dept. of Biochemistry, Adelaide Univ., GPO Box 498, South Australia, 5001, 61-08-2285721.

string is a *Drosophila* homolog of the yeast mitotic activator *cdc 25* (Edgar & O'Farrell, 1989). In *S.pombe*, *cdc 25* has been shown to activate MPF (mitosis promoting factor), by dephosphorylating the p34cdc2 kinase component (reviewed by Millar & Russell, 1992). The active form of MPF is then able to drive cells from G2 into mitosis.

During *Drosophila* embryogenesis cell proliferation follows a set pattern. The earliest divisions occur synchronously across the embryo but later these cease and a period of G2 arrest is observed. The divisions that follow this G2 arrest are asynchronous with patches of tissue, termed mitotic domains entering mitosis at specific times. These mitotic domains occur in an invariant spatio-temporal pattern and in many cases correlate with organ primordia (Foe, 1989).

Several lines of evidence suggest that *string* is responsible for the spatio-temporal pattern of these divisions:

-*string* mutant embryos never undergo these domain specific divisions (Edgar & O'Farrell, 1989),

-*string* mRNA expression occurs in the same spatio-temporal pattern as the mitotic domains but precedes it by about 20 minutes (Edgar & O'Farrell 1989) and,

-ectopic expression of *string* can override the mutant phenotype (Edgar & O'Farrell 1990).

The work presented here investigates the regulation of *string*. It seems likely that the patterning genes will be involved at some level in generating the pattern of mitotic domains and it is possible that *string* is responsible for integrating this information. We therefore anticipate that the regulation of *string* may be complex, with many enhancers being required to generate the correct pattern of *string* expression.

A *lacZ* reporter gene approach has been undertaken to define enhancers that are required for *string* expression This work has identified a region upstream of *string* that activates transcription in several mitotic domains. The enhancer(s) within this region require elements adjacent to the transcription start site (other than those provided in the basal promoter of the reporter gene) to be present before their detection is possible.

B.A.Edgar & P.H.O'Farrell (1989) Cell 57, 177-187.
J.B.A.Millar & P.Russell (1992) Cell 68, 407-410.
V.E.Foe (1989) Development 107, 1-22.
B.A.Edgar & P.H.O'Farrell (1990) Cell 62, 469-480.

TATA box and amino acid mutations in the *sn*-glycerol-3-phosphate gene in *Drosophila melanogaster*.

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Two naturally occurring variants of the *sn*-glycerol-3-phosphate gene have been characterized and found to produce very low enzyme activity in all life stages. The variant genes were cloned. DNA sequence data from one variant indicated that the nucleotide changes in the eight coding exons were unlikely to lead to alterations in GPDH activity. Further analyses of the 5' region of the transcription unit showed that the region of the normal TATA box was deleted but a 20bp insertion in the same region included a putative TATA box like sequence. Studies showed that the variant produced two very weak transcripts, one of which is initiated from the inserted TATA box sequence and the other from a region some 200 nucleotides upstream. Sequence data from the other variant showed two amino acid changes present in exons 1 and 5. While unable to positively determine which mutation is behind the low activity, the arginine to cysteine substitution in exon 5 may play a role in destabilising the GPDH protein conformation.

The *Polcomblike* gene of Drosophila melanogaster is required for the correct expression of the homeotic genes of the Antennapedia and Bithorax complexes.

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Clonal analysis indicates that the expression of parasegmental specific sets of homeotic genes from the Antennapedia (ANT-C) and Bithorax (BX-C) complexes is required throughout *Drosophila* development. However, the expression of the early patterning genes, which direct specific homeotic gene expression , is transient, ending just after germ band extension. A mechanism must exist by which the determined state of cells within a parasegment can be maintained throughout development.

Mutations in any one of a group of unlinked genes known as the *Polycomb* group (Pc-G), lead to the derepression of homeotic gene expression after the early patterning gene products have degraded. Mis-expression of the homeotic genes as a result of these mutations results in transformations of body segments to resemble those normally found in a more posterior position. Various lines of evidence suggest that the Pc-G gene products act as a multimeric protein complex, specifically recognising genes that are repressed by the early patterning genes, forming a chromatin structure that is transcriptionally inactive and clonally heritable.

We have recently cloned a member of the Pc-G known as *Polycomblike*. Sequence analysis of the *Polycomlike* cDNA has revealed one large ORF, which when conceptually translated encodes a protein of ~ 100kD with no significant homology to other known proteins. *In situ* hybridisation indicates that the Pcl transcript is expressed throughout the embryo. We have raised a polyclonal antiserum to the *Pcl* protein and used it to localise the *Pcl* product in situ. These experiments have revealed that Pcl is a nuclear localised protein. Further, we have found that Pcl binds specifically to approximately 120 sites on polytene chromosomes. These sites are identical to those bound by two other members of the Pc-G : Polycomb and polyhomeotic[1]. We have demonstrated that Pcl is co-immunoprecipitated with Polycomb from embryonic nuclear extracts, indicating that they interact with each other in vivo. These data supports a model in which members of the Pc-G form a protein complex which specifically recognises, binds and represses target genes in response to the local chromatin environment.

1. Franke, A., De Camillis, M., Zink, D., Cheng, N., Brock, H., Paro, R.(1992) *EMBO* J. **11**, 2941-2950

GENOMIC AND GENETIC CHARACTERISATION OF THE CYCLIN E GENE OF *DROSOPHILA MELANOGASTER*

Lynn Jones, Helena Richardson and Robert Saint
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G1 cyclins are regulatory proteins involved in control of the cell cycle at the G1 to S phase transition. They associate, as regulatory subunits, with members of the Cdk family of serine/threonine protein kinases. Candidate metazoan G1 cyclins include cyclin C, D and E. Cyclin E is an excellent candidate for a rate limiting regulator of the G1 to S phase transition, on the basis of its expression and associated kinase activity at the G1 to S phase boundary. We have recently isolated a *Drosophila* cyclin E homolog. *Drosophila* provides an ideal system to investigate the role of cyclin E in the regulation of cell proliferation during development.

Drosophila cyclin E (*DmcycE*) has been mapped to region 35D on the left arm of chromosome two. This region is genetically well characterized, as it is near the *Alcohol dehydrogenase* gene (*Adh*) which maps to 35B. The existence of a number of deficiencies and lethal alleles in this region gave us a starting point to more precisely locate the *DmcycE* gene with the aim of identifying a mutation in the gene. Southern analysis of the available deficiency strains revealed that *DmcycE* maps to a new genetic locus between the 35Da and *snail* loci. P element and EMS mutagenesis studies are currently underway to generate a specific *DmcycE* mutation.

The isolation of cosmid clones derived from the *DmcycE* region and Southern analysis, using cDNA clones, has revealed that the gene is complex. The gene covers at least 20 kb of sequence and contains many introns. The intron/exon boundaries and the 3' end of the two types of *DmcycE* transcripts have been determined by sequence analysis. The location of the putative 5' ends of the gene have also been detemined. This analysis has demonstrated that the two alternative 5' domains of the gene share a 3' acceptor splice site for a common 3' domain.

HOMEOTIC GENES AND SELF/NON-SELF RECOGNITION IN THE MUSHROOM *COPRINUS*

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Mating between genetically different haploid cells is an essential step in the life cycle of the inkcap mushroom *Coprinus cinereus* - no special cells are required and no cell signalling mechanism, simply chance fusion between somatic hyphae. If cells belong to different mating types, hyphal fusion triggers a major change in gene expression that initiates a developmental pathway culminating in sexual reproduction. The genes that determine mating type and whose role is to distinguish self from non-self cell fusions, map to two gene complexes termed the A and B mating type factors. What is remarkable about these genes is that they are multiallelic and several thousands of non-self compatible interactions must be recognised in nature. We have cloned and sequenced genes of the A mating type factor and show that these encode two dissimilar homeodomain proteins. By transformation and gene disruption we can show that a single protein of each class is sufficient to trigger sexual development but these must come from different A factors. A simple model can now explain non-self recognition and the simultaneous regulation of cell type gene expression following mating - the ability of these two classes of proteins to heterodimerise and to form a transcriptional activator/repressor of the genes they regulate. Sequence analysis allows us to predict possible functional domains within the two classes of proteins and current work is directed towards testing our model in both homologous and heterologous systems.

A → 160 alleles
B → 79 → 12640 mating types

Compatible matings - must differ at both loci.

A - two closely linked genes - α + β - both multiallelic
0·07 map units apart.
- only α or β needs to be different.

α - 2 loci (α1-1), α2-1 pseudogene??
β - 3 loci β1-1, β2-1, (β3-1), β4-1, (β5-1)

7kb - homologous hole - no transcripts, but site of recombinational shuffling

No cross hybridisation between loci or even some alleles at a particular locus - if hybridises, doesn't elicit clamp cell development.

A locus - 2 classes of genes + product. HD1-1 ⇌ HD2-1 - divergently transcribed
In Saccharomyces HD1-2 HD2-2

4 × 6 × 3 ⇒ 72 mating types

Evolution of an esterase gene cluster implicated in organophosphate resistance in insects.
(1) Organisation and sequence of the cluster in *Drosophila melanogaster*

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2. Division of Botany and Zoology, ANU, GPO box 4, Canberra, ACT 2601.

Four biochemically defined esterase phenotypes map to the 84D3 region of the *Drosophila melanogaster* genome. These are Est9, Est23, malathion carboxylesterase (MCE) and ali-esterase. The homologs of the last three are implicated in organophosphate (OP) resistance in various Diptera. We have cloned the cluster in *D.melanogaster* by screening a YAC clone, containing genomic DNA spanning the 84D3-10 region, with oligonucleotide probes derived from consensus esterase sequences. Eleven distinct regions of esterase homology were identified within a 90kb stretch of genomic DNA. Sequence data indicate at least ten of these regions could be functional genes. We use the sequence, and the variation in gene orientation and intron location, to reconstruct the evolution of the cluster.

Evolution of an esterase gene cluster implicated in organophosphate resistance in insects.
(2) Correlation of the cloned genes with biochemical phenotypes in *Drosophila melanogaster*.

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Four of eight biochemically defined esterase phenotypes in *Drosophila melanogaster* which have been genetically mapped lie within the 84D3 region of chromosome 3R. Well over 20 other esterases have also been identified on biochemical criteria but have not yet been mapped genetically. The aim of this project has been to determine which of the various biochemically defined esterases are encoded by the ten esterase genes we have cloned from the 84D3 cluster. Our first step has been to carry out strand-specific developmental Northern analysis on the ten cloned genes. These data are then compared with the known developmental profiles of the various biochemical phenotypes. By these means we identify putative gene products for several of the cloned genes. In particular, we can identify candidate genes for three of the enzyme phenotypes that have been implicated in OP insecticide resistance. We then re-interpret the sequence of these candidate genes in terms of the active site structures required for an esterase to hydrolyse OP's.

C(3)

Evolution of an esterase gene cluster implicated in organophosphate resistance in insects. (3) The molecular basis of diazinon resistance in the Australian sheep blowfly, *Lucilia cuprina*.

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2. **Division of Botany and Zoology, ANU, GPO Box 4, Canberra, ACT 2601.**

The Australian sheep blowfly, *Lucilia cuprina*, has evolved resistance to most of the insecticides used in an attempt to control it. Field resistance has been described to dieldrin, malathion and most recently diazinon. Resistance to diazinon is associated with a naphthyl acetate non-staining form of the esterase isozyme, E3, which maps in with a number of other esterase genes on the left-arm of chromosome four. Also mapping within this esterase cluster is the locus responsible for malathion resistance.

The homologous region in *Drosophila melanogaster* has been cloned and the presentation by C. Robin *et al.* details preliminary analysis of this 90kb cluster of 10 esterase genes. To clone the E3 gene from *L. cuprina*, esterase and cluster specific PCR primers have been used to amplify candidate esterases sequences. In turn these sequences have been used to characterise the molecular structure of the esterase cluster in *L. cuprina* and in particular, along with the data presented by Court *et al.*, identify esterase genes in the cluster responsible for insecticide resistance.

A discussion of the potential insights into the evolutionary process that the cloning of the diazinon resistance gene will provide is presented.

GENETIC VARIATION IN MERINO SHEEP

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The Australian merino has arisen through selective breeding over a period of approximately 150 years. The Spanish merino is recognised as being the most notable ancestor of the Australian merino but judicious crossing with a number of other breeds also occurred. The full history of the genetic contribution to the Australian merino will probably never be known but records available to-day indicate a somewhat diverse ancestral gene pool. Given the variety of ancestral genetic material and the short time since the evolution of the breed it would seem reasonable to expect a high degree of genetic variation within the Australian merino breeds. Despite this it is sometimes supposed that genetic variation could be limited due to the hierarchical closed flock system practised by Australian sheep breeders.

We have carried out an investigation to determine the degree of genetic variation in a representative merino breed, the medium Peppin, based mainly on restriction fragment length polymorphisms. Polymorphisms at a number of loci, including keratin-associated-protein genes, immunoglobulin genes and various hormone genes, have been examined and allelic frequencies and PIC values determined. The results of this study indicate that the level of genetic variation in merino sheep is quite high.

$\hat{H}$ = 31.5% ± 6%
looking at polymorphic loci only (6 loci)
~13% for all loci included

MOLECULAR CHARACTERISATION OF AUSTRALIAN COMMERCIAL AND FERAL HONEYBEE STRAINS

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Earlier DNA studies on honeybees involved only those sites where specific enzymes cleave. Such information allows a characterisation of honeybee races, but generally lacks the power to determine lineages within races. Sequencing DNA greatly increases the volume of information and therefore improves our ability to distinguish strains. I am now able to sequence DNA from single bees, thus allowing the development of rapid diagnostic tests for particular strains.

Once information is available in terms of the sequence of nucleotides of mitochondrial DNA from various strains, any new strain can not only be identified as such to the extent allowed by the variation actually uncovered, but its relationship along the maternal line to other strains determined.

Sequence information has been obtained for part of the ATPase 6 gene, a short intergenic region, the COIII gene, the cytochrome b gene and the ND2 gene for 40 hives one of which is Africanised (obtained in alcohol from Glenn Hall, USA). RFLP analysis has also been performed to ascertain to which races the haplotypes found belong.

Analysis of the ATPase 6 and COIII region in Australian bees revealed 2 alleles differing by 7 base substitutions. A third allele represents the Africanised bee. The mean divergence (and 95% confidence limits) between the two Australian mtDNA haplotypes is $0.020 \pm 8 \times 10^{-4}$. Given the smaller effective population size of social insects, persistence of these alleles for so long is unexpected and may indicate their origin from more than one group of subspecies in Europe. Unfortunately the mtDNA evolutionary rate has not been calibrated in bees. However, given a conservative estimate of 2% per million years the time of divergence must be well before the glaciation of Europe.

Analysis of the cytochrome b and ND2 region revealed 4 alleles in the Australian bees. A fifth allele represents the Africanised bee. Although more variation was found in these regions, the nature of this variation suggests a much more recent time of divergence than that for the ATPase 6 and COIII region.

GENETIC VARIANCE IN HONEY BEES FOR PREFERRED FORAGING DISTANCE

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The worker population in a honey bee (*Apis mellifera*) colony is divided into sub-families (patrilines), each sired by a different drone. In an experimental colony comprised of two identifiable sub-families, I found that bees of one sub-family preferentially attended a feeding station close to their hive, rather than a more distant station. Bees working at their subfamily's "preferred" foraging distance executed communication dances at a higher frequency than bees working at their non-preferred distance. Recruit bees preferentially attended dances which described a feeding site at their sub-family preferred distance.

Analysis of dances performed by bees of this colony for natural sources of food confirmed that these sub-families had the foraging distance preferences suggested by the feeding station experiment. In additional experiments with two other colonies, I again found sub-family differences in feeding places, suggesting that genetic variance for feeding preferences is widespread. I speculate that genetically determined specialism in foraging tasks may reduce intra-colonial competition for resources, and may help maintain multiple mating in honey bees.

The Testis-Determining Protein SRY Enhances Transcription of Fos-related antigen-1 (Fra-1) Promoter Constructs.

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The Y-linked *SRY* gene has been shown to be the mammalian testis determining gene, TDF. In mouse *Sry* is expressed in somatic cells just prior to development of the testis, but shows germ cell dependence in the adult mouse testis. *Sry* encodes a protein containing an HMG-box motif that is capable of binding DNA, suggesting that SRY may regulate gene expression. *SRY* is expressed during embryogenesis, where its role is to cause the genital ridge to develop as a testis but it is also expressed in the adult testis, where its role is unknown. The SRY protein recognizes the general core sequence 5' -CCTTTGT.

The *fos*-related antigen-1 (*fra*-1) gene is closely related to the proto-oncogene c-*fos* and encodes a member of the transcription factor AP-1. *Fra-1* is expressed in a number of cell types following a diverse range of stimuli and during spermatogenesis. The 5' regulatory region of the *fra*-1 gene contains five copies of the general core sequence to which the SRY protein will bind.We wished to determine whether SRY is involved in the regulation of *fra-1* during spermatogenesis.

Our results demonstrate that purified SRY protein can bind strongly to one of the HMG-box response elements (HBRE's) in the *fra-1* promoter, and that SRY significantly enhances the transcription of *fra-1* promoter constructs in co-transfection experiments. These results suggest that *SRY* can act as a transcriptional activator, and that the function of the SRY protein is mediated, at least in part, by activation of members of transcription factor AP-1.

SRY - single exon gene
- HMG box - DNA binding motif

binds CCATTGTTCT
in minor groove
- bends DNA 85°

SRY expressed in adult testis
- circular RNA molecule

The CREA protein from *Aspergillus nidulans* is a DNA binding protein

by Nicole D. Chamalaun and Joan M. Kelly

Carbon catabolite repression (or glucose repression) is a wide domain regulatory phenomenon in which the synthesis of enzymes and permeases involved in the utilisation of less favoured carbon sources is repressed in the presence of a more easily metabolised carbon source such as glucose. Carbon catabolite repression operates in addition to induction whose role is to ensure enzymes are turned on only when a substrate is present.

In the eukaryote *A.nidulans* carbon catabolite repression is mediated through the negatively acting regulatory gene, *creA*. The *creA* gene has been cloned and sequenced (1) and the theoretical protein contained a zinc finger DNA binding motif of the TFIIIA, C_2H_2 type.

The techniques of gel mobility shift assays and DNase sensitivity assays (footprinting) were used to locate regions in the 5' promoters of two genes where evidence for CREA control exists. These are the structural gene for acetamidase encoded by *amdS* (2), and *facB*, a gene that encodes a regulatory protein involved in acetate utilisation (3).

Results of experiments using the *E.coli* expressed fusion protein between glutathione-S-transferase and CREA show that CREA does bind DNA in a sequence specific manner. Furthermore this interaction with DNA is dependent on Zn^{2+} ions. Comparison of the binding sites in the *amdS* and *facB* promoter regions with those reported for *alcA* and *alcR* promoters (Kulmberg *et al.*, in press) led to a consensus sequence of 5' C/G Py GGGG 3'.

In addition, sequence preferences were determined using cyclic amplification and selection of targets (CASTing). In this procedure random oligonucleotides were selected using the CREA fusion protein, amplified, cloned and sequenced.

1. Dowzer, C.E.A. and Kelly, J. M. (1991) *Mol. Cell Biol.* **11**: 5701-5709.
2. Corrick, C.M., Twomey, A.P. and Hynes, M.J. (1987) *Gene* **53**: 63-71.
3. Katz, M.E. and Hynes, M.J. (1989) *Mol. Cell Biol.* **9**: 5696-5701.

Carbon and nitrogen metabolite repression of the *amdS* gene of *Aspergillus nidulans*. Michael J. Hynes, Julie A. Sharp and Meryl A. Davis, Department of Genetics, University of Melbourne.

The *amdS* gene of *Aspergillus nidulans* is regulated by a number of specific induction controls as well as by nitrogen and carbon metabolite repression. Either carbon or nitrogen limitation is sufficient for derepression of *amdS* expression. Experiments involving *in vitro* mutagenesis of the 5' region of the *amdS* gene and a two-step gene replacement method with an *amdS-lacZ* reporter construct have been used to investigate these control mechanisms. The *areA* gene encoding a member of the GATA family of transcription factors mediates nitrogen metabolite repression and mutation of a single consensus binding site eliminates approximately 90% of this regulation. Deletion of two consensus binding sites for the carbon catabolite repression protein CREA completely relieves glucose repression but also causes decreased expression under carbon limitation conditions. This may suggest a positive control function acting at these sequences. A CCAAT element has been shown to have a general enhancer effect on expression independent of inducer and carbon or nitrogen limitation. These studies also provide evidence for even more uncharacterized control mechanisms.

Genetic variation in the seagrass *Posidonia australis*: an investigation into clonal biology using RAPDs.

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Seagrasses have a high degree of apparent clonality. This renders investigations into their genetic systems problematic. Seagrass meadows can cover large areas, sometimes many kilometres in diameter. The genetic structuring within these large areas of seagrass meadow remains untested. This is in part due to the hitherto unavailability of techniques that are sensitive to the potentially small differences in genotypes among closely related individuals.

Posidonia australis is the most widespread of the southern Australian endemic seagrasses. It has a distribution from Shark Bay in Western Australia, across to northern Tasmania and up to Lake Macquarie in New South Wales.

The current study has used RAPD (Random Amplified Polymorphic DNA) analysis to examine genetic variation in *P. australis*. It has shown that there are differences in RAPD phenotypes within small areas (m^2) of seagrass meadow. This variation may reflect multiple seedling recruitment events or intertwined ramets of larger genets. This finding has important implications to the understanding of seagrass genetics and for future investigations into genetic variation in this species.

CHLOROPLAST DNA DIVERSITY IN *EUCALYPTUS NITENS*

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Diversity in the chloroplast genome within and between populations of *Eucalyptus nitens* was investigated. Restriction fragment length polymorphism was determined for twenty restriction enzymes using heterologous probes. Ten individuals from eight populations covering the geographic range of the species were analysed for variation in the cp genome. The level of variation detected in this species is relatively high for the chloroplast genome. 25 polymorphisms were detected in the survey of which 15 were restriction site mutations and 10 were length mutations. The mean nucleotide diversity in the species was 0.084%. The N_{ST} value of 80.2% indicated that most of the diversity in the genome is maintained between populations. Analysis of population relationships split the populations into two groups, with the central Victorian and southern New South Wales populations forming one group and the eastern Victorian and northern New South Wales populations forming a second group. Four of the mutations were population specific mutations. Three of these occurred in the Barrington population which is a very small population in northern New South Wales. These results suggest that sufficient variation exists for chloroplast DNA to be used to investigate evolutionary forces in some eucalypt species.

Discordance between chloroplast & genome (isozyme) data.

? Barrington with introgressed chloroplast genome.

MOLECULAR SYSTEMATICS OF NEW GUINEAN DASYURIDS

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Although most members of the marsupial family Dasyuridae are found in Australia, several species are endemic to New Guinea. These endemics occur at several levels in the taxonomic hierarchy, including the subfamily (Phascolosoricinae, Muricinae), genus (*Myoictis*), and species (*Antechinus* spp., *Planigale novaeguinea*) levels. Previous molecular analyses of dasyurid phylogeny using DNA hybridization (Kirsch *et al.*, 1990) and microcomplement fixation (MC'F Baverstock *et al.*, 1990) have suggested that endemic New Guinean genera are sister groups to Australian clades. These results are largely confirmed by analysis of DNA sequence data from the mitochondrial cytochrome-b gene. Phascolosoricinae (*Phascolosorex* and *Neophascogale*) appears to be allied with Dasyurinae. Muricinae (*Murexia*) is clearly a relative of Phascogalinae, as are the New Guinean members of the genus *Antechinus*, though these are more closely related to *Murexia* than to their Australian congeners. *Myoictis* appears to be part of a clade that includes some of the Australian false antechinuses. The position of *Planigale novaeguinea* is not yet clear; it appears to be sister to one or more Australian species, but not to the entire Australian planigale assemblage.

Baverstock, Krieg, and Birrell, 1990, P. 131 in 'Mammals from Pouches and Eggs', Marshall-Graves, Hope, and Cooper (eds.), CSIRO, Australia

Kirsch, Krajewski, Springer, and M. Archer, 1990, Aust. J. Zool. 38: 673

DNA SEQUENCE CONSERVATION AT THE GENE LEVEL IN A CHROMOSOMALLY CONSERVED SEGMENT OF TWO RELATED *PSEUDOMONAS* BACTERIAL SPECIES

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At a previous GSA meeting (Adelaide 1990) we reported DNA and deduced amino acid sequence comparisons of a pair of isofunctional biosynthetic genes of two phylogenetically closely related bacterial species, *Pseudomonas aeruginosa* and *P. putida*. This was that of *argA* encoding N-acetylglutamate synthase, the first gene of the arginine biosynthetic pathway. As discrepancies were subsequently identified in the DNA sequence determination due to G/C compressions as *Pseudomonas* DNA is high in G/C (60-65%) the sequencing was repeated using the Applied Biosystems Dye Primer Sequencing Kit with DNA Taq polymerase at 70-95°C to minimize potential secondary structures that could lead to compressions.

The reanalysis of the comparative sequences of the subsequently obtained DNA sequences of these pair of *argA* genes shows a high level of homology. That is overall homologies of 81% and 84% at the nucleotide and deduced amino acid sequence levels, respectively, were observed. This high level of homology was also reflected in very similar hydropathy profiles of the encoded proteins, patterns of codon usage including rare codon usage and amino acid composition. This high level of homology has also previously been shown by others for pairs of six genes of the tryptophan biosynthetic pathway (*trp*) between the two *Pseudomonas* species. The *argA* and *trp* genes are located within a conserved chromosomal segment of the respective chromosomes of *P. aeruginosa* and *P. putida*, referred to as the auxotrophic-rich region, and thus the genetic conservation maintained at the chromosomal level is also reflected in the high level of conservation at the nucleotide sequence level of the genes.

These findings suggest that despite chromosomal rearrangements (inversions and transpositions) previously detected in the auxotrophic-rich region, there appears to have been selective pressure to maintain overall genetic conservation at the chromosomal and gene level in the region.

COPPER RESISTANCE IN ENTERIC BACTERIA ISOLATED FROM PIGS IN THE U.K. AND AUSTRALIA.

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Tetaz and Luke (1983) showed that copper-resistant strains of *Escherichia coli* isolated from pig faeces carried a conjugative plasmid pRJ1004. This plasmid confers a 3 fold increase in resistance to copper sulphate in Luria broth agar to the host due to *p*lasmid-borne *co*pper resistance (*pco)* genes.

The *pco* determinant has been cloned and sequenced. The sequence data indicate that there are seven open reading frames which have been named *pco* ABCDRSE. Copper resistance in *Pseudomonas syringae* pv.*tomato* has been localized to approximately 4.5 kilobases of the plasmid pPT23D, encoding at least six genes labelled *copABCD.RS* The predicted amino acid sequences of the first four proteins of the *pco* determinant show extensive idenity to the four proteins of the *cop* determinant. Although the *pco* and *cop* determinants are similar genetically, they differ fundamentally in the mechanism of copper resistance. The mechanism of resistance of the *cop* system operates by accumulation/sequestration of copper whereas that of the *pco* system operates as enhanced export of copper from the cell.

A recent study of enteric isolates from 3 piggeries in the U.K. has shown that determinants very closely related to *pco* are present in a number of *Escherichia, Citrobacter and Salmonella* strains. Southern analysis using various portions of the *pco* region as probes demonstrated 1) that there is conservation of the DNA responsible for copper resistance in the Australian (Victoria) and U.K. isolates and 2) that DNA adjacent to the strongly homologous *pco* determinants was not identical. More detailed investigation of the degree of homolgy of the *pco* determinants using PCR analysis show that sequences spanning portions of the genes within the *pco* determinants are probably identical.

Quantitation of the copper resistance levels by survival assays revealed differences in the expression of the copper resistance determinant in the wild-type host and in two derivatives of *E.coli* K12.Recent isolates from Victoria indicate that they carry cryptic plasmid-encoded copper resistance genes.

The data obtained could shed light on both the origin and mode of dissemination of copper resistance determinants in bacteria.

The effects of a retrotransposon insertion on the expression of the *Gpdh* gene in *Drosophila melanogaster*.

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The enzyme *sn*-glycerol-3-phosphate dehydrogenase (GPDH, EC 1.1.1.8.) in *Drosophila melanogaster* is present in three isozymic forms. They all are encoded by the same structural gene and arise through the differential processing of the two exons at the 3'-end of the gene. The isozymes are expressed in a tissue- and life-stage-specific manner.

A unique variant (named *Ky33*) that produces a novel (fourth) isozyme of GPDH and has an altered tissue distribution of isozymes has recently been isolated from a natural population. Detailed studies have shown that there is a large insertion (~ 8 kb) in the non-coding part of exon 7 of the *Ky33* gene. The flanking regions of the insertion have been sequenced and they share a high homology with the LTR of the *blood* retrotransposon, previously described associated with a mutation at the *white* locus. Comprehensive analyses of the *Ky33* variant have shown that the coding regions are unchanged and the insertion, by altering the normal pattern of transcription, is solely responsible for the unusual phenotype.

A model will be proposed that fully explains the manifold effects that the retrotransposon insertion has on the expression of the *Gpdh* gene and also provides a new perspective on the normal control processes.

SURVEY OF MICROSATELLITE REPEATS IN THE PIG (*Sus domestica*) AND THE CHICKEN (*Gallus domesticus*).

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Microsatellites are short tandem repeats found in the genomes of many organisms and display high levels of polymorphism due to variation in repeat number. Repeat units vary in size from mononucleotides up to tetranucleotides and empirical evidence from a variety of species indicates that repeats with multiplicities less than 10 are unlikely to be polymorphic.

DNA sequence databases are rich sources of frequently unrecognised information. A search was therefore made of all pig and chicken sequences in the Genbank database (July 1992) for all 90 possible mono-, di-, tri and tetranucleotide repeats of about 20 bp or greater in length. 13% of pig genes (24/181) and 10% of chicken genes (53/531) contained one or more microsatellites. PCR primers for microsatellite analysis were designed for 19 different pig genes and for 50 different chicken genes. Most of the pig microsatellites and several chicken microsatellites have been analysed and many useful polymorphisms have been detected (see papers and posters by Muladno *et al* and Toye *et al*, this conference). The markers detected have the advantage of being suitable for comparative gene mapping (Type 1 markers), as they are located within known genes, but also are highly polymorphic (Type 2 markers). As a genetic map for the rat of about one hundred loci has been constucted almost entirely from microsatellites derived from similar database searching, it is anticipated that these loci will make a useful contribution to the maps of the pig and the chicken.

Comparison of the spectrum of repeats found in chicken and pig suggest that there may be a difference between birds and mammals. Dinucleotides comprised 30% of the microsatellites found in pigs (cf 28% in humans and 53% in rats), but only 12% in the chicken. AC/GT dinucleotides comprise 21% of the cases found in the pig, whereas they make up only 8% of the cases in the chicken.

A CA REPEAT IN AN INTRON OF CR2 USED TO EXAMINE LINKAGE DISEQUILIBRIUM

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A CA repeat was identified within an intron of the human complement receptor 2 gene (CR2) when it was sequenced from a subclone from a YAC containing part of the regulator of complement activity complex (RCAC) on human chromosome 1q32. The RCAC contains several genes whose products are involved in deactivating the complement cascade on the surface of the bodies own cells. These genes include membrane cofactor protein (MCP, CD46), which is 300 kb from CR2. There is a genetic polymorphism for preference of splicing forms for MCP (1). This polymorphic site is in strong linkage disequilibrium with RFLPs detected with an MCP probe on Southern blots of *Hin*dIII, *Pvu*II or *Bgl*II digested DNA (1). The sites define haplotypesof which only a few of the many possibilities exist. The nearby CA repeat within the CR2 gene has been used as a polymorphic marker to examine the extent of this extremely strong linkage disequilibrium and to further define haplotypes in the region.

Primers were designed to the DNA immediately flanking the 48 bp CA repeat in the CR2 intron to give a PCR product of 98 base pairs. The products of 30 cycles of PCR with 5 sec at 95°C, 2 sec at 72°C, 10 sec at 60°C are highly variable and range in size from 88 bp to 120 bp. PCR product radioactively labelled by end-labelling one of the primers were run on sequencing gels. Ladders of shadow bands at 2 bp intervals decreasing in size which are produced by slippage during PCR can make distinguishing heterozygotes from homozygotes difficult. Densitometry on the bands was used to overcome most difficult typings. Even with densitometry some typings were difficult, especially when a second PCR on PCR product ("bump-up") was necessary to detect product due to inhibitors in the DNA.

1. Wilton, A.N., Johnstone, R.W.. McKenzie, I.F.C. and Purcell, D.F.J. (1992) *Immunogenetics* **36**, 79 - 85.

LOW LEVELS OF RESTRICTION FRAGMENT LENGTH POLYMORPHISM IN CLASS II MAJOR HISTOCOMPATIBILITY COMPLEX GENES IN THE TAMMAR WALLABY

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The major histocompatibility complex (MHC) loci have been shown to be highly polymorphic in most eutherian ("placental") species studied. Several hypotheses have been advanced for the maintenance of this exceptional level of genetic variation, such as selection for disease resistance, mating preferences and maintenance of successful eutherian reproduction. Marsupials (metatherians) and eutherians are the only two orders of viviparous mammals, but their modes of reproduction are quite distinct. Although marsupials have placentae, they are generally shorter lived and less invasive than in eutherians. Genetic variation at marsupial MHC class I loci is probably high. However, weak or non-existent mixed lymphocyte culture (MLC) responses previously reported in several marsupial species have suggested a lack of class II variation. We have therefore gathered data on the level of restriction fragment length polymorphism (RFLP) at MHC class II beta-chain encoding loci of a marsupial, *Macropus eugenii* (the tammar wallaby). This level is shown to be low, between the level of MHC variation found in cheetahs and a population of lions with a restricted genetic base. The apparent difference in level of MHC class II variability between eutherians and marsupials suggests the hypothesis that the higher level in eutherians might be connected to the prolonged contact between maternal and fetal tissues during pregnancy in eutherians.

Enhanced Germ Cell Specific Transcription of *Tctex-1* due to deletion of Repressor Sequences

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Tctex-1 is a small gene family which maps to the *t*-complex of the mouse and is a candidate for one of the factors involved in preferential paternal transmission of *t*-haplotypes. *Tctex-1* encodes a sperm tail component, but is also abundantly expressed in oocytes. *Tctex-1* messenger RNA is overexpressed at least eight fold in testis and oocytes of mice homozygous for the t-complex. The overexpression of the gene is caused by the deletion in one copy of a ten base pair motif in the upstream regulatory region. This motif has striking homology to the *XAP-2* binding site, but does not bind mammalian *AP-2*. The motif retards a band specifically from testis and ovary whole cell extracts on gel mobility shift assays. Deletion of this motif from wild-type promoter/CAT reporter contructs elevates CAT activity in transfected GC-1/spg cells, an immortalized male germ cell line.

Molecular analysis of the genetic controls of meiotic recombination in Neurospora.

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Meiotic recombination in Neurospora is subject to controls that determine the frequency of genetic exchange independently in different parts of the genome. Three *rec* loci are known, each regulating recombination in more than one chromosomal region. In each case the dominant allele blocks recombination. One of the recombinators regulated by *rec-2* has been identified. This is designated *cog* and is located on linkage group I between *ad-3* and *his-3* where it determines the frequency of recombination between the three genes and also between alleles of *his-3*. Two polymorphic alleles of *cog* are known, the dominant allele is a better recombinator. The system has been most recently reviewed by Catcheside[1].

Bowring and Catcheside[2] have shown that *cog* is located 3′ of *his-3*. More recently, we have used RFLP to delimit the region containing *cog* to 0.3kb to 3.5kb 3′ of *his-3* and sequenced both alleles. The sequence differences are substantial, *vide* Yeadon and Catcheside, poster session, this conference.

We have shown that *rec-2* regulates excision of transforming DNA as well as meiotic exchange[3] and is located 5′ of *am* on linkage group V. We are walking from *am* to find *rec-2*, *vide* Bowring and Catcheside, poster session, this conference. Repeat induced point mutation (RIP) which inactivates duplicated DNA during the heterokaryotic phase preceding karyogamy and meiosis by altering up to 30% of C-G base pairs to T-A is not influenced by *rec-2*[3].

1 Catcheside, D.E.A., (1986). A restriction and modification model for the initiation and control of recombination in Neurospora. Genetical Research, **47**: 157-165.

2 Bowring, F.J. and D.E.A. Catcheside, (1991). Mol. Gen. Genet. The initiation site for recombination *cog* is at the 3′ end of the *Neurospora crassa his-3* gene. **229**: 273-277.

3 Bowring, F.J. and D.E.A. Catcheside, (1993). The effect of *rec-2* on repeat-induced point mutation (RIP) and recombination events that excise DNA sequence duplications at the *his-3* locus in *Neurospora crassa*. Current Genetics. (in press).

Cloning of *brainiac;* a neurogenic gene of *Drosophila* which is required for multiple follicle cell functions during oogenesis.

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brainiac (brn) is a newly characterized, X-linked locus of *Drosophila* (map position 5.9) which is required in both the embryo for the correct segregation of neuroblasts from ectodermal cells, and in the female germline to regulate at least three *Drosophila* EGF receptor (DER)-dependent follicle cell activities during oogenesis[1]. Both *brn* and the DER are necessary for the separation of each cyst within a separate follicular epithelium, for the establishment/maintenance of a continuous follicular epithelium, and for the determination of the dorsal-ventral polarity of the ovarian follicle[1]. Thus, the *brn* gene product appears to function in both the neurogenic and tyrosine kinase intercellular signaling pathways.

We have cloned *brn* in order to initiate the molecular analysis of its function(s). The gene maps to the 3F7-8;4A3-6 region of the X-chromosome[1] and we cloned this region by chromosome walking. The location of *brn* within our walk was determined using a combination of deficiency breakpoint mapping, restriction fragment length polymorphism (RFLP) mapping, and RNA rescue experiments. Our preliminary data indicates that *brn* encodes a small molecular weight protein (<30 kDa) with a putative transmembrane domain. (Supported by ACS NP-730D, NIH HD 17608, and T32-GM 07197).

1. S. Goode, D. Wright and A.P. Mahowald (1992). Development 116: 177-192.

Characterisation of a gene associated with expression of self-incompatibility in *Phalaris coerulescens.*

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Abstract

In *Phalaris coerulescens*, gametophytic self-incompatibility is controlled by two unlinked genes, S and Z. A cDNA clone has been identified that shows an RFLP which cosegregates with S genotypes and is a putative S clone. The corresponding gene is about 3 kb in length and is split by 5 introns. Expression of the gene is restricted to mature pollen. Both the putative S_1 and S_2 genes have been isolated and fully sequenced. They show high homology. All variations at the protein level are concentrated at the N-terminus, which is proposed as the region determining allele specificity. There is absolute conservation at the C terminus, which has a secondary structure similar to the thioredoxins.

EVOLUTION OF REGULATORY SEQUENCES CONFERRING SEX SPECIFIC EXPRESSION ON *D.MELANOGASTER EST 6.*

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Regulation of the *D.melanogaster* esterase 6 gene/enzyme (*Est 6*/EST 6) system is constrained by several factors. First, the gene must maintain a generalised expression pattern (as exemplified by haemolymph activity) that is common to many *Drosophila* species. In addition, *Est 6* in *D.melanogaster* and its close relatives has acquired a sex limited expression profile that leads to high levels of enzyme activity in the ejaculatory duct/bulb complex of the male reproductive tract. A second major constraint is posed by the close proximity of *Est P*, a gene which lies only 197bp 3' of *Est 6*. The genes have distinct expression profiles suggesting distinct functions. Here we describe our current understanding of the regulatory mechanisms adopted by the *D.melanogaster Est 6* gene to maintain its ancestral functions and accommodate a newly acquired sex-limited function without comprising the functional integrity of a closely linked gene.

The *Est 6* flanking sequences were subjected to six sequential 5' deletions, three sequential 3' deletions and two modifications of the putative TATA box. The deleted/modified genes were reintroduced into the genome via P-element mediated transformation and the effects on EST 6 activity analysed in five developmental stages and five tissue types, with particular emphasis on haemolymph and male reproductive tract expression. Wildtype levels of EST 6 activity in haemolymph in both larvae and adults can be achieved with only 174bp of 5' and 174bp of 3' flanking DNA. However, additional DNA regions that activate or repress haemolymph activity (two activators, two repressors) are located both 5' and 3' of the coding region. EST 6 activity in the male ejaculatory duct/bulb complex is regulated by four regions that all activate expression, one of which lies 3' of the *Est 6* coding region and overlaps the *Est P* coding region. The three 5' activating regions are located at -174 to -284bp, -284 to -614bp and -614 to -845bp. Histochemical studies revealed that EST 6 activities in the ejaculatory duct and bulb are independently regulated. The -614 to -845bp sequences are essential for ejaculatory duct activity and confer a five-fold change in activity, while the ejaculatory bulb has an essential requirement for the -174 to -284bp sequences.

The results to date suggest that the generalised ancestral function (e.g. haemolymph function in several developmental stages) is regulated by sequences relatively close to the coding region and intra- and inter-specific comparisons indicate that these sequences are highly conserved. In contrast, regulation of the recently acquired male reproductive tract activity occurs via more distant sequences lying both 5' and 3' of the coding region that are more tolerant of nucleotide variation.

Complexities in *Est6* regulation uncovered using chimeric *D. melanogaster* and *D. yakuba Est6* promoters.

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The *Est6* gene of *Drosophila* has undergone major changes in expression between related species, and has therefore potentially changed function over evolutionary time. Given that *Est6* is structurally conserved between related species, it is likely that changes in the regulation of the gene are responsible for the rapid evolution of *Est6* function in *Drosophila*. The aim of this study was to identify the contribution of regulatory DNA variation to the functional evolution of *Est6*. Chimeric promoters were created from *D. melanogaster* and *D. yakuba Est6* by exchanging DNA between shared restriction sites over 1 kb of the 5' promoter. These chimeric promoters were placed upstream of the *LacZ* reporter gene, and expression was analysed in *D. melanogaster* germ-line transformants.

Under the control of either the *D. melanogaster* or *D. yakuba Est6* promoter, *LacZ* was expressed in a wide variety of adult tissues, including some sites where EST6 had previously not been detected. Marked species-specific quantitative variation in *LacZ* expression was observed in some of these tissues. Several tissue-specific differences in *LacZ* expression between the *D. melanogaster* and *D. yakuba Est6* promoters were also identified. In particular, *LacZ* was detected in the adult male ejaculatory duct and ejaculatory bulb of transformants carrying the *D. melanogaster Est6* promoter, whereas the *D. yakuba Est6* promoter drove expression of the *LacZ* gene in fat body and Malpighian tubules.

Analysis of *LacZ* expression in transformants carrying chimeric promoters revealed regulatory complexity. Normal tissue-specific and/or quantitative expression was often disrupted by separating *cis*-sequences, suggesting cooperativity between regulatory elements. For example, expression of *LacZ* in the Malpighian tubules potentially involves two *D. yakuba Est6* promoter sequences separated by 130 bp. Sequences controlling expression in the ejaculatory duct and bulb on the *Est6* promoter of *D. melanogaster* are able to be separated, and a region controlling expression in the ejaculatory bulb was identified. By analysing expression pattern differences using chimeric promoters, it is clear that regulatory signals controlling *Est6* expression in *D. melanogaster* and *D. yakuba* are complex, and may involve general promoter elements associating with tissue-specific or sex-specific regulators.

Artificial selection for development time and the correlated response in the ribosomal genes of Drosophila hydei.

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Natural populations of Drosophila hydei show variation in the number of rDNA repeats containing an insertion sequence. Large numbers of inserted repeats on the X chromosome produce a mutant phenotype (bobbed) which is characterized by short post-scutellar bristles and delayed larval development. In order to test hypotheses about the maintenance of these insertion sequences, strains created from wild populations containing mixtures of bobbed and wild type chromosomes were subjected to selection for both fast and slow development (egg-eclosion time). The correlated responses of bristle length and rDNA insert proportion as determined through southern blots were measured throughout the experiment. The high heritability of development time and the high correlated responses suggest that balancing selection for development time, as opposed to transposition or molecular drive, could be responsible for maintaining Type I insertion sequences in this population.

Evidence that an allele of the *Notch* homologue of the Australian sheep blowfly is an asymmetry modifier.

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In *Lucilia cuprina* (Australian sheep blowfly) field resistance to the organophosphorous (OP) insecticide diazinon is determined by an allele of the *ROP-1* gene. The product of this gene is a carboxylesterase which displays similar substrate specificity and inhibitor sensitivity to those observed for acetylcholinesterase. As well as conferring OP resistance, in the absence of insecticide, there is a reduction in fitness for the resistant heterozygote and homozygote compared with susceptible flies. Further the presence of a resistance allele causes an increase in bristle asymmetry. This is measured as the absolute difference in the total numbers of sensory bristles of three different classes between the left and right halves of the organism.
Persistent use of diazinon after the resistance allele had evolved provided strong selection for a fitness and asymmetry modifier (*M*). This variant has a dominant effect in increasing fitness levels and decreasing asymmetry levels to those observed in susceptible homozygotes.

In this talk we present four lines of genetic evidence that the neurogenic gene *Scalloped wings* (*Scl*), the *Notch* homologue in *L. cuprina*, is the fitness/asymmetry modifier :-

1. *Scl* and *N* mutants display elevated levels of bristle asymmetry compared with wild type.
2. *M* behaves as an allele of *Scl* in reducing the asymmetry associated with *Scl* mutations.
3. *M* behaves as an allele of *Scl* in increasing the severity of the *Scl* phenotype in *Scl/M* heterozygotes, in comparison to *Scl/+* .
4. *Scl* and *Rop-1* interact with respect to *Scl* phenotypes.

A speculative hypothesis which explains the molecular mechanism for the effects of the *ROP-1* product in generating bristle asymmetry, and a variant *Scl* product in modifying it, will also be presented.

PHENOTYPICALLY "NEUTRAL" TRANSLOCATIONS WHICH PREDISPOSE TO ONCOGENIC MUTATION IN HUMAN MALIGNANCY.

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Characteristic chromosome mutations are associated with a wide variety of human malignancies. Such gross mutations often act to promote the malignant phenotype through specific gene deletion or amplification. Deletion may be achieved through whole or partial loss of the relevant chromosome. The former is readily achievable through mitotic non-disjunction, but this carries a disadvantage in that all syntenic loci will also be lost, to the detriment of the evolving malignant cell. A similar argument applies to those mutations involving whole chromosome gain. The latter is well illustrated in the case of human chromosome #1. Trisomy 1 is rarely observed in malignancy, but trisomy 1q, without any concomitant gain of 1p, is very common. This complex event cannot occur in a single step. Three mutations are required, the first being trisomy 1. A second step, seen in myeloid diseases, comprises reciprocal exchange between chromosmes 1 and 7. This produces two novel linkage groups: 1p7q and 1q7p. Molecular studies have shown that this exchange involves the satellite sequences D1Z5 and D7Z1, suggesting no mutation of any expressed gene. This rearrangement is presumably neutral in its effect on cell phenotype. Nonetheless, clonal proliferation ensues, due to the continued presence of trisomy 1q. Loss of 1p7q follows soon after, with progression to an increasingly aggressive malignancy. We here describe an analogous 3-step pathway in an unrelated disease (myxoid liposarcoma) involving satellite sequences of chromosomes 1 and 16. We propose that such "neutral" (ie non-genic) exchanges may be of widespread significance in predisposing to secondary oncogenic mutations of the soma. Insofar as such mutants reduce fitness of the individual, a gross karyotypic form which minimises the somatic development of malignancy may have been favoured during our evolution.

OBSERVED AND IMPLIED ANEUPLOIDY IN HUMAN GERM CELLS

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Germ cell aneuploidy has recently been studied in ejaculated semen and in oocytes which have failed to fertilize in IVF programmes. Both techniques give valuable information but there is a need to exercise caution in the interpretation of the results and extrapolation of the data.

We used F.I.S.H. to simultaneously study aneuploidy of X and Y chromosomes in ejaculated sperm. Our results for aneuploid XX ranged from 0.16%-0.28% in three analyses (total 22,636 cells), YY were constant at 0.21% and aneuploid XY were 0.08%-0.21%. Unpublished results from two other laboratories show lower rates of XX and YY but a similar range of results for XY. Whilst some of the differences may be technically induced, our data suggest that there may be individual differences in aneuploidy rates. Sampling of very infrequent abnormalities can lead to large errors and appropriate statistical evaluation of these data is critical.

Studies of miscarriage and live-births show a highly significant association of trisomy with increasing maternal age. Studies of unfertilized oocytes from IVF programmes have given the only observations of gametic karyotypes in human females and the oocytes studies thus far are few and highly selected.

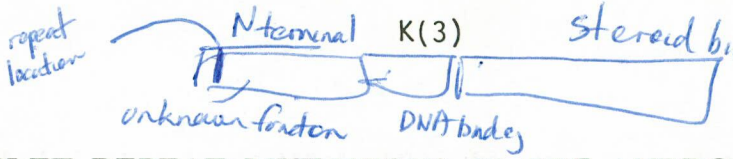
Studies of IVF oocytes have suggested that these do not show an age related increase in aneuploidy. However, appropriate consideration of the age-related rates of pregnancy loss and the karyotypic findings by age, allows an accurate estimate of the expected karyotypic frequencies, by age, at 8 weeks gestation. Comparison with the IVF results shows that if hypoploidy is excluded (because of its early lethality) that the rate of hyperploidy is not different from the expected. There is however an increase in diploid arrested oocytes in IVF which is marginally significant in women under 35 and highly significant in those over 35. This is known to result from excessive ovarian response to gonadotrophins.

Comparison of hyperploidy of individual chromosomes between IVF meiosis II and unselected miscarriage shows that the rate of trisomy 16 is highly significantly reduced in IVF. Possible reasons for this are discussed.

D3:\VFabstGSA

Chrom 16 - most prev. trisomy
- result of maternal error

Testicular feminisation - mutation in androgen receptor



No neurological signs in androgen insensitivity + ♀ hets

TRIPLET REPEAT MUTATIONS IN THE ANDROGEN RECEPTOR GENE CAUSE KENNEDY'S DISEASE

Helen E. MacLean*, Wai-Ting Choi*, Simon Chu*, Garry L. Warne* and Jeffrey D. Zajac# *Centre for Child Growth and Hormone Research, Royal Children's Hospital, Parkville, Vic, 3052 and #Dept. Medicine, University of Melbourne, Royal Melbourne Hospital, Parkville, Vic, 3052.

Kennedy's disease is an X-linked inherited form of motor neurone disease which affects adult males. Symptoms include coarse generalised fasciculations, muscle weakness, facial fasciculations and dysphagia, caused by degeneration of spinal and bulbar motor neurones. The majority of affected males also show a mild degree of gynaecomastia (breast enlargement), and reduced fertility has been reported in some cases. A mutation in exon A of the androgen receptor (AR) gene, which encodes a transcription factor responsive to androgens, has shown to be the cause of all cases of Kennedy's disease. This mutation, an enlargement of a CAG triplet repeat sequence, causes an increase in the number of glutamine residues in the amino-terminal domain of the receptor. We have investigated the AR gene function in 12 patients with Kennedy's disease and their relatives. The male patients are aged between 45 and 67 years, all have gynaecomastia, and all have children. Using the polymerase chain reaction (PCR) to amplify each exon of the AR gene, we have identified the exon A enlargement in all patients with Kennedy's disease. All patients have an enlargement of approximately 90 bp, or 30 CAG repeats. We have confirmed the carrier status of 10 putative heterozygote females, who show no signs of neurological dysfunction, all of whom have one normal gene and one gene carrying the exon A enlargement. We have seen no correlation between the size of the triplet repeat enlargement and the severity of symptoms.

Cultured supra-pubic skin fibroblasts from 8 patients have been assayed for the presence of AR, using the synthetic androgen methyltrienolone (R1881) to measure androgen binding. Results show a significant decrease in the maximal androgen binding levels in 8 Kennedy's patients (mean 7.1 fmol ^{3}HR1881 bound/mg protein) compared to normal controls (mean 13.4 fmol/mg). Scatchard analysis indicates that both the B_{max} and K_d of the AR is lowered in the cultured fibroblasts from patients with Kennedy's disease. The ability of the mutant receptors to regulate transcription of responsive genes is currently being investigated. We are transfecting a MMTV-CAT reporter gene construct, which contains an androgen response element (ARE), into cultured fibroblasts from normal controls, patients with Kennedy's disease, and negative controls (patients with testicular feminisation, who lack a functional AR), to measure the ability of the AR to activate transcription of the CAT gene via the ARE.

The classical role of the AR is to mediate the action of androgens, the male sex hormones, to cause development of the male phenotype. Although we have demonstrated an abnormality in the ability of the AR to bind androgen in cultured genital skin fibroblasts, all patients with Kennedy's disease show normal genital development, and appear to have apparently normal AR function in sex target tissues. However the AR must have another role in the nervous system, involving the maintenance of neuronal survival, and the mutation causing Kennedy's disease must disrupt this role in an as yet to be determined manner.

3' untranslated

Myotonic dystrophy

No X chromosome fragility associated with repeat.

KO - decreased binding of androgen. - however normal masculinisation
- very low affinity for androgen

PROBLEMS IN APPLYING GENETICS IN CONSERVATION: THE NEED FOR A NON-EQUILIBRIUM APPROACH

Craig Moritz, Shane Lavery and Michael Cunningham

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One of the questions most often asked by wildife managers is: "How connected are the populations of a threatened species"? The question of connectivity is particularly relevant where the habitat of the species has been recently fragmented. Even with modern radio or satellite tracking technologies,it is very difficult question to measure interpopulation migration through ecological studies and there have been numerous suggestions that genetic comparisons will provide the solution. However, genetic measures can be slow to respond to changes in demography and the existing distribution of genetic diversity may reflect the long-term more than the recent history of the populations. This is a particular problem for analysis of threatened species because they are particularly likely to have undergone recent changes in population size and connectivity.

In this paper we review two case studies - declining species of coconut crabs in the Indo-Pacific and rainforest skinks in fragmented habitat from the Atherton Tablelands. In each case, the current patterns of variation in mitochondrial DNA appear to be dominated by events occurring some thousands of years ago. For the crabs, the distribution of differences between alleles appears to respond more slowly than the distribution of alleles. One solution to this problem may be to use more rapidly evolving loci such as microsatellites, although the application of such loci to problems in population genetics remains to be explored.

THE POPULATION DYNAMICS OF THE INTERTIDAL ASCIDIAN *PYURA STOLONIFERA* - A GENETIC ANALYSIS

N. Kirk, Division of Botany and Zoology, Australian National University, ACT, 0200.

Naturalists, fishermen and commercial interests have reported a decline in the populations of some littoral and marine species found on the Australian eastern seaboard. The intertidal ascidian, *Pyura stolonifera*, or cunjevoi, has suffered a patchy decline - while remaining common in some locations, its decline has been heavy to complete in others. These observations beg the need for an assessment of the conservation status of this species. Other authorities have noted the absence of genetic studies of philopatric larval dispersers in marine environments.

Allozyme electrophoresis was employed to study the population genetics of *Pyura stolonifera* across a major portion of the species' range in Australia. High levels of polymorphism and heterozygosity were detected. Further, and contrary to the results of some previous work on *P.stolonifera*, unique alleles were detected in highly heterogeneous subpopulations, with high genetic distances between subpopulations. The lack of panmixia inferred by these results was confirmed by geographically unpredictable levels of gene flow among subpopulations. The differences between these results and those of earlier work may be attributed to the superiority of Titan gels (starch gels were used earlier) in facilitating the distinction of alleles for certain loci; and to temporal factors - the observed decline in the numbers of *P.stolonifera*, which would be expected to promote genetic subdivision, has occurred in the last 10-15 years and since the earlier work.

Given these results, a similar study was undertaken at Mystery Bay in southern NSW, using samples from three neighbouring populations that were separated by sandy beaches. A lower level of genetic subdivision was detected.

These results enabled the testing of several supply-side ecology models for dispersal in the intertidal zone. The role of physical oceanographic processes, and the structuring of intertidal communities through competition (with emphasis on solitary versus colonial strategies) were considered. Further non-genetic studies were conducted to test the suitability of ecological models.

This study concluded that *P.stolonifera* employs near-shore oceanographic effects for philopatric dispersal, with a very limited and unpredictable level of panmixia and/or dispersal occurring through off-shore currents. Modelling suggests that *P.stolonifera*, a solitary ascidian, may employ some of the dispersal and recruitment strategies normally peculiar to a colonial animal.

The implications for the conservation of *P.stolonifera*, and for seascape management in general, are clear. The species would not appear to be at general risk, however its depletion in many locations, considered in the context of its philopatric mode of dispersal, is disturbing. This study confirms the utility of genetic analysis in conservation studies of littoral and marine species.

MOLECULAR BIOLOGY AND CONSERVATION GENETICS OF KOALA POPULATIONS.

B.A. Houlden, W.D. Greville, and W.B. Sherwin, School of Biological Sciences, University of NSW, Kensington 2033, Australia.

Koalas (Phascolarctos cinereus) are restricted to living in eucalyptus forests. In recent times the destruction and partitioning of their environment has led to a decline in their numbers and distribution, and in some areas koalas have become locally extinct. A management program has been undertaken which has restocked these areas by relocating animals from other localities. Hoverer, many thousands of animals relocated to southeastern Australia were derived from populations on islands in Westernport bay. These island populations were established from few founders, so genetic variation may be severely reduced throughout many southeastern Australian populations, with the potential for genetic problems.

Prior to this study, there was little information on the genetic variation within and between koala populations. Six southeastern Australian koala populations with different histories have been studied. We will report on our initial investigation of the nature and extent of genetic variation in a selected gene family: the Major Histocompatibility Complex (MHC). The MHC genes determine graft rejection and a number of other principally immunological functions. MHC genes can be found in all mammals, and because of their importance, there is complete gene sequence information for many species, which we have applied to obtain genetic information in koalas.

Study of the MHC is particularly advantageous for a number of reasons. The degree of variation in MHC genes can effect disease resistance, mating success and fertility, which are all of major concern to koala managers. In addition, study of variation in MHC genes is statistically very powerful, because these genes are the most polymorphic genes present in the genome of most mammals. Furthermore, it has been established that quantification of variation in MHC genes correlates very well with estimates of variation obtained by other genetic analysis.

We have utilized conserved regions of MHC gene sequence to design oligonucleotide primers which encompass most of the variable region of the molecule. Using these primers, we have amplified this region using PCR technology, and subcloned these sequences into a plasmid vector. Sequencing has revealed variation among individuals, as expected. Our long-term goal is to attempt to correlate our analysis of genetic variation within and between these populations with environmental and conservation issues relevant to koalas, in order to assist with the future management of koala populations.

Multi-locus theory and intragenic evolution

Ian Franklin
CSIRO Division of Animal Production
PO Box 239 Blacktown NSW 2148

In recent years, population geneticists have used DNA sequencing techniques to study the extent of genetic variability within a locus. A number of surveys of nucleotide diversity around known loci, particularly in *Drosophila*, have revealed that strong non-random associations may exist between nucleotide polymorphisms separated by up to 10 kb or more. Commonly, the patterns of nucleotide diversity and the associations between nucleotides at different sites have been attributed to population bottlenecks and to selective sweeps, or hitchhiking. However, there is ample opportunity for selectively generated disequilibrium between polymorphic sites, and because the recombination distances are so small, the selective forces need not be large. I will show that small interactions between sites within a gene are capable of generating very strong linkage disequilibrium. I will discuss the role of multilocus theory in interpreting gene sequence data and its contribution to our understanding of gene evolution.

The evolution of dominance: a theory whose time is past?

Oliver Mayo

CSIRO Division of Animal Production

PO Box 239

Blacktown NSW 2148

H. A. Orr (1991 *Proc. Natl Acad. Sci.* **88**: 11413-5) has stated that

> "mutations are recessive just as often among haploid as diploid species. This result falsifies Fisher's theory of dominance and provides strong support for the alternative physiological theory."

Is this the case? Does Orr's demonstration of dominance in vegetative diploids of *Chlamydomonas* falsify Fisher's theory (and Haldane's and Plunkett's, as claimed by Orr)? The results are consonant with the predictions of Kacser's metabolic control theory; is this the case for dominance in all of its manifestations?

The talk will address these questions through a consideration of the various theories mentioned above and the experimental results bearing on them.

Inbreeding and extinction

R. Frankham,
School of Biological Sciences, Macquarie University, Sydney, NSW 2109

The fundamental assumption underlying the application of genetics within conservation biology is that inbreeding and loss of genetic variation increase the probability of extinction. While there is a considerable body of evidence that inbreeding depresses components of reproductive fitness in outbreeding species, this cannot readily be extrapolated to extinction. Evidence of cumulative extinction in inbred lines has been interpreted as establishing a relationship between in breeding and extinction. However, control levels of mating failures must be distinguished from the effects of inbreeding. By adjusting for control levels of mating failures, inbreeding was shown to significantly increase rates of extinction in a range of species. Different models relating inbreeding and extinction will be reviewed and their characteristics examined. There has been no prior data presented on the shape of the relationship between the inbreeding and extinction. It will be shown that this can be determined from generation to generation transitions in proportion of inbred lines surviving at different levels of inbreeding. *Drosophila melanogaster*, *D. virilis* and mice all exhibit quadratic relationships between incremental extinction and inbreeding, with little extinction at low levels of inbreeding, and rapidly increasing extinction rates at intermediate level of inbreeding. These results have important implications in conservation biology.

Marsupial ß-globin gene organization and expression.

Steven Cooper, Rachael Murphy, Gaynor Dolman, Guy Webber and Rory Hope.
Department of Genetics, University of Adelaide.

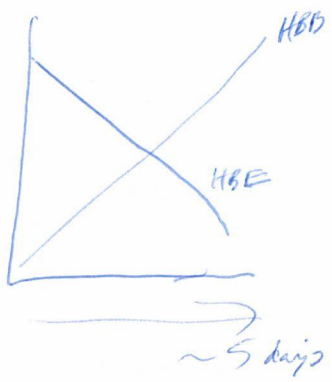
Gene duplication followed by functional and ontological specialization have been the hallmarks of haemoglobin gene evolution. These events have led to the complex arrays of developmentally regulated globin genes found clustered in gene families in extant mammals. The timing of some of these duplication events corresponds approximately to the timing of the separation of the marsupial and eutherian lineages. Therefore, comparative molecular studies of the haemoglobin genes in these two major mammalian groups are likely to be informative in the context of molecular evolution.

There are additional reasons for interest in marsupial and monotreme globins. New born marsupials commence breathing at a very early stage in their development and do so in an environment (the pouch) where respiratory gas levels differ from those experienced by new born eutherian mammals. These and other features of marsupial cardio-respiratory systems are distinct from those which apply to eutherian mammals. Comparative molecular, biochemical and physiological studies of eutherian and marsupial haemoglobin genes and proteins will therefore provide insights into the relationships between gene structure, function and evolution.

While there is a large body of data on the molecular biology of eutherian haemoglobin genes, relatively little work has been done on marsupial globins. We have studied the molecular organization and regulation of ß-globin genes in the marsupial *Sminthopsis crassicaudata*. Interestingly, instead of the five or more genes characteristic of the ß-globin families of many eutherian mammals, only two such genes have been identified in *S. crassicaudata*. One of these genes is expressed in the embryo, the other in the adult. The switch from embryonic to adult gene expression occurs over a period of only several days immediately after birth. With only two genes in the family and a single developmentally regulated switching event, *S. crassicaudata* provides a useful model system for studying the molecular basis of ß-globin gene regulation. Our findings lend support to the hypothesis that only two progenitors of the 5 eutherian ß-globin genes existed at the time of the eutherian/marsupial separation. Other noteworthy features of the ß-globin genes in marsupials (for example their unusually long second introns) will be referred to.

HBE - decreases in exp after birth

HBB - increases



The *Caenorhabditis elegans* Homologue of the Novel *Drosophila flightless-I* Gene: A Study in the Evolution of the Gelsolin Gene Family.

Charles Claudianos [1,2] and Hugh D. Campbell [1].

1. Research School of Biological Sciences, Australian National University,ACT., 0200.

2. Division of Botany and Zoology, Australian National University, ACT., 0200.

It has been observed in eukaryotes that internal duplications in genes and proteins have occurred frequently in evolution. Most have involved one or more domain duplications by polymerization of DNA sequences which are often derived from multiplication of a primordial sequence. The result is a repetitive structure which takes up a major portion of the entire length of the protein. In such cases duplication and other rearrangement events may be traced along a phylogenetic trail among protein families to a common ancestry. The gelsolin family of proteins, which includes the novel *flightless-I* gene of *Drosophila melanogaster*, has provided such a trail.

A molecular analysis has been conducted on a homologous gene to *flightless-I* of *D. melanogaster* in *Caenorhabditis elegans*. Two corresponding cDNAs were isolated from a *C. elegans* cDNA library. DNA sequence analyses of both strands of a 4.6 kilobase cDNA enabled the prediction of the complete amino acid sequence of the protein. An alignment of the predicted *C. elegans* protein of 1,257 amino acids with the *D. melanogaster flightless* protein of 1,256 amino acids reveals a 49% identity, or 69% similarity when conservative amino acid substitutions are considered, over the entire length. Both predicted proteins have a strong sequence similarity to the actin-binding protein gelsolin, and in addition have an amino terminal domain consisting of a repetitive leucine-rich amino acid pattern.

Phylogenetic analyses of the gelsolin family including these *flightless* proteins were conducted. The sequence alignments verify unique protein domain modules related to an ancient protein or protein segment. Phylograms and cladograms place the severin protein of *D. discoideum* at the base of the tree and the vertebrate actin filament capping proteins Mbh1, gCap39, and Mcp as more recent arrivals. The relationship between protein segments established by these methods distinguishes an order of appearance which supports the observation that a combination of internal gene duplications and rearrangements has occurred during protein evolution. It is proposed that at least one domain duplication, two gene module captures and one domain deletion, would be necessary to account for the structural complexity of this related group of proteins.

Imprinting and loss of ABO antigens in leukaemia

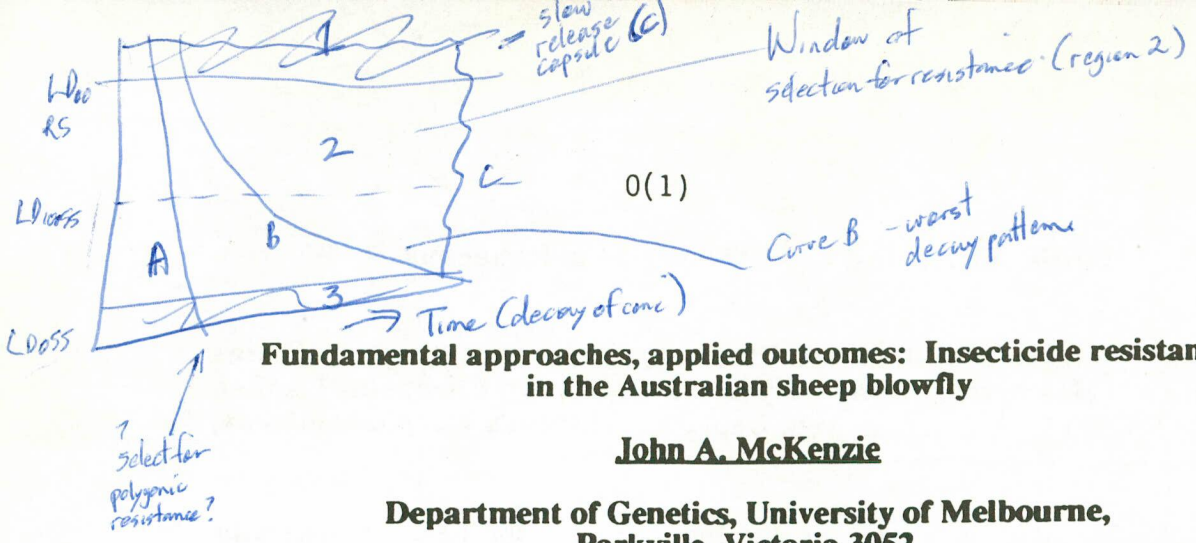
Alexander Dobrovic [1], Denise O'Keefe [1], R. Edward Sage [1], Elaine Batchelder [2]. [1] Haematology/Oncology Dept., The Queen Elizabeth Hospital, Woodville SA 5011, [2] Haematology Dept., St. Vincent's Hospital, Fitzroy, VIC 3065

Loss of function of both alleles of a tumour suppressor gene is generally necessary to abrogate its activity. The first allele is usually inactivated by a mutation but a subsequent mutation in the second allele is considerably rarer than a variety of other events that lead to the physical loss of the second allele in the tumour resulting in loss of heterozygosity of the chromosomal region around the tumour suppressor gene.

For some childhood tumours, the maternal allele is lost in an overwhelming proportion of cases, consistent with imprinting of the tumour suppressor gene. More recently, the Philadelphia translocation has been shown to involve the paternal chromosome 9 and the maternal chromosome 22.

We have been studying the loss of red blood cell ABO antigens which occurs sporadically in patients with haematological malignancy. The changes in ABO expression may be a signpost to genetic changes occurring in the region of chromosome 9 where the ABO gene is located- a region that includes the ABL and other oncogenes. There have been several reports of simultaneous decrease of activity of the enzyme adenylate kinase 1 (AK1) and loss of ABO antigens. Recently, it has been shown that the ABL locus at the 9q34 breakpoint of the Philadelphia translocation maps between the ABO and AK1 loci. We thus decided to investigate if there was any parental influence on the allele that was lost.

Many of the patients that we have obtained information on were elderly and there was accordingly no opportunity to type their parents. We were able to get information on 4 patients. In each of 4 cases, the allele that was lost must have been maternally derived. This suggests that imprinting affects other loci in addition to the ABL locus on chromosome region 9q34 and that this imprinting persists into adulthood, at least in haemopoietic stem cells. No tumour suppressor genes have been reported in 9q34 although the recurrent loss of ABO and AK1 is indicative of one in the region. If such a tumour suppressor were to be imprinted, this would explain the observations presented here.



Fundamental approaches, applied outcomes: Insecticide resistance in the Australian sheep blowfly

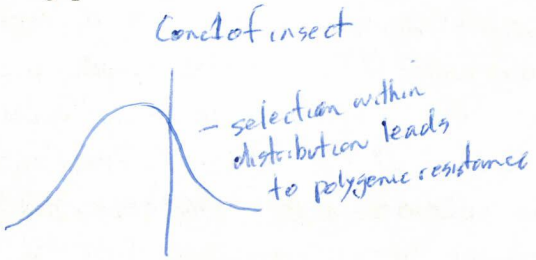
John A. McKenzie

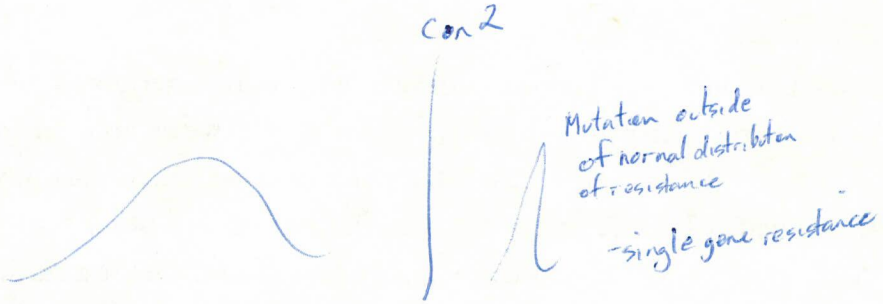
Department of Genetics, University of Melbourne, Parkville, Victoria 3052

Studies of insecticide resistance in the Australian sheep blowfly, *Lucilia cuprina*, have proved useful in defining the forces responsible for genetic change in natural populations and the relationship between the mode of selection and an evolutionary outcome.

The results have enabled models that minimize the probability of the evolution of insecticide resistance to be developed and provide an opportunity to predict the genetic basis of resistance to a new pesticide before resistance evolves in natural populations. In this context, it is feasible to manage susceptibility rather than attempting to manage resistance in the usual pattern of damage control. This leads to a more effective use of pesticides.

The talk will emphasize that studies of pesticide resistance, based on a philosophy of evolutionary genetics, not only have significant benefits for the derivation of pest-management strategies, but also make fundamental contributions to an understanding of the evolutionary process.



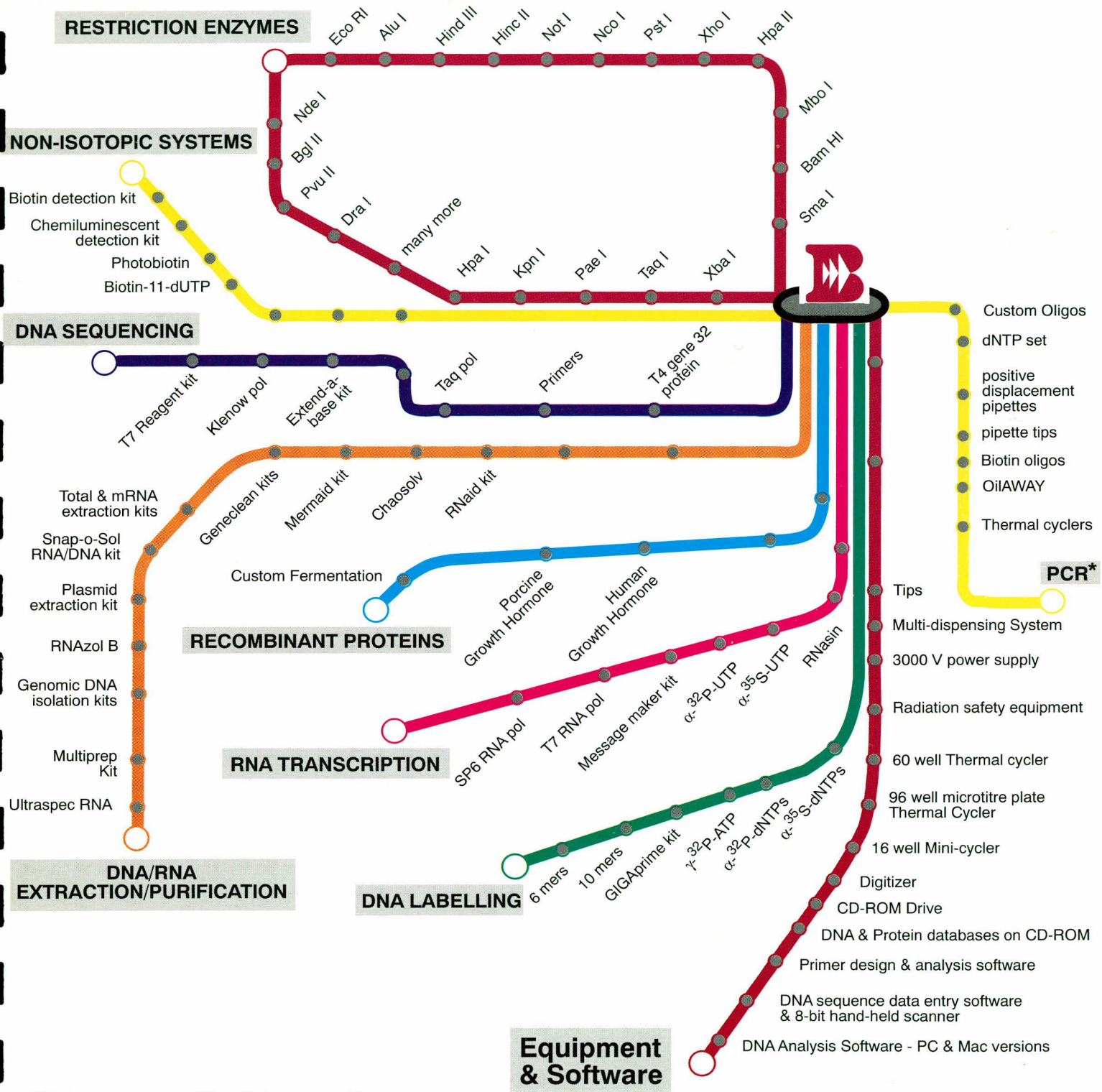


Prob?

Conc 2 always degenerates into conc 1!

Do we see any polygenic resistance??

Fast Track Your Research!



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* PCR is covered by US patent no. 4,683,202 isued to Cetus Corp.

POSTERS

Poster 1

Ancient DNA and Australian prehistory: Preliminary analysis of DNA sequences derived from sub-fossil Australian Aboriginal bone.

Greg Adcock[1,2] and Thomas H. Loy[1]

1. Department of Prehistory, Research School of Pacific Studies, Australian National University, Canberra, A.C.T., 2601
2. CSIRO Division of Plant Industry, Black Mountain, A.C.T. 2601

What are the origins of the people who have inhabited this continent for more than 40,000 years, and how have these people, known only from their skeletal remains and cultural artefacts, contributed genetically to future generations? The population genetics of modern people and skeletal analysis of archaeological finds offer some clues to such problems, but they are indirect and provide sometimes ambiguous data. Comparison of the DNA sequences directly from relevant groups, both ancient and modern, hold the promise of being a fruitful approach.

DNA has been extracted from 10 individuals 500 to 20,000 years old, as 1g bone samples of varying preservation quality. Methods were developed to maximise yield and reduce the chances of contamination during extraction and curatorial practices. The heavily degraded DNA was treated with anion exchange purification methods to remove factors which inhibit PCR amplification. A PCR strategy has been designed which amplifies ancient mitochondrial D-loop DNA (mtDNA) and which minimises contamination problems. The resulting PCR products have been sequenced directly. Contaminating sequences were recognised as being derived from carryover from a modern positive PCR reaction control. Elimination of this control, the adoption of new primers and increased ultraviolet pre-treatment of PCR reagents resulted in the negative controls showing no amplified products. The sequences of mtDNA amplified from some of the ancient individuals under study have been compared with the extensive data-base available for the region. At polymorphic sites within the D-loop, base differences were observed and found to be consistent with those present in Aboriginal Australians or native New Guineans but none were uniquely European.

Having established the correct method to amplify and sequence DNA from ancient bones, it will now be possible to enlarge the sample and directly investigate specific questions about the human genetic history of Australian Aboriginal peoples.

Poster 2

MOLECULAR GENETICS AND DEMOGRAPHIC MODELLING IN MANAGEMENT OF AN ENDANGERED SPECIES: THE NORTHERN HAIRY-NOSED WOMBAT.

Deryn Alpers and Bill Sherwin
University of NSW

Less than 100 individuals remain of the species Lasiorhinus krefftii (the Northern Hairy-nosed Wombat), all of which exist as a single colony at Epping Forest National Park in central Queensland. Small populations are at risk of losing genetic variability through inbreeding. Adverse consequences of reduced genetic diversity, for a population, include lowered fertility, survivorship and disease resistance. To aid the management of this species both molecular techniques and computer modelling have been used to investigate the genetic and survival potential remaining in the population.

Molecular genetics potentially can provide information on the general level of genetic variation within the species, as well as identification of individual wombats via genetic marker systems. In a related study, Taylor et al., investigated levels of genetic variation present in L. krefftii using simple sequence polymorphisms. The present study investigates male-specific polymorhpism with the aim of mapping paternal lineages for pedigree information. Good pedigree data would be of obvious benefit to close management protocols.

The establishment of a second colony, to reduce the risk of single catastrophe effects, is currently under investigation. Computer modelling programs are being used to ascertain the potential genetic and demographic problems involved in this proposal. Using available knowledge of the biology of this species, simulated populations are modelled and tested under different conditions. Various rates of harvesting are used in an attempt to establish the optimal transfer rate of animals from the original to the second colony.

Poster 3

Characterisation of DNA Polymerase 1 in the marine bacterium *Vibrio natriegens*.

Z.M. Baxter and S. M. Thomas
School of Biological Sciences, Flinders University of South Australia, GPO Box 2100 Adelaide 5001.

Marine bacteria play a fundamental role in nutrient cycling, trophic production and the transmission of disease. Despite their key role in the environment, comparitively little is known about their genetics. This investigation is examining the DNA polymerase genes of marine bacteria, and specifically those of *Vibrio natriegens*. This organism is abundant in temperate waters and is able to replicate rapidly, hence it may be a useful laboratory tool. The project aims to compare the activity and structure of the *V. natriegens* DNA polymerase I gene to the functionally similar *Escherichia coli* DNA polymerase I gene. An existing functional assay for DNA polymerase activity has been modified for use with a non-isotopic label, and adapted for use with *V. natriegens*. Qualitative comparisons of the DNA polymerase I activity of both species suggest that *V.natriegens* polymerase I is active at a narrower pH and salt range than *E.coli* polymerase I and that it is active between the temperature ranges 25-42°C, whereas *E.coli* polymerase (from K12 strain AB1157) shows no detectable activity in the assay at 25°C. This qualitative assay will form the basis of a strategy to isolate the *V.natriegens* DNA polymerase I gene, which will then allow quantitative characterisation.

The search for *rec-2*: a gene that modulates the level of meiotic recombination in *Neurospora crassa*.

F.J. Bowring and D.E.A. Catcheside.

School of Biological Sciences, Flinders University, GPO Box 2100, Adelaide 5001, South Australia.

In *Neurospora crassa*, a number of genes that alter the frequency of meiotic recombination in various genomic regions (*rec* genes) have been identified. *rec-2*, one of the three known genes in this class, controls both the frequency of recombination between alleles at *his-3* and crossing over at three other remote loci[1]. Two naturally occurring alleles of *rec-2* have been identified: *rec-2* and *rec-2*$^+$, the latter being a completely dominant suppressor of recombination at the target loci. The presence of *rec-2*$^+$ in a cross leads to a thirty three fold reduction in recombination between *his-3* alleles compared to levels observed in crosses homozygous *rec-2*^{2}. *rec-2* has been mapped to linkage group V (LGV) between the *am* and *spray* loci.

We are currently walking out from *am* in a *rec-2*$^+$ cosmid library using a number of LGV markers to assess our progress. The identification of clones containing *sp* will define the region of LGV containing *rec-2*$^+$. Clones identified in this manner are then assayed for *rec-2*$^+$.

It has recently been demonstrated that duplicated DNA in *N. crassa* is subjected to hyper-mutation after the commitment to meiosis but prior to nuclear fusion[3]. This phenomenon, termed repeat-induced point mutation (RIP), complicates the assay for *rec-2*. The introduction of cloned DNA containing *rec-2*$^+$ into a *rec-2* strain may not lead to the expected reduction of recombination at *his-3* since there is the likelihood that a duplication is created and the possibility that this may be RIPed. Thus, we are also looking for relaxed recombination as a result of RIP in a *rec-2*$^+$ strain transformed with putative *rec-2*$^+$ clones.

1 Catcheside, D.G., (1975). Regulation of genetic recombination in *Neurospora crassa*. In *The eukaryote chromosome*. W.J. Peacock & R.D. Brock (eds.). Canberra, Australian National University Press.

2 Angel, T., B. Austin & D.G. Catcheside, (1970). Regulation of recombination at the *his-3* locus in *Neurospora*. Aust. J. Biol Sci., **23**: 1229-1240.

3 Cambareri, E.B., B.C. Jensen, E. Schabtach, and E.U. Selker, (1989). Repeat-induced G-C to A-T mutations in *Neurospora*. Science, **244**: 1571-1575.

ORIGINS OF POLYMORPHISM AT A POLYPURINE HYPERVARIABLE LOCUS

H M Brereton and D R Turner

Haematology Unit, School of Medicine, Flinders University, Bedford Park, South Australia 5042.

We have located a new and unusual hypervariable locus, D8S210, in the telomeric region of the short arm of human chromosome 8. The locus is highly polymorphic, with alleles varying in size from 1.8 kb to 24 kb and an observed heterozygosity >99%. It resembles a minisatellite locus having a tandemly repeated sequence in its variable length region. Alleles are inherited in a Mendelian manner and one mutation to a new length allele has been observed in the analysis of 51 meioses. We have also detected a more ancient mutation, a common deletion of single copy DNA between the repetitive region and a flanking Alu-family sequence, in 5 / 73 unrelated individuals we have studied. This locus is particularly unusual, however, because the DNA of its repetitive region is entirely polypurine on one strand and polypyrimidine on the other, with a tetranucleotide repeating unit GGAA at the margins and diverged versions of this motif internally. As some alleles extend more than 20 kb, it is the longest polypurine·polypyrimidine region yet described. We present evidence that the polypurine region can adopt triplex conformations *in vitro*. Such structures *in vivo* may facilitate loss or gain of unique sequences in the genome, contribute to mutation at conformation transition points and drive the hypervariability of this locus.

Strain Identification of Lactic Acid Bacteria using Genetic Fingerprints Generated by Arbitrarily-Primed PCR

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Genetic "fingerprints" of organisms can be produced by PCR using a single arbitrarily-chosen primer under low-stringency conditions. This strategy (known as AP-PCR [1] or RAPD [2]) has been applied to strain typing of both prokaryotes and eukaryotes. Strain identification by AP-PCR requires no genetic or phenotypic knowledge of the strain, making it particularly useful in studies of poorly-characterized organisms.

We have previously reported an AP-PCR method that uses a fluorescently-labelled primer [3], permitting separation and detection of amplification products using equipment and software commonly used for automated DNA sequencing. We have now applied AP-PCR to studies of strains of *Streptococcus thermophilus* and *Lactobacillus plantarum*, important lactic acid bacteria in dairy food fermentations. *L. plantarum* isolates were readily distinguished by this method, as they showed only some similarities. AP-PCR amplification profiles of *S. thermophilus* isolates were difficult to distinguish, indicating close genetic relationships.

[1] J.Welsh and M.McClellend (1990). Fingerprinting genomes using PCR with arbitrary primers. Nucleic Acids Res. **18:** 7213-7218.

[2] J.G.K.Williams, A.R.Kubelik, K.J. Livak, J.A.Rafalski and S.V. Tingey (1990). DNA polymorphisms amplified by arbitrary primers are useful as genetic markers. Nucleic Acids Res. **18:** 6531-6535.

[3] M.R.Cancilla, I.B.Powell, A.J.Hillier and B.E.Davidson (1992). Rapid genomic fingerprinting of *Lactococcus lactis* strains by arbitrarily primed polymerase chain reaction with ^{32}P and fluorescent labels. Appl. Environ. Microbiol. **58:** 1772-1775.

Poster 7

Cytoplasmic incompatibility and sex ratio distortion in insects infected with *Wolbachia pipientis*: mode of spread, host mtDNA evolution and work toward pest and disease control.

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Abstract: *Wolbachia pipientis* is a maternally inherited Rickettsia-like microorganism which infects at least 24 species of insect across 7 orders and is suspected in several more. Geographic distribution of infection varies according to host from localised to pan-continental. It can cause male sterility in insects with obligate sexual reproduction, or sex-ratio distortion in those which display facultative parthenogenesis. The dynamics of spread through populations is discussed as is the concomitant reduction of host cytoplasmic variation. Knowledge to date is summarized and work toward biological control of insect pests, involving intra- and interspecific transfer of infection, is presented.

CHARACTERISATION OF "EXPRESSED SEQUENCE TAGS" FROM A MARSUPIAL MAMMARY GLAND LIBRARY

Chris Collet and Ros Joseph

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In comparison to eutherian mammals, there is a paucity of gene mapping data for marsupials. The adoption of the tammar wallaby (*Macropus eugenii*) as the model marsupial organism in genetic, molecular biological and physiological studies coupled with the ability to manipulate the tammar's reproductive cycle permits more extensive gene mapping analysis to be undertaken. The *in situ* hybridisation of eutherian probes to the tammar chromosomes has enabled the localisation of a number of highly conserved genes. The use of heterologous probes is in itself problemmatical as, under reduced conditions of stringency, small regions of local similarity may also bind the probe. With improved automated DNA sequencing methodologies becoming available, an alternative approach for mapping genes in higher eukaryotes utilises the sequencing of expressed sequence tags (ESTs), or random pieces of cloned cDNA, as a means of rapid gene isolation and identification. The characterisation of expressed human gene sequences will prove to be an important adjunct to the Human Genome Project in providing, not only gene localisations, but also entry points for chromosomal walking and data on coding regions. Unidentified ESTs are also useful for making gene maps as these sequences will be classified by similarity to genes from other organisms as those sequences become available.

As a means of increasing the number of genes that can be physically mapped to the tammar genome, the generation and characterisation of ESTs from a lactating mammary gland has been undertaken in our laboratory. This poster presents the results of a limited sequence characterisation of ESTs from a tammar mammary gland cDNA library and the identification of sequences encoding two caseins, ß-galactosidase, acetyl CoA synthetase, lipoprotein lipase, cytochrome c oxidase I and an ATP-dependent RNA helicase. The use of ESTs for mapping purposes appears a viable approach in those organisms not as well studied or funded as is Man, yeast, *Drosophila* or *Caenorhabditis*.

A MOLECULAR ASSESSMENT OF THE TAXONOMIC STATUS OF COX'S SANDPIPER (*Calidris paramelanotos*)

K. Davies[1], L. Chrisitdis[2] and M. Westerman[1].

Cox's Sandpiper (*Calidris paramelanotos*) is an extremely rare migratory wading bird, which is believed to breed within the Arctic circle but is known only from individuals overwintering in southern Australia. The first Cox's Sandpiper was collected in 1975 at Saint Vincents Gulf, South Australia by John Cox (Cox, 1976). Subsequent morphological analyses have suggested that the Cox's Sandpiper may in fact be either an aberrant Pectoral Sandpiper (*C. melanotos*), or even a hybrid between the Pectoral Sandpiper and Curlew Sandpiper (*C. ferruginea*).

Data obtained from morphological and allozyme studies have failed to unequivocally determine the taxonomic status of Cox's Sandpiper. In order to differentiate between these three possibilities, a 288 base pair fragment of the mitochondrial cytochrome *b* gene was sequenced from individuals of Cox's Sandpiper, Pectoral Sandpiper and Curlew Sandpiper. The results of this study of mitochondrial sequences have allowed us to resolve the taxonomic status of this bird.

Cox, J.B. (1976) . The Pectoral Sandpiper: an unusual specimen. *South Australian Ornithologist* **27**:110-111.

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MOLECULAR PHYLOGENY OF THE GALLIFORM BIRDS - EVIDENCE FROM MITOCHONDRIAL DNA SEQUENCE

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We investigated the phylogenetic relationships of the mound-building birds, the megapodes- a group from the Australian region which incubate their eggs in mounds of composting vegetation or volcanically warmed soils. DNA/DNA hybridisation studies suggest that they are related to the South American group, the curassows and that both of the these groups are related to the gallinaceous birds e.g., the chicken, pheasant, quails etc. This indicates a Gondwanan origin for the megapodes and the curassows. However, the validity of the DNA/DNA hybridisation results has been challenged (Sarich *et al.* 1989 Cladistics 5, 3-32). Here we test the alternative hypotheses of galliform phylogeny, those based on the DNA/DNA hybridisation studies and morphological evidence, with nucleotide sequences from the mitochondrial cytochrome b gene. The results of maximum parsimony analysis of these sequences will be presented.

PCR Products Can Be Directly Sequenced Without Intermediate Purification

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One of the most rapid, cost-effective methods of generating sequence data is to sequence the double-stranded PCR fragments that are generated by the polymerase chain reaction (PCR). Initial problems with the sequencing of double-stranded PCR products have been largely overcome by carrying out the sequencing reactions in a PCR-like manner (1), i.e. using one of the PCR primers (or an internal primer), *Taq* DNA polymerase and thermal cycling. The most labour-intensive step in this procedure is the purification of the PCR product prior to carrying out the sequencing reactions. The similarity between the initial PCR reaction with the PCR-like sequencing reactions (1) prompted us to compare their constituents. This revealed that the only PCR reactants that were incompatible with the PCR-like sequencing reactions were the PCR primers. The presence of multiple primers during the sequencing reaction would lead to multiple populations of teminated DNA fragments.

The recent report that S1 nuclease is active at neutral pH in the presence of Mg^{2+} (2) prompted us to assess the usefulness of such nucleases for the removal of PCR primers from PCR reactions. In the present study, both undigested and mung bean nuclease-digested PCR products were directly sequenced using the PCR-like sequencing reactions described in (1), and the accuracy determined by comparison with the authentic sequence. When undigested PCR product was used as the template in the sequencing reaction, the sequence generated showed only 90% homology compared to the authentic sequence. The presence of the non-sequencing primer at approximately 7 nM (sequencing primer at 160 nM) presumably resulted in a competing sequencing reaction that contributed to the error-calling. However, when mung bean nuclease-digested PCR product was used as the template, up to 300 bases of highly accurate sequence (99% homology) were obtained. This demonstrates that mung bean nuclease can be used to generate sequencing templates from PCR reaction products. No intermediate purification of the PCR product is necessary. The procedure can be carried out completely in a thermal cycler and thus greatly reduces the amount of sample manipulation needed to generate sequencing templates from PCR products. It can be easily adapted to both amplify and sequence DNA fragments in a single tube, and thus may have applications for genome sequencing.

REFERENCES

1. Carothers, A.M., Urlaub, G., Mucha, J., Grunberger, D. and Chasin, L.A. (1989) *BioTechniques*, **7**, 494-499.
2. Esteban, J.A., Salas, M. and Blanco, L. (1992) *Nucleic Acids Res.*, **20**, 4932.

Poster 12

SPECIES IDENTIFICATION AND SEX DETERMINATION USING PCR

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Species identification of a forensic sample is presently carried out by the Ouchterlony immunodiffusion technique. This technique takes two days and requires a separate antibody for each species. A PCR method has been proposed which identifies all species in one reaction and takes only one day. The PCR primers are complementary to conserved regions in the 28S ribosomal RNA gene and flank a region variable in length which is used to identify different species.

Another use for PCR in forensic science is sex determination. This method uses two sets of primers, one set complementary to a region of the testis-determining factor (TDF) gene and the other complementary to the glucose 6-phosphate dehydrogenase (G6PD) gene. The TDF gene is located on the Y chromosome and is believed to be responsible for phenotypic maleness. The G6PD gene is X-linked. The products are analyzed by electrophoresis, with male samples producing two products and female samples only one.

THE CHARACTERISATION OF AN *Aspergillus nidulans* STRAIN, DEFECTIVE IN THE PRODUCTION OF EXTRACELLULAR PROTEASES UNDER CERTAIN GROWTH CONDITIONS

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A number of systems of wide domain regulation exist in *A. nidulans* which control the synthesis of enzymes involved in nitrogen, carbon, sulphur & phosphorus nutrition. One of these enzymes, extracellular protease, is secreted when the preferred carbon, nitrogen or sulphur source is limiting in the growth media.

A strain, defective in the secretion of extracellular protease under nitrogen-limiting conditions, was found to be defective in the secretion of extracellular protease under carbon - limiting conditions, however normal extracellular protease levels were detected under sulphur - limiting growth conditions. The phenotype of this mutant strain appears to be due to a single mutation, designated *xprE1*, which has been mapped by haploidisation studies to linkage group VI.

The structural gene for one of the *A. nidulans* extracellular proteases has been isolated. This gene has been used to examine alkaline extracellular protease mRNA levels in an *xprE1* strain grown under various growth conditions. The results indicate that the protease enzyme levels observed in the assays reflect protease transcript levels.

It is tentatively suggested that *xprE1* is a regulatory mutant which, in diploid studies, is recessive to its wild-type allele, indicating a positive mode of action. Screening of an *A. nidulans* genomic cosmid library is being undertaken at the moment in the hope of isolating the *xprE* gene by complementation of the *xprE1* mutation. Cosmid DNA - mediated co-transformation of an *argB*$^-$ strain containing the *xprE1* mutation is the technique chosen for this screaning.

MOLECULAR CHARACTERISATION OF EARTHWORM SPECIES & STRAINS IN SOUTHERN AUSTRALIAN SOILS

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Earthworm fauna in Southern Australian Soils

The potential for earthworms to improve soil structure and fertility is well established. The existing fauna in Australian agricultural soils is dominated by accidentally introduced species, mostly from Europe. The goal of this research is to use molecular techniques to identify exotic earthworm species and to determine if different strains of these species occur in the soils of Southern Australia. It is ultimately intended to trace the origins of these strains of exotic earthworms back to their native (likely European) distributions. It is desired to establish methods for screening earthworm populations which may be introduced into local soils in the future, so as to select for. or maximise their potential agronomic value.

Species identification

Most species can be identified by their morphology. Two, namely *Aporrectodea trapezoides (At)*, and *A. caliginosa (Ac)*, are difficult to distinguish, particularly in juvenile form, from their external morphology (they were previously considered to be different morphs of the same species). These species are also amongst the most common of all the introduced species. We have prepared repetitive sequence libraries from both *At* and *Ac* and have located clones which, on limited testing, have proven to be diagnostic of these very closely related species.

Strain identification

Aporrectodea trapezoides (At) and *A. caliginosa (Ac)*, as well as other introduced earthworms, are presently being evaluated for strain differences. Preliminary data indicates that different strains of these exotic earthworms exist in South Australian soils, and this could be evidence of their differing (European) origins. These conclusions are drawn from molecular analyses based upon the polymerase chain reaction (PCR). This includes random-priming (RAPD) PCR analyses as well as analysis of intergenic rRNA gene regions. Work is currently in progress to characterise in detail the differences between these strains based on these tests.

Molecular and Genetical Analysis of the *Scalloped wings* Gene of *Lucilia cuprina*.

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Previous work has shown that the *Scalloped wings (Scl)* gene of *Lucilia cuprina* is homologous to the *Notch (N)* gene of *Drosophila melanogaster*. To further examine this association a total of seven new *Scl* mutants were isolated by EMS and gamma ray mutagenesis. These have been subjected to detailed complementation, recombination and phenotypic analysis (in embryos and adults). One of the EMS induced mutants is of particular interest being a mutation of the *Abruptex* class. This mutant, in addition to another *Scl* weak allele (Scl^5), have been useful in examining the phenotypic interaction between *Scl* and a fitness/asymmetry modifier of diazinon (*Modifier*). In each case flies with a *Scl/Modifier* genotype have a more extreme phenotype than flies with a *Scl/+* genotype (see also Davies *et al.*, Platform presentation).
Current data on the level of sequence homology between *Scl* and *N* will also be presented.

Further evidence for differences in recombination frequency between the sexes of marsupial species

David Hayman and Meredith Smith

Sex differences in recombination frequency between the sexes of *Sminthopsis crassicaudata* are associated with differences in the location of chiasmata at meiosis. Studies of chiasmata distribution in additional marsupial species have shown a pattern of presence or absence of such differences between the sexes. The poster summarises our present knowledge of this pattern.

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Isolation of the *sex-lethal* gene of the Australian sheep blowfly, *Lucilia cuprina*

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Manipulation of the sex-ratio of a field population of insects is one way to implement modern biological control over a target population. Many of the genes involved in sex-determination in *Drosophila melanogaster* have been cloned and characterized and the pathway controlling somatic sexual development in this insect is well understood. We have used this information to clone a section of the *Sex-lethal* (*Sxl*) gene from the Australian sheep-blowfly, *L. cuprina*. In *D. melanogaster*, *Sxl* is a major regulatory gene controlling the processes of somatic and germ-line sexual differentiation and dosage compensation (1).

Initially, using primers designed to the ribonucleoprotein consensus sequences of the *D. melanogaster Sxl* gene, we were able to amplify, using PCR, a 290bp fragment from first strand cDNA prepared from adult female *L. cuprina*. Sequencing of this fragment revealed that it contained sequences 73% similar to the corresponding region of the *D. melanogaster Sxl* gene. Translation of the putative open reading frame showed a peptide highly similar to the corresponding region of the *D. melanogaster* Sxl peptide. Translation of this ORF also revealed a codon bias similar to that reported for *L. cuprina* and different to that established for *D. melanogaster*. Southern blot analysis performed under high stringency conditions to genomic DNA prepared from *D. melanogaster* and *L. cuprina* confirmed that this amplified fragment was present in the *L. cuprina* genome as a single copy. No hybridization to *D. melanogaster* genomic DNA was observed under these hybridization conditions. This fragment was then used to screen a cDNA library prepared from *L cuprina* late pupae. Four identical positive clones 1.2kb in size were obtained and sequencing of one of these has revealed it contains the distal half of the *Sxl* gene of *L. cuprina*. The organization of the *L. cuprina Sxl* gene supports the pattern of transcription observed for *D. melanogaster Sxl* gene in later life stages (2). Work is in progress to clone the proximal portion of the *L. cuprina Sxl* gene and to determine whether this gene is differentially regulated between the sexes. We are also in the process of isolating other *L. cuprina* genes involved in sexual development.

1. Belote, J. M. (1992), *Seminars in Developmental Biology*, **3**; 319-330.
2. Samuels, M. E., Schedl, P, and Cline, T. W. (1991), *Molecular and Cellular Biology*, **11**; 3584-3602

Poster 18

GENOMIC AND GENETIC CHARACTERISATION OF THE CYCLIN E GENE OF *DROSOPHILA MELANOGASTER*

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G1 cyclins are regulatory proteins involved in control of the cell cycle at the G1 to S phase transition. They associate, as regulatory subunits, with members of the Cdk family of serine/threonine protein kinases. Candidate metazoan G1 cyclins include cyclin C, D and E. Cyclin E is an excellent candidate for a rate limiting regulator of the G1 to S phase transition, on the basis of its expression and associated kinase activity at the G1 to S phase boundary. We have recently isolated a *Drosophila* cyclin E homolog. *Drosophila* provides an ideal system to investigate the role of cyclin E in the regulation of cell proliferation during development.

Drosophila cyclin E (*DmcycE*) has been mapped to region 35D on the left arm of chromosome two. This region is genetically well characterized, as it is near the *Alcohol dehydrogenase* gene (*Adh*) which maps to 35B. The existence of a number of deficiencies and lethal alleles in this region gave us a starting point to more precisely locate the *DmcycE* gene with the aim of identifying a mutation in the gene. Southern analysis of the available deficiency strains revealed that *DmcycE* maps to a new genetic locus between the 35Da and *snail* loci. P element and EMS mutagenesis studies are currently underway to generate a specific *DmcycE* mutation.

The isolation of cosmid clones derived from the *DmcycE* region and Southern analysis, using cDNA clones, has revealed that the gene is complex. The gene covers at least 20 kb of sequence and contains many introns. The intron/exon boundaries and the 3' end of the two types of *DmcycE* transcripts have been determined by sequence analysis. The location of the putative 5' ends of the gene have also been detemined. This analysis has demonstrated that the two alternative 5' domains of the gene share a 3' acceptor splice site for a common 3' domain.

Poster 19

A TWO-COLOUR *IN SITU* HYBRIDISATION STUDY OF mRNAs IN TAMMAR WALLABY MAMMARY GLAND

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Eutherian milks vary greatly in composition between species but change only slightly with respect to the concentration of the various constituents during lactation. Marsupial milk, however, changes in composition to reflect the developmental stage and nutritional requirements of the pouch young. The tammar wallaby (*Macropus eugenii*) produces two types of milk, early and late phase, which vary significantly in composition. The changeover to late phase milk is characterised by the appearance of a stage-specific protein, late lactation protein (LLP).

This poster presents an *in situ* hybridisation study of the glandular and cellular distribution of the mRNAs encoding the major milk proteins in the early and late phases of lactation in the tammar. Glandular or lobular specialisation is a possibility given that induction of gene expression of the four major milk proteins, α-lactalbumin, ß-lactoglobulin and α- and ß-casein, is under different regimes of hormonal control. Of particular interest is the distribution of cells synthesising LLP. A novel technique was developed using oligonucleotide probes labelled with different fluorescent tags such that application of different wavelengths of UV allowed the direct visualisation of different mRNA species within the same cell.

Our results show that all epithelial cells are producing the same suite of four milk proteins mRNAs, α-lactalbumin, β-lactoglobulin, α-casein and β-casein, during the early phase of lactation. In late phase lactation, the cells producing these four milk proteins are also synthesising LLP. This argues against a completely new population of secretory cells arising to produce late phase milk or LLP. The results support the hypothesis that milk secretion is an all or none process with all epithelial cells in a given alveolus producing the same suite of milk proteins.

Poster 20

The avian order Pelicaniformes: monophyletic or polyphyletic?

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The Pelicaniformes are a diverse order comprising the pelicans, gannets and boobies, darters and cormorants, tropicbirds and frigatebirds. Despite the apparent morphological heterogeneity, several key characters unite the group, particularly the totipalmate foot (having all four toes connected by a web), absence of an incubation patch and location of the salt-excreting gland. All morpholigical studies, whether based on internal or external characters, confirm the monophyly of the group. However, recent DNA-DNA hybridization data suggest the group is polyphyletic, and have split its component lineages among the penguins, procellariformes, egrets, herons, storks, loons and grebes. Further, the DNA-DNA hybridization data suggest that all these groups are part of a single order (termed the Ciconiiformes) which also includes the birds of prey.

Two important implications of the hybridization data are that morphological change in birds can be extremely rapid and major, and secondly that convergence can be quite dramatic in birds if all the supposed unique features linking the Pelicaniformes have in fact evolved independently on several ocasions. Given the controversial nature of the DNA-DNA hybridization data, we have been conducting a series of DNA sequence-based studies to assesss the validity of the Ciconiiformes (*sensu* Sibley and Ahlquist, 1990). Part of this study involves sequencing the mitochondrial cytochrome *b* gene to establish whether the Pelicaniformes are monophyletic or polyphyletic. The results of this study are presented and discussed in connection with patterns of morphological change and adaptation.

Sibley, C. and J. Ahlquist (1990). Phylogeny and classification of birds: a study in molecular phylogeny. Yale University Press, New Haven

Putting a finger on *amdS* regulation by the *amdA* gene of *Aspergillus nidulans*. **Robyn Lints, Meryl Davis and Michael J. Hynes, University of Melbourne.**

Semi-dominant mutations in the *amdA* gene lead to elevated expression of *amdS* encoding acetamidase. These mutations also cause constitutive expression of the acetate inducible *aciA* gene. The *amdA* gene has been cloned by a chromosome walk between the *gatA* and *alcC* genes on chromosome VII. *amdA* was localized by complementation of a loss-of-function mutation and hybridization analysis of an *amdA* deletion mutation. The gene encodes a protein with a C_2H_2 zinc finger motif at the N-terminal end. The *cis*-acting mutations, *amdI66* and *amdI666*, specifically affect *amdA* regulation of *amdS*. These mutations define an 18 bp GA-rich sequence. A similar sequence is found in the 5' region of *aciA*. Analysis of deletions of the *amdS* 5' sequence using an *amdS-lacZ* reporter construct further define this sequence. Interestingly the sequence partially overlaps a binding site for the *creA* gene product which is involved in catabolic repression.

Poster 22

The *Polcomblike* gene of Drosophila melanogaster is required for the correct expression of the homeotic genes of the Antennapedia and Bithorax complexes.

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Clonal analysis indicates that the expression of parasegmental specific sets of homeotic genes from the Antennapedia (ANT-C) and Bithorax (BX-C) complexes is required throughout *Drosophila* development. However, the expression of the early patterning genes, which direct specific homeotic gene expression , is transient, ending just after germ band extension. A mechanism must exist by which the determined state of cells within a parasegment can be maintained throughout development.

Mutations in any one of a group of unlinked genes known as the *Polycomb* group (Pc-G), lead to the derepression of homeotic gene expression after the early patterning gene products have degraded. Mis-expression of the homeotic genes as a result of these mutations results in transformations of body segments to resemble those normally found in a more posterior position. Various lines of evidence suggest that the Pc-G gene products act as a multimeric protein complex, specifically recognising genes that are repressed by the early patterning genes, forming a chromatin structure that is transcriptionally inactive and clonally heritable.

We have recently cloned a member of the Pc-G known as *Polycomblike*. Sequence analysis of the *Polycomlike* cDNA has revealed one large ORF, which when conceptually translated encodes a protein of ~ 100kD with no significant homology to other known proteins. *In situ* hybridisation indicates that the Pcl transcript is expressed throughout the embryo. We have raised a polyclonal antiserum to the *Pcl* protein and used it to localise the *Pcl* product in situ. These experiments have revealed that Pcl is a nuclear localised protein. Further, we have found that Pcl binds specifically to approximately 120 sites on polytene chromosomes. These sites are identical to those bound by two other members of the Pc-G : Polycomb and polyhomeotic[1]. We have demonstrated that Pcl is co-immunoprecipitated with Polycomb from embryonic nuclear extracts, indicating that they interact with each other in vivo. These data supports a model in which members of the Pc-G form a protein complex which specifically recognises, binds and represses target genes in response to the local chromatin environment.

1. Franke, A., De Camillis, M., Zink, D., Cheng, N., Brock, H., Paro, R.(1992) *EMBO* J. **11**, 2941-2950

Poster 23

Genetic and Molecular Study on A Putative Regulatory Gene in *Aspergillus nidulans*

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The filamentous fungus *Aspergillus nidulans* provides a versatile model for a better understanding of gene regulation systems in eukaryotes. In our laboratory we are studying the regulation and secretion of extracellular proteases in *A. nidulans* where the production of proteases is regulated in response to carbon, nitrogen and sulphur limitations. A number of mutations in genes which affect the secretion of proteases in *A. nidulans* have been already studied - the gene mediating nitrogen metabolite repression (*areA*), two genes affecting carbon catabolite repression (*creB* and *creC*) and also a new regulatory gene, *xprE* (M.E. Katz, unpublished data).

We have identified a new putative regulatory locus which is, in addition, involved in secretion of extracellular proteases. A pleiotropic recessive mutation in this locus, designated *xprF1*, appears to elevate the level of enzyme secretion in response to starvation for nitrogen or carbon (in particular) nutrients. Furthermore, the *xprF1* mutation results in a negative effect on the utilisation of nitrogen sources in the presence of glucose. The *xprF1* suppresses loss-of-function mutations in *xprE* and *areA* genes. Genetic data suggests that *xprF* is located on the linkage group VII and presumably has a wide-domain, negatively acting regulatory nature.

We are currently using chromosome-specific recombinant DNA libraries (Brody et al., 1991, *Nucleic Acids Research*, 19(11): 3105-3109) to isolate the *xprF* gene.

MAPPING POLYMORPHIC MICROSATELLITES IN THE PIG (*Sus domestica*).

Muladno, Paul Le Tissier and Chris Moran
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Microsatellites from known genes identified by database searching were typed in a panel of 3 individuals from each of nine European breeds sampled in Australia to estimate allelic variation and suitability for within-breed genetic mapping studies. The CTG trinucleotide repeat in the tumour necrosis factor beta gene had 13 different alleles from the 27 animals typed; osteopontin (AC repeat), 8 alleles; diacyl glycerol kinase (AC repeat) and the chorionic gonadotrophin alpha polypeptide (CT-CTTT compound repeat), 7 alleles each; and insulin like growth factor 1 (AC repeat), 3 alleles.

The same microsatellites were also typed on reference families from the European collaborative PiGMaP programme, comprising grandparents, parents and F2 offspring of a Chinese Meishan by European Large White cross, enabling assignments to chromosomal locations.

Some potential microsatellites failed to amplify or multiple bands were produced which could not be eliminated by increasing the stringency of annealing. As some microsatellites were found within cDNA sequences during database searching, the presence of introns makes prediction of PCR product size difficult and may prevent amplification due to interruption of the primers or due to excessive size of the genomic region between the primers. PCR products which do not reveal microsatellite polymorphism are also being analysed by temperature gradient gel electrophoresis or heteroduplex analysis to determine whether any sequence polymorphism can be recognised.

Generation and identification of putative mycorrhizal mutants of *Hordeum vulgare* cv. 'Galleon'.

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Vesicular-arbuscular (VA) mycorrhizal associations can occur between approximately 120 species of fungus and an estimated 85% of all terrestrial angiosperms. Functional VA mycorrhiza development can increase growth, abiotic stress tolerance and pathogen resistance in the plant. These beneficial effects have been primarily ascribed to the increased uptake of phosphorous into the host roots.

To complement our current investigation of plant gene expression in barley roots during VA mycorrhiza formation, we have generated two populations of mutagenised 'Galleon' barley by ^{60}Co irradiation. Subsequent screening of 1500 M2 plants has identified several lines which appear to be VA mycorrhizal (*myc*) mutants. These putative *myc* mutants have been partially characterised. The plants are now being backcrossed to the parent line to stabilise and further characterise the apparent mutations.

Poster 26

An Important *fac*-tor in *amdS* Gene Regulation

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The *facB* gene of *Aspergillus nidulans* encodes an activator which mediates acetate induction of the acetamidase-encoding *amdS* gene and genes required for acetate metabolism via the glyoxylate bypass. Cloning and sequence analysis of *facB* has revealed a $Zn(II)_2Cys_6$ DNA binding motif.

The *facB88* mutation was selected due to its resultant high level, constitutive expression of *amdS*. The molecular nature of the mutation is a reciprocal translocation with breakpoints in the *facB* gene on chromosome VIII and a previously unidentified gene on chromosome IV designated gene*X*.

The two novel recombinant genes resulting from the *facB88* translocation have been cloned. Sequence analysis across the breakpoints has revealed that the *facB* DNA binding domain has fused in frame to a portion of gene*X* encoding two C_2H_2-type zinc fingers. To elucidate the function of gene*X* a null mutant will be constructed using insertional inactivation.

Allelic silencing is responsible for the loss of ABO blood group antigens in malignancy

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Loss of ABO blood group antigens is found on the surface of carcinomas and on the red blood cells of some patients with haematological malignancies. As the ABO locus *per se* is unlikely to be important in cancer progression, the loss of antigenic expression is a signpost to other genetic changes in the malignant stem cell population. Selection against a syntenic tumour suppressor gene may be the underlying cause of these changes.

We have used PCR to determine the genotype and to assess loss of heterozygosity at the ABO locus in four patients with haematological malignancy showing loss of red cell ABO antigens. All patients were heterozygous (AO) and PCR genotyping showed no difference in allele dosage. Thus, loss of ABO is not due to loss of heterozygosity. Reverse transcription PCR showed that these four patients had markedly decreased transcription of the A allele relative to the O allele. Therefore, in these cases, loss of ABO antigens results from the silencing of a single allele of the ABO gene. Similar loss of phenotypic but not genotypic heterozygosity was obtained with colon carcinoma biopsies and with colon carcinoma cell lines. Allelic silencing represents a type of (epi)genetic event which may be under-recognised in human malignancy and genetic disease.

Computer Simulations for Population Genetics

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Monte Carlo simulations are being used to explore the dynamic properties of single populations. Both demographic and genetic "data" can be recorded, allowing a comparison between observed levels of change in the genetic constitution of the population with those predicted from analytic theory. Conventional calculations use the demographic data alone and are algebraic models. The intention is to assess the efficacy of such calculations, and to examine the relative merits of a simulation based methodology. With this approach it is possible to test the sensitivity of both types of model to violations of the various assumptions on which they are based.

Investigation of the Dynamics of Coefficients of Linkage Disequilibrium

OBO-LINK is a special purpose, individual-based model using a representation of individuals with *two chromosomes and two allelic loci*. One is regarded as a disease locus, and can exert selective pressures, the other is regarded as a neutral marker. The recombination rate, between these two loci, as well as a number of other parameters, can be specified by the user. In each "year", it's possible to record haplotype frequencies and calculate coefficients of linkage disequilibrium. Thus the dynamics of these statistics, under a variety of conditions, can be visualised graphically.

Comparisons can be made, between two configurations of the model that are identical in all but one respect. Quantitative results are thus obtained, to measure the relative effect of specific factors such as initial haplotype distribution, sex-linkage, differing selective regimes and overlapping generations, as well as general life strategies.

Poster 29

CHROMOSOMAL EVOLUTION IN ONYCHOPHORANS FROM AUSTRALIA (ONYCHOPHORA : PERIPATOPSIDAE)

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The Australian Onychophora are widespread and often locally abundant. They are generalist predators on arthropod species living in soil, logs or leaf-litter, a lifestyle which makes them potentially valuable indicator species. Despite considerable morphological variation, and the existence of viviparous and oviparous forms, only 10 species comprising eight genera are currently described. A detailed karyotypic analysis of 48 populations, embracing 6 described species of Australian onychophorans has revealed the presence of 27 distinct karyomorphs, distinguishable on the basis of the number of chromosomes and their size distribution.

Poster 30

PHYLOGENETIC ANALYSIS OF EXTINCT AND EXTANT DIPROTODONTIAN MARSUPIALS USING DNA SEQUENCING.

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The true phylogeny of Diprotodontian Marsupials (Wombats, Kangaroos, and Possums). is still unknown, although many hypotheses have been proposed. The skeletal morphology is ambiguous, that is the phylogeny is dependent on the set of characters used. As well as the traditional dental and skeletal morphological data, many other character sets such as brain and chromosome morphology, serrology, DNA-DNA hybridisation, and micro compliment fixation have been used to create phylogenetic hypotheses.

This project involves obtaining sequences of the mitochondrial cytochrome b (cyt b) gene and using this to form a phylogenetic tree. This tree will then be compared to the other existing trees, in an effort to determine which morphological character sets are most important for phylogenetic analysis, in the light of the molecular data.

The extension of this is to extract ancient DNA from the extinct Marsupial Lion, *Thylacoleo*, to find where it fits in the marsupial phylogeny. Ancient DNA is generally sheared to lengths of only a few hundred bases, for this reason only very short (50-100 bp) sections of ancient DNA may be successfully amplified at once. To this end the project also involves using the sequences obtained from extant material to design primers specific to Diprotodontian Marsupials. These primers will only have a short amplification distance, and thus will be ultimately used on the fossil material.

Sequences obtained will be analysed using programs available on the PHYLIP package. To date 310 bp sequences have been obtained for five Diprotodontian marsupials, namely *Trichosurus*, *Petaurus*, *Pseudocheirus*, *Lasiorhinus*, *Phascolarctos*, as well as several kangaroo species obtained from the South Australian Museum. Analysis of these has indicated that the sequences, when compared to a generally accepted Diprotodontian phylogeny, support the placing of Koala with Wombat. However, *Trichosurus* (the common brushtail possum) is consistently placed with the Wombat and Koala as opposed to being placed with the other possums. *Pseudocheirus* (the ringtail possum) is never grouped with *Petaurus* (the sugar glider), but is placed with either *Trichosurus* or as a separate group.

Molecular Phylogenetic Relationships of the Pachycephalinae (Passeriformes: Aves).

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Dept. Ornithology, Museum of Victoria

The Pachycephalinae are a morphologically diverse group of songbirds centred in the Australasian region. Its typical members include the whistlers and shrike-thrushes. There are, however, several aberrant monotypic genera whose affinities are poorly understood. They occur in the montane regions of New Guinea and the dry woodland scrub areas of Australia. To establish relationships between the various disparate genera, a 860 bp fragment of the mitochondrial cytochrome b gene was sequenced from 10 of the 14 currently recognised genera. The phylogenetic and biogeographical implications of the results are detailed.

Poster 32

MAPPING A GENE INVOLVED IN ARABINOSE METABOLISM IN *ARABIDOPSIS THALIANA*.

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An arabinose-sensitive mutant of *Arabidopsis thaliana* was fortuitously isolated following EMS mutagenisis of wild-type seed. Growth comparisons of wild-type, mutant and progeny of crosses between the two, on a selective concentration of L-arabinose, indicate that the arabinose-sensitive phenotype is semi-dominant and caused by a mutation at a single nuclear locus, designated *ARA1*. Plants carrying a mutation at this locus exhibit a reduced ability to incorporate exogenous labelled L-arabinose into ethanol insoluble polysaccharides and show a decrease in arabinose kinase activity to approximately 10% of the wild-type level.

Genetic mapping studies indicate that the *ARA1* locus is located on chromosome 4 of the *Arabidopsis* linkage map and is tightly linked to the visible marker *FCA* (0.15cM), mutants of which exhibit a "late-flowering" phenotype. Thus, by positioning recombination events between these two loci, it is expected that a precise location for *ARA1* will be obtained.

RFLP mapping has shown close linkage between the *ARA1* locus and RFLP marker λAt226. A number of YAC clones have been identified in this region, one or more of which is likely to carry the *ARA1* gene. End-probes from these clones have been used to position *ARA1*-*FCA* recombination events and a chromosome walk undertaken to clone the *ARA1* gene. Analysis of putative deletion mutants of the *ARA1* locus, along with functional complementation of the arabinose-sensitive mutant, are being performed for positive identification of this gene.

Poster 33

Analysis of mutations affecting carbon catabolite repression in *Aspergillus nidulans*.

Robert Shroff and Joan Kelly

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Carbon catabolite repression functions to repress a wide range of genes involved in the use of alternative carbon sources when readily metabolised carbon sources are present. Genetical analysis of *Aspergillus nidulans* suggests that the protein CREA is a wide domain, negatively acting regulator protein in the process of carbon catabolite repression (Arst and Bailey, 1977). The *creA* gene has been cloned in both *Aspergillus nidulans* and *Aspergillus niger* (Dowzer and Kelly, 1989; Drysdale *et al.* 1993). Theoretical translation of the DNA sequence shows the presence of a Cys_2-His_2 zinc finger region, an alanine rich region and S(T)PXX motifs, consistent with the proposed function of CREA (Dowzer and Kelly, 1991). In addition, a comparison between *A. nidulans* and *A. niger* shows a domain of 42 amino acids which are identical. Significant homology exists between this region and one found in RGR1 - involved in carbon catabolite repression in *Saccharomyces cerevisiae* (Drysdale *et al.* 1993).

A PCR-SSCP approach has been used to characterise a number or *creA* mutations. The mutations fall into two distinct classes: (1) missense mutations in the zinc finger region. Comparisons with mutations studied in other proteins in the Cys_2-His_2 zinc finger family show that these mutations are consistent with a disruption of the zinc finger region and reduced DNA binding affinity. (2) nonsense or frameshift mutations in the vicinity of the RGR1 homologous sequence suggesting a functional role for this domain, the nature of which requires further analysis.

References

Arst, H.N. and Bailey, C.R. (1977) in *Genetics and Physiology of Aspergillus nidulans*. Academic Press, London, p131-146.
Dowzer, C.E.A. and Kelly, J.M. (1989) *Current Genetics*, **15**, 457-459.
Dowzer, C.E.A. and Kelly, J.M. (1991) *Molecular and Cellular Biology*, **11**, 5701-5709.
Drysdale, M.R., Kolze, S.E. and Kelly, J.M. (1993) *Gene*, In Press.

PROLIFIC INBRED LINES OF MICE

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NSW 2006

Seven sublines have been established from a highly prolific non-inbred Swiss strain of mice having 17.2 +/- 0.3 pups per litter born alive (averaged over the first two litters). In these sublines, full-sib inbreeding with selection for high litter size and low inter-litter interval has been applied for a minimum of 18 generations. Six of the seven sublines have survived. At generation 18, average born-alive litter sizes (of the first two litters) ranged from 9.5 +/- 0.9 to 15.1 +/- 0.6, and average inter-litter interval ranged from 26.0 +/- 2.9 to 30.1 +/- 2.2 days. In contrast, the comparative average litter size for the standard inbred strain C57BL/6 is 7.0 +/- 0.5. By the end of 1993, all six lines will have reached generation 20, at which stage they can be designated as inbred lines.

During the inbreeding programme, ovulation and implantation data have been collected at generation 5 and at every generation from 10 onwards, from full-sib sisters of breeding pairs. At generation 15, the number of corpora lutea ranged from 18.3 +/- 0.3 to 23.2 +/- 0.3, and the number of implanted embryos ranged from 11.3 +/- 0.3 to 16.5 +/- 0.3.

The sublines are a valuable resource for investigating the genetic and endocrinological basis of prolificacy.

Assessing population structure in the cotton bollworm, *Helicoverpa armigera (Lepidoptera:Noctuidae)*, using variation in the AT-rich region of mitochondrial DNA.

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Helicoverpa armigera is an introduced cosmopolitan pest that attacks a wide variety of agricultural crops in Australia, especially cotton. Genetic variation in the AT-rich region of mitochondrial DNA is being examined amongst individuals from different geographical locations to assess population structure. A 700bp DNA fragment is amplified using primers in conserved genes that span the AT-rich region. Using one of the same primers, direct double-stranded sequencing yields 250bp of AT-rich sequence from individual moths. To date eighteen polymorphic nucleotide positions within this sequence has allowed the identification of 26 different genotypes among 70 moths. Some genotypes appear to be relatively common and occur in most regions while other genotypes are unique to a particular region. The relatively high degree of variation suggests a large effective population size and/or a high mutation rate. To quantify the degree of population substructure, and to assess the value of this approach for the ecologists attempting to understand the extent and patterns of moth movement, further sampling is underway. In addition, this AT-rich region promises to be of value for the clarification of current taxonomic ambiguities in the genus *Helicoverpa*.

The mechanism of P element-induced recombination in *Drosophila melanogaster*. J Sved, L Blackman, R Colless and Y Svoboda, School of Biological Sciences, University of Sydney, NSW 2006.

It has been known for many years that P elements generate male recombination in *Drosophila melanogaster*. We (*Genetics* **124**: 331; 1990) have used a 2-element system, the genetically marked incomplete P element P[*CaSpeR*], and the transposase-producing element P[Δ2-3](99B), to generate recombination levels up to 1%, comparable to that found in crosses using naturally occurring P strains. Furthermore with the P[*CaSpeR*] element present in homozygous condition, the frequency of recombination rises to 15-20% (*Mol. Gen. Genet.* **225**: 443; 1991).

We have cloned the region containing one particular P[*CaSpeR*] insertion at 50C, and designed primers around the element. Using these, we have analysed recombinant chromosomes and shown that many appear to contain complete copies of the element at the original site. The combination of reciprocal recombinant chromosomes containing the complete element re-generates the high recombination effect. Neither of these results is in agreement with a simple prediction that recombination arises as a consequence of repair following P element excision (*Cell* **62**: 515; 1990)

In a separate experiment, we have produced chromosomes which contain internal deletions of the P[*CaSpeR*] element at 50C. This has yielded a range of elements, some of which have deleted ends and some internal deletions. The combination of elements with non-overlapping deletions should yield useful information about models of recombination.

(Supported by Australian Research Council)

Poster 37

Identification of varieties of *Gaeumannomyces graminis* and their isolates by analysis of nuclear ribosomal DNA.

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SUMMARY

Analysis of nuclear ribosomal RNA genes in *Gaeumannomyces graminis* have revealed two levels of variation, one allowed identification of *G. graminis* varieties and the other enabled discrimination between isolates within varieties. The three varieties of *G. graminis graminis* (*Ggg*), *G.g. avenae* (*Gga*, cause of oat take-all) and *G.g. tritici* (*Ggt*, cause of wheat take-all) can be differentiated at the rDNA region. Take-all is the most damaging root disease of wheat world-wide and causes Australian farmers about $200 million loss in revenue annually. Isolates within each variety can be differentiated by restriction site differences in the intergenic spacer region and by variation in repeat length. Within the collection of isolates identified as *Ggt* on morphological criteria, 3 subgroups (T1, T2 and T3) could be distinguished based on the size of *Eco*RI, *Hin*dIII/*Eco*RI and *Hin*dIII/*Pst*I fragments generated from the 26S rRNA coding region. Subgroup T2 possessed minor amounts of an additional fragment that is typical of var. *avenae*. Subgroup T3 possessed all the fragments characteristic of var. *avenae* and none of T1. All isolates in T2 and T3 were pathogenic to wheat, oats and bentgrass. The ability to detect oat-attacking *Ggt* will help farmers in their crop rotation programme where oats is frequently used as a break crop. The presence of oat-attacking *Ggt* will suggest that oats will not be a suitable 'cleaning' break crop.

Inheritance of resistance to herbicides that inhibit ACCase in annual ryegrass.

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Annual ryegrass (*Lolium rigidum*) biotype WLR96 is resistant to a range of herbicides that inhibit the plastid enzyme acetyl-coenzyme A carboxylase (ACCase). Crosses were made between resistant WLR96 and individuals from a susceptible biotype. The response of the reciprocal F_1 families was identical showing that the resistance trait was nuclearly inherited. The F_1 plants had a response to the herbicide haloxyfop that was intermediate between the resistant and susceptible parents. LD_{50} values of the F_1 were 6 times lower than that of resistant plants but more than 100 times higher than that of susceptible plants, indicating partial dominance. Treatment of the F_2 families at two different rates of haloxyfop resulted in a 3:1 (resistance:susceptibility) segregation ratio indicating single gene action. It is therefore concluded that herbicide resistance in biotype WLR96 is inherited as a single, semi dominant, nuclear gene. This result is consistent with the fact that the biochemical basis for resistance in this biotype is an altered form of the target site enzyme ACCase.

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SIMPLE SEQUENCE POLYMORPHISMS AS A MEASURE OF GENETIC VARIABILITY IN THE ENDANGERED NORTHERN HAIRY-NOSED WOMBAT

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This study provides the first demonstration of a new molecular genetic approach to measure variation in bottlenecked populations. We investigated the utility of hypervariable simple sequence loci to measure genetic variability and relatedness of the northern hairy-nosed wombat, one of Australia's rarest mammals. This species suffered a dramatic range and population reduction over the past 120 years and now exists as a single colony of about 70 individuals in Epping Forest, central Queensland. Our past (unpublished) research on mitochondrial DNA and multilocus DNA fingerprinting has not revealed informative variation in this population. Consequently, we chose to examine variation in simple sequence repeats, a class of loci known to be highly polymorphic in mammals. Our results show that appreciable levels of variation still exist in the Epping Forest colony, although they have only 41% of the heterozygosity shown in a population of a closely-related species. From museum specimens collected in 1884, we also assessed simple sequence variation in an extinct population of the northern hairy-nosed wombat near Deniliquin, New South Wales, over 1000 kilometres to the south of the extant population. Heterozygosity levels in the historic population appear higher than in their conspecifics at Epping Forest. The apparent loss of variation in the Epping Forest colony is consistent with an extremely small effective population size of 10-20 individuals throughout their 120 year decline.

Analysis of an *Aspergillus* Transcription Fac-tor.

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The *facB* gene of *Aspergillus nidulans* encodes an activator which mediates acetate induction of *amdS* (encoding acetamidase) and genes required for acetate metabolism *via* the glyoxylate bypass. Cloning and sequence analysis of *facB* has revealed a $Zn(II)_2Cys_6$ DNA binding motif and potential acidic activation domains.

Temperature sensitive *facB* mutants fall into two classes. One class shows temperature sensitive synthesis of glyoxylate bypass enzymes and acetamidase. This is consistent with a regulatory role for *facB*. The second class of *facB* mutants displays thermolability of enzymes of the glyoxylate bypass but not of acetamidase. This indicates that *facB* also plays a structural role in acetate metabolism.

To localise these diverse functions to specific regions of FacB, seven mutant *facB* alleles have been cloned and sequenced. In addition, cloning and sequence analyses of the *facB* homologues from *A. oryzae* and *A. niger* have been undertaken to reveal conserved regions in *facB*. In combination, these approaches have identified regions in the predicted protein product which are involved in regulatory and structural functions. The cross-species studies have also revealed conserved regions that are potential regulatory sequences 5' of the gene.

Poster 41

THE USE OF HIGHLY POLYMORPHIC DNA MARKERS USING PCR IN FORENSIC SCIENCE

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Historically, forensic scientists have approached the individualization of body tissue samples using polymorphic protein systems. DNA technology, and in particular the PCR methodology, offers considerable improvement over some of the limitations inherent in the protein systems. In particular, PCR allows the use of extremely small as well as degraded samples because of the ability to amplify the amount present. A wider range of tissues (eg bones, hairs) becomes amenable to testing. In addition, the DNA systems are even more polymorphic than the protein systems and thus have a higher discrimination power (P_D). As a consequence, the population genetics of the systems have become more of an issue.

We have been using PCR in criminal casework for about two years using the HLA.DQA1 locus. More recently we have used 3 length polymorphic loci (D1S80, Apo B, YNZ22). We are looking at further loci, in particular using STR systems. The abundance of highly polymorphic markers in the human genome now presents forensic scientists with the additional dilemma of a plethora of choices.

Poster 42

Investigation of the *trp* and *trpl* phototransduction genes of *Drosophila melanogaster*

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The *trp* and *trpl* genes of *Drosophila* are both believed to encode light-sensitive ion channels present in the photoreceptor cells of the *Drosophila* compound eye. The *trpl* gene was isolated as a calmodulin-binding protein, and it has now been demonstrated that the *trp* protein also binds calmodulin. It is therefore possible that both genes encode ion channels, or channel subunits, which are modulated by calmodulin.

In order to investigate the mechanism by which calmodulin regulates the activity of the two proteins, calmodulin-binding studies are being carried out. The aim of these studies is to identify the calmodulin-binding sites in the two proteins and the relative affinities of the sites for calcium/calmodulin. This is being undertaken by making constructs containing putative calmodulin-binding sites in the expression vector *pMal* and producing fusion proteins. Calmodulin-binding is then detected by probing Western blots with biotinylated calmodulin.

Both the *trp* and *trpl* proteins also contain an ankyrin-like repeat motif. This motif has been identified in a number of very diverse proteins, and while its function remains unclear, it may be involved in the specific recognition of other proteins or in binding proteins to the microtubular cytoskeleton. The role of this motif in the *trp* and *trpl* proteins may be clarified by identifying proteins which bind to the motif. In order to isolate such proteins, fusion proteins containing the motif are being made in *pMal* and used to produce an affinity matrix for the purification of proteins which bind to the *trp*/*trpl* ankyrin repeat motifs.

Poster 43

The Distribution of Rhizopine genes in *Rhizobium* populations

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Rhizobia are soil bacteria capable of nodulating leguminous plants. Within nodules atmospheric nitrogen is converted to ammonium by differentiated rhizobia (bacteroids) and made available for plant growth. Rhizopines are selective growth substrates synthesised by bacteroids and catabolised by free-living rhizobia. Only strains capable of directing rhizopine synthesis can catabolise these compounds. Rhizopines play an important role in enhancing the competitiveness of their rhizobial host by providing a selective nutrient source both inside the nodule and in the rhizosphere following nodule senescence. Rhizopine genes were first investigated in the lucerne-nodulating strain *Rhizobium meliloti* L5-30 which synthesises the inositol-based rhizopine 3-*O*-methyl-*scyllo*-inosamine (3-*O*-MSI). In *R. meliloti* L5-30, rhizopine genes are located on the Symbiotic plasmid (which also encodes genes for nodulation and nitrogen-fixation).

To determine the frequency of rhizopine genes in the rhizobial population we surveyed over 300 strains of *R. meliloti* and *R. leguminosarum* (the microsymbiont for peas, clover and beans) for their ability to catabolise 3-*O*-MS I as a sole carbon source. In total 24 rhizopine catabolising strains were isolated. All strains examined so far are able to synthesise 3-*O*-MSI. The rhizopine synthesis (*mos*) and catabolism (*moc*) genes of a pea-nodulating strain, *R. leguminosarum* bv. viciae 1a, have been cloned and were found to be located on the Symbiotic plasmid. DNA from rhizopine-catabolising *R. leguminosarum* bv. viciae was isolated and probed with *moc* and *mos* genes from strains 1a and L5-30. Restriction Fragment Length Polymorphisms have been determined and these results will be presented. A sub-group of *R. leguminosarum* bv. viciae (previously tested for 3-*O*-MSI catabolism) have been analysed by Multi Locus Enzyme Electrophoresis and their Electrophoretic Types (ETs) determined. We discuss the distribution and significance of rhizopine genes within the rhizobial population.

MOLECULAR EVIDENCE FOR THE GENERATION OF VARIABILITY IN *RHYNCHOSPORIUM SECALIS*. **D. Whisson and Herdina, SA Department of Primary Industries, Waite Campus, University of Adelaide, Adelaide, Australia.**

Rhynchosporium secalis (Oud.) Davis is the fungal pathogen of barley that causes the foliar disease, scald or leaf blotch. Breeding for scald resistance has been largely unsuccessful due to the extreme pathogenic variability of the fungus and to the limited knowledge of resistance genes. Studies on the inheritance of scald resistance have indicated that the interaction follows Flor's "gene-for-gene' hypothesis but the lack of a known sexual stage for *R. secalis* precludes a formal genetic analysis. From studies on this and other *Deuteromycete* fungi, it has been postulated that the diverse pathogenic variability may be due to parasexual recombination and heterokaryosis. Because such fungi lack a sexual stage these processes may be fundamental to the generation of variability. Previous evidence that parasexual recombination occurs in *R. secalis* comes from isozyme studies and experiments on the generation of new races following a mixed infection of several known races. The present molecular investigation provides further evidence for parasexual recombination; but also presents the possibility that other events such as chromosome rearrangements, ploidy, transposable elements or other genetic mechanisms may be contributing to the generation of pathogenic diversity. For instance, mixtures of 2 pathotypes identified by distinct RFLP patterns using a dispersed repeat sequence, mixed together on a leaf or in culture produced new pathotypes with novel RFLP patterns. This suggests that asexual recombination has taken place and agrees with the isozyme work for mixtures of isolates. However, single spore isolates, when maintained in culture for a long period of time, also demonstrated changes but these were usually for a single band. The pathogenicity of the isolates remained unchanged. The results suggest that mobile elements may play a role in the generation of variation and research is in progress to resolve this and the possible involvement of ploidy changes and chromosome rearrangements.

Sequence differences between *cog* and *cog*$^+$, two alleles of a recombinator in the *histidine-3* region of *Neurospora crassa*.

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cog and *cog*$^+$ are located between *histidine-3* and *adenine-3* on Linkage group I[1] of the fungus *Neurospora crassa*. The frequency of meiotic recombination in this region is under genetic control, and is higher when *cog*$^+$ is present, providing the dominant allele of *rec-2* (*rec-2*$^+$; LG V) is absent from the cross[1,2].

We have identified the region responsible for the difference between *cog* and *cog*$^+$ by locating crossovers in *cog* and *cog*$^+$ recombinant progeny of a cross between Yale (*cog*$^+$) and St Lawrence (*cog*) wild-type strains. This was done by using the considerable number of restriction fragment length polymorphisms in this region between the two strains. Thus we now know that the sequences determining the difference between *cog* and *cog*$^+$ are more than 300 bp and less than 3.5 kb 3′ of the 3′ end of the *his-3* coding sequence.

The regions have been cloned both from Yale and St Lawrence. Both *cog* and *cog*$^+$ DNA has been sequenced and differences between the two identified. The major difference is a stem-loop structure about 100 bp in length present only in St Lawrence, but other differences exist. These include another small insertion in the St Lawrence sequence (about 50 bp with no obvious structure) and regions containing multiple single base-pair insertions in St Lawrence when compared to Yale. The significance of these differences will be discussed.

1. Angel, T., B. Austin and D.G. Catcheside, (1970). Regulation of recombination at the *his-3* locus in *Neurospora crassa*. Aust. J Biol. Sci., **23**: 1229-1240.

2. Smith, B.R., (1968). A genetic control of recombination in *Neurospora crassa*. Heredity, **23**: 162-163.

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Second Edition

Daniel L. Hartl, Washington University School of Medicine, and Andrew G. Clark, Pennsylvania State University

"What Hartl and Clark have provided is clearly written, quite current and impressively thorough. In addition to traditional mendelian population genetics, the text includes quantitative genetics, some ecological genetics and a brief touch on the genetics of speciation....For students with some prior knowledge of the issues and the difficulty of population genetics, or for instructors willing to create this context, this is clearly the book choice. For clarity and completeness it is unsurpassed."
Jody Hey, Trends in Genetics

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1. Darwinian Evolution in Mendelian Populations - 2. Random Genetic Drift - 3. Mutation and the Neutral Theory - 4. Natural Selection - 5. Inbreeding and Other Forms of Nonrandom Mating - 6. Population Subdivision and Migration - 7. Molecular Population Genetics - 8. Evolutionary Genetics of Quantitative Characters - 9. Ecological Genetics and Speciation Suggestions for further Reading - Bibliography - Author Index - Subject Index

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Product Profile

QIAamp Blood Kit

DNA purification for PCR from blood and cell culture

The QIAamp Blood Kit provides a fast and easy way to efficiently purify DNA directly from whole blood, plasma, blood cells, or cell culture for reliable PCR* amplification.

QIAamp Blood Kit means:

- DNA purification from whole blood, plasma, or cells in less than 20 minutes.
- Complete removal of hemoglobin and other contaminants.
- DNA sized from 20-40 kb for reliable PCR amplification.
- No phenol or chloroform extraction.
- Concentrated DNA eluted in water or buffer for direct amplification.

The procedure requires no cell separation or phenol/chloroform extraction, and is ideal for rapid processing of multiple samples. The purified DNA can be added directly to the PCR reaction or stored for later reference.

High Yield and Purity

With QIAamp, 8-15 µg of genomic DNA can be purified directly from 300 µl of whole blood. The DNA is free of protein and other contaminants such as heme which may inhibit PCR amplification even when present in only trace amounts. The A_{260}/A_{280} ratio of DNA purified on QIAamp columns typically ranges from 1.7-1.9.

Safe, Easy Handling

The QIAamp Blood Kit allows rapid isolation of pure genomic DNA from multiple samples in less than 20 minutes. Potentially infectious agents can be inactivated, without any effect on DNA yield or quality, by incubating the sample at 98°C for an additional 5 minutes after cell lysis.

Whole blood, plasma, blood cells or cultured cells

↓

Lyse cells and load into QIAamp-spin column

↓

Wash out proteins, hemoglobin and other contaminants

↓

Elute pure total DNA ready for direct amplification

Figure 1. Flow diagram of the QIAamp Blood Kit procedure.

Efficient Amplification

The purified DNA ranges in length from 1-50 kb (majority 20-40 kb) and is ideally sized for efficient PCR amplification, requiring no further shearing or digestion before use. DNA of this length denatures completely and shows the highest efficiency in PCR amplification. This is demonstrated in Figure 2, which compares the amplification efficiency of various amounts of target DNA prepared with the QIAamp Blood Kit, to the amplification efficiency of sheared or high molecular weight genomic DNA.

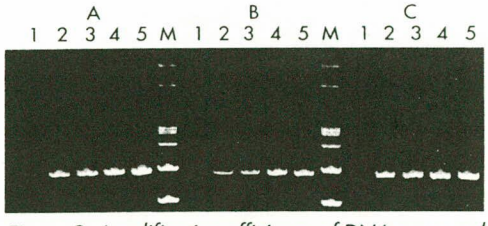

Figure 2. Amplification efficiency of DNA prepared by various methods. A: QIAamp Blood Kit. B: High MWt genomic DNA. C: Sheared genomic DNA. Lanes 1: 1 ng of template. 2: 50 ng. 3: 100 ng. 4: 200 ng. 5: 500 ng. M: DNA size markers 78 bp - 881 bp.

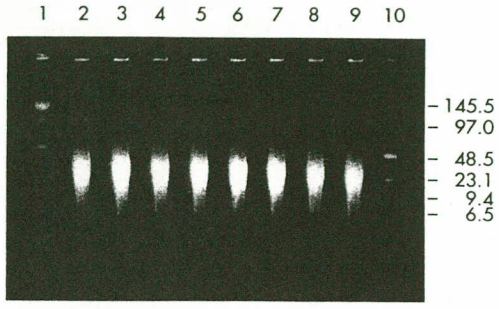

Figure 3. DNA purified using the QIAamp Blood Kit from 300 µl samples of human whole blood, analyzed on a 1.5% agarose gel by PFGE. Lanes 1,10: DNA MWt markers. Lanes 2-9: 3.5 µg purified human whole blood DNA.

Simple Procedure

The QIAamp Blood Kit is easy to use. Simply lyse the blood sample or cell suspension for just 10 minutes, then load it directly into the QIAamp-spin column by microcentrifugation. Each QIAamp-spin column is sealed to prevent aerosol formation and avoid cross-contamination.

Two brief, high speed washes strip the DNA of proteins and efficiently remove all contaminants without affecting DNA binding to the QIAamp-spin column.

The pure, partially fragmented DNA is eluted from the QIAamp-spin column by a final centrifugation step. DNA is eluted in either water or buffer, ready for direct addition to the PCR reaction. The DNA is free of nuclease and can be safely stored for later use.

Biological Sample Sources

QIAamp Blood Kit is suitable for use with fresh or frozen whole blood treated with either citrate, heparin or EDTA, leukocytes, buffy coat, plasma, or cultured cells.

Each QIAamp Blood Kit contains 50 or 250 QIAamp-spin columns, Collecting Microtubes, buffers and reagents, and a comprehensive handbook.

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Elder Hall	K9	Elder Hall

LECTURE THEATRES / PERFORMANCE SPACES / MEETING ROOMS

Benham Lecture Theatre	C9	Benham Laboratories, Gnd. fl.
Bragg Lecture Theatre	F5	Bragg Laboratories, Gnd. fl.
Chapman Lecture Theatre	F11	Engineering North, level 1
Fisher Lecture Theatre	C12	Fisher Bld. Gnd. fl.
Flentje Lecture Theatre	H9	Plaza Bld., Level 2
Florey Lecture Theatre	C15	Medical Sch. Nth. level 1
Hartley Concert Room	F1	Hartley Bld., 1st fl.
Hone Lecture Theatre	D15	Medical School South, Gnd. fl.
Horace Lamb Lecture Theatre	G9	Horace Lamb Bld., Gnd. fl.
Hughes Lecture Theatre	J8	Hughes Bld., Level 2
Kerr Grant Lecture Theatre	H5	Physics Bld., 1st fl.
Law Theatre 1	L13	Ligertwood Bld., 2nd fl.
Law Theatre 2	L13	Ligertwood Bld., 3rd fl.
Little Theatre	E5	Union House, Level 5
Macbeth Lecture Theatre	D3	Badger Laboratories, Gnd. fl.
Madley Dance Space	E3	Madley Bld., Gnd. fl.
Maths G02	K12	Napier Bld., Gnd. fl.
Mawson Lecture Theatre	B11	Mawson Bld. Gnd. fl.
Medico1 Sch. Sth. Rm. 106	D15	Medical Sch. Sth. level 1
Medical Sch. Sth. Rm. 210	D15	Medical Sch. Sth. level 2
Napier 102 Lecture Theatre	K12	Napier Bld., 1st fl.
Napier LG 28	K12	Napier Bld., lower Gnd.
Napier LG 29	K12	Napier Bld., lower Gnd.
Napier Theatre 102	K12	Napier Bld., 1st fl.
Napier Theatre G03	K12	Napier Bld., Gnd. fl.
Napier Theatre G04	K12	Napier Bld., Gnd. fl.
Polygon Lecture Theatre	G9	Horace Lamb Bld. level 4
Rennie Lecture Theatre	C2	Johnson Laboratories, Gnd. Fl.
Schulz Lecture Theatres	G2	Schulz 7th fl.
Scott Theatre	G2	
Sterling Lecture Theatre	D15	Medical School South, Gnd. fl.
Union Hall	D9	
V.A. Edgeloe Room	M8	Mitchell Bld., 1st fl.

MUSEUMS / PUBLIC DISPLAYS

Classics	M8	Mitchell Bld., 1st fl.
Tate (Geology)	B11	Mawson Bld., 1st fl.
Dentistry	F15	Dental Hospital, Gnd. fl.
Barr Smith Library	F8	Main Foyer

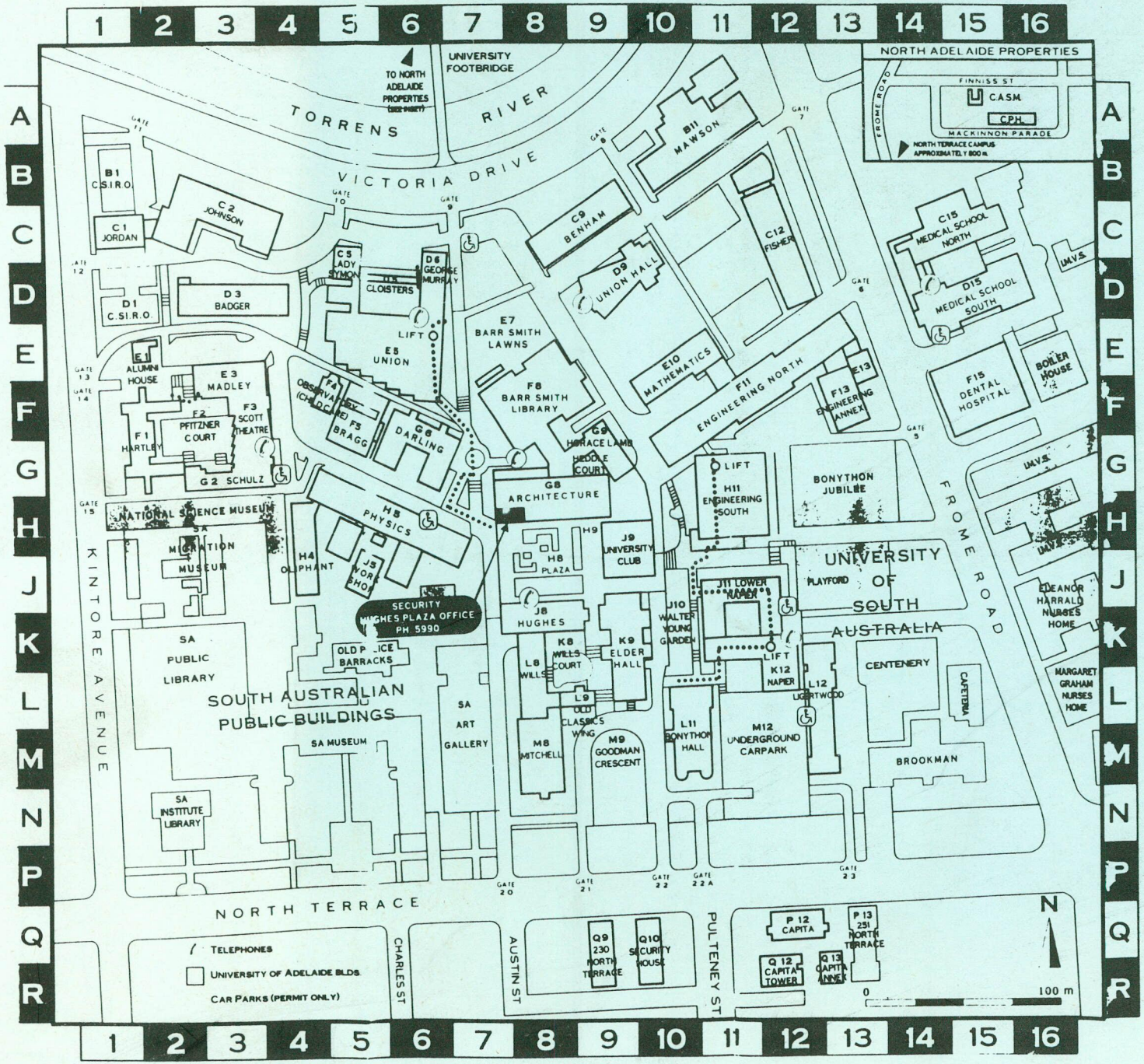
CHILD CARE CENTRES

George Murray	D6	George Murray Bld., 1st fl.
Gilberton		50 Gilbert St. Gilberton
Observatory	F4	Observatory Bld.
Rose Park		22 Watson Ave. Rose Park

RESEARCH CENTRES

Gene Technology	G6	Darling Bld., Gnd. fl.
Telecomunications Studies	F11	Engineering North, 1st fl.
Amphibian Studies	C12	Fisher Bld., 4th fl.
Asian Studies	H4	Oliphant Wing, 4th fl.
Australian Space Engineering	H11	Engineering South, 3rd fl.
Biomedical Engineering	C15	Medical Sch. Sth., 1st fl.
British Studies	K12	Napier Bld., 5th fl.
Cereal Biotechnology		Main Admin. Bld., Waite
Computer systems and software engineering	H9	Plaza Bld., level 1
Electron Microscopy & Microstructure Analysis	C15	Medical Sch. Nth., Basement
Intercultural Studies and Multicultural Education	K12	Napier Bld., 2nd fl.
International Economic Studies	K12	Napier Bld., Gnd. fl.
Languages Teaching and Research	K12	Napier Bld., 7th fl.
Materials Welding and Joining (CRC)	H11	Engineering South, 1st fl.
Sensor Signal and Information Processing (CRC)		Levels Campus, U. of S.A. c/o Faculty office
Soil and Land Management (CRC)		Main Admin. Bld., Waite
South Australian Economic Studies	K12	Napier Bld., Gnd. fl.
Information Technology, Telecommunication	H11	Engineering South, 1st fl.
Tissue Growth and Repair (CRC)	G6	Darling Bld., 2nd fl.
Viticulture (CRC)		Wine Institute, Waite Campus
Chinese Economy Research Unit	H4	Oliphant Wing, 4th fl.
Corporate and Business Law	L13	Ligertwood, 2nd fl.
Dental Statistics and Research Unit	F15	Dental Hospital, Gnd. fl.
Environmental Law and Policy Unit	L13	Ligertwood, 3rd fl.
Gallium Arsenide (VLSI) Circuit Technology	H11	Engineering South, 2nd fl.
Labour Studies Research Centre	G2	Schulz Bld., 11th fl.
National Centre for Petroleum Geology and Geophysics (CRC)		Bld. 31, Thebarton Precinct
Nat. Health and Medical Res. Council	D15	Medical Sch. Sth., 2nd fl.
South East Asian Ceramics	Q12	Capita Tower, 1st fl.
Women's Studies	K12	Napier Bld., Gnd. fl.
Basic & Applied Plant Molecular Biology		Main Admin Bld., Waite
S. A. Centre for Australian Studies	K12	Napier Bld., 4th. fl.
Teletraffic Research (Telecom)	E10	Mathematics Bld., 2nd fl.

*CRC = Cooperative Research Centre



THE UNIVERSITY OF ADELAIDE
NORTH TERRACE CAMPUS

BUILDINGS AND GROUNDS

E1	Alumni House	C2	Johnson Laboratories **
G8	Architecture Building *	C1	Jordan Laboratories **
D3	Badger Laboratories **	C5	Lady Symon Building
E7	Barr Smith Lawns	L13	Ligertwood Building **
F8	Barr Smith Library **	J11	Lower Napier
C9	Benham Laboratories	Q9	230 North Terrace
L11	Bonython Hall	E3	Madley Building
F5	Bragg Laboratories	E10	Mathematics Building *
Q13	Capita Annex	B11	Mawson Laboratories
P12	Capita Building	C15	Medical School North **
Q12	Capita Tower	D15	Medical School South **
D5	Cloisters	M8	Mitchell Building
B1	C S I R O	K12	Napier Building **
D1	C S I R O	F4	Observatory (Child Care)
G6	Darling Building *	L9	Old Classics Wing
F15	Dental Hospital	H4	Oliphant Building *
K9	Elder Hall	F2	Pfitzner Court
E13	Engineering Annex (b)	H5	Physics Building *
F13	Engineering Annex (a)	H9	Plaza Building *
F11	Engineering North *	G2	Schulz Building
H11	Engineering South	F3	Scott Theatre
C12	Fisher Laboratories **	Q10	Security House
D6	George Murray Building	M12	Underground Carpark
M9	Goodman Crescent	D9	Union Hall **
F1	Hartley Building	E5	Union House **
G9	Heddle Court	J9	University Club *
G9	Horace Lamb Building	J10	Walter Young Garden
J8	Hughes Building **	K8	Wills Court
H8	Hughes Plaza	K8	Wills Building **

ACCESS INFORMATION FOR THE DISABLED

For Assistance,
Contact Hughes Plaza Office (Security)
Building H8 Open 24 Hours
Ph 228 5990 (external) Ext 5990 (internal)

* Partly accessible (ground)

** Access by wheelchair (all floors)

••••••• Access routes upper to lower levels of campus via lifts

Disabled car park

Note: An electric wheelchair battery charging point is available at Hughes Plaza office

DISABLED TOILETS

Badger Laboratories	D3	Lower Ground
Barr Smith Library	F8	Level 3
Engineering South	H11	First Floor
Hughes Building	J8	Level 4
Medical School South	D15	Ground Floor
Napier Building	K12	Gnd, 1st Flr. & Lower Gnd
Union House	E5	Level 4
Schulz Building	G2	Ground

Produced by Technical Services Division Building Branch Printed in 1993

HYBRIDISATION INCUBATOR HI-2001

— *FEATURES* —

ACCURATE ...

Fan forced convection ensures even temperature distribution. Electronic proportional temperature controller regulates and displays the temperature to 0.1 degrees C. Rotation speed is fixed to six rotations per minute.

COMPACT ...

The HI-2001 is designed to utilize a minimum amount of bench space. It is only 320mm wide and small enough to fit under a standard laboratory bench. The controls are located on the top front panel allowing the unit to be safely placed on the floor.

EFFICIENT ...

Triple wall insulation and construction provides effective prevention of heat loss. Solid and insulated outer door, with a glass inner door, adds to its environmentally friendly and safe operation.

FLEXIBLE ...

The same rotor accepts various size hybridisation tubes. By using a tube matched to the size of the membrane, the amount of hybridisation solution can be minimised. Tubes can be made to the users specifications — please consult your representative.

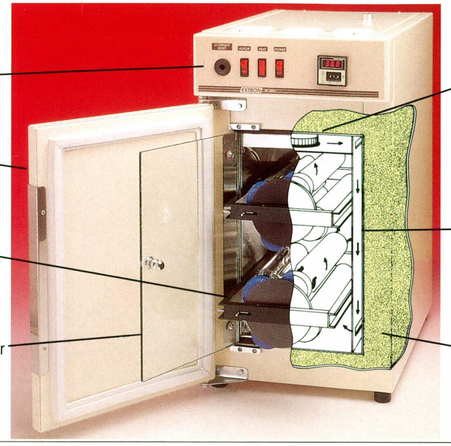
VERSATILE ...

In seconds the rotor assembly can be removed from the incubator and replaced by the optional Rocker Platform Module, which provides gentle rocking agitation for flasks and other items placed on the platform.

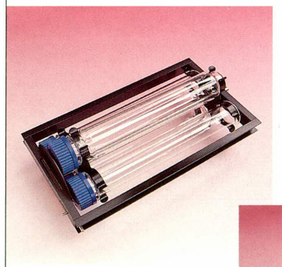
With the rotor removed the incubator is available for general use. Two trays are supplied with each unit for this purpose.

HI-2001 IS MANUFACTURED IN AUSTRALIA
SERVICED and GUARANTEED BY YOUR SUPPLIER

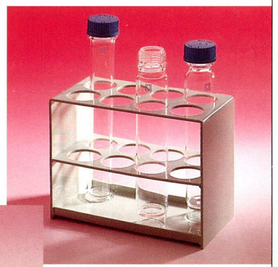
HI-2001 ENVIRONMENTALLY FRIENDLY, COMPACT AND UNIQUE



ACCESSORIES

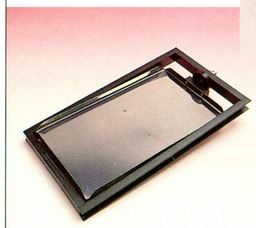


ROTOR SHELF
Cat. No. HI-1500

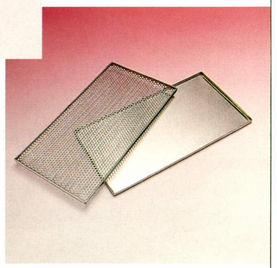


PLASTIC RACK
Cat. No. HI-1118

TUBE SIZES
8 x 40 x 300mm *or*
8 x 52 x 300mm *or*
8 x 40 x 140mm *or*
8 x 65 x 300mm



ROCKER PLATFORM
Cat. No. HI-ROCK



WIRE TRAY Cat. No. HI-1502
SPILLAGE TRAY Cat. No. HI-1501

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Please indicate your requirements:

								Sterile Filters	☐
								Selex 20 Membranes	☐
								Gel-Blotting Paper	☐
								Vertical Slab Elect. Cell	☐
								Multistaltic Pump	☐
								Power Supplies	☐
								Density Gradient System	☐
								Semi-Dry Blotter	☐
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Reinforced Nitro-Cellulose	☐			Transa-Bind	☐			Nytran-N/Polyamide	☐
				Minifold Slot Blot	☐			Minifold Dot Blot	☐

	PRICE	INFO	DEMO
HI-2001	☐	☐	☐
ITC-100 Thermocycler	☐	☐	☐
BT-1000 Biotrap Electro-elution Chamber	☐	☐	☐

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HEIDELBERG WEST

VIC. 3081

TECHNICAL DETAILS

Stainless Steel Interior

Solid State Temperature Control — Digital Read Out

Independent Overheat Control

Updraft Fan Forced Heating

6RPM Rotor — Rotation. *Optional Dual Speed.

Temperature Uniformity ± 0.25 deg. C.

Internal size H x W x D — 330mm x 200mm x 370mm

External size H x W x D — 620mm x 320mm x 500mm

Power Rating 300W

Voltage 240V 50Hz (110V 50Hz on request)

Temperature range 5 deg. C above ambient to 95 deg. C

ORDERING INFORMATION:

Cat. No. HI-2001 **Description:** Hybridisation Incubator complete with:
2 x HI-1500 Rotor Shelf
4 x HI-1520 Hybridisation Bottle 52 x 300mm
4 x HI-1115 Hybridisation Bottle 40 x 300mm
1 x HI-1501 Spillage Tray
1 x HI-1502 Wire Tray

*Optional: Dual Speed Shelf Rotors, 8 rpm Bottom Rotor, 4 rpm Top Rotor

ACCESSORIES:

Cat. No.		Cat. No.	
HI-ROCK	Rocker Platform	HI-1520	Hybridisation Bottle 52 x 300mm
HI-1500	Rotor Shelf	HI-1115	Hybridisation Bottle 40 x 300mm
HI-1501	Spillage Tray	HI-1116	Hybridisation Bottle 40 x 140mm
HI-1502	Wire Tray	HI-1117	Hybridisation Bottle 65 x 300mm
		HI-1118	Plastic Rack

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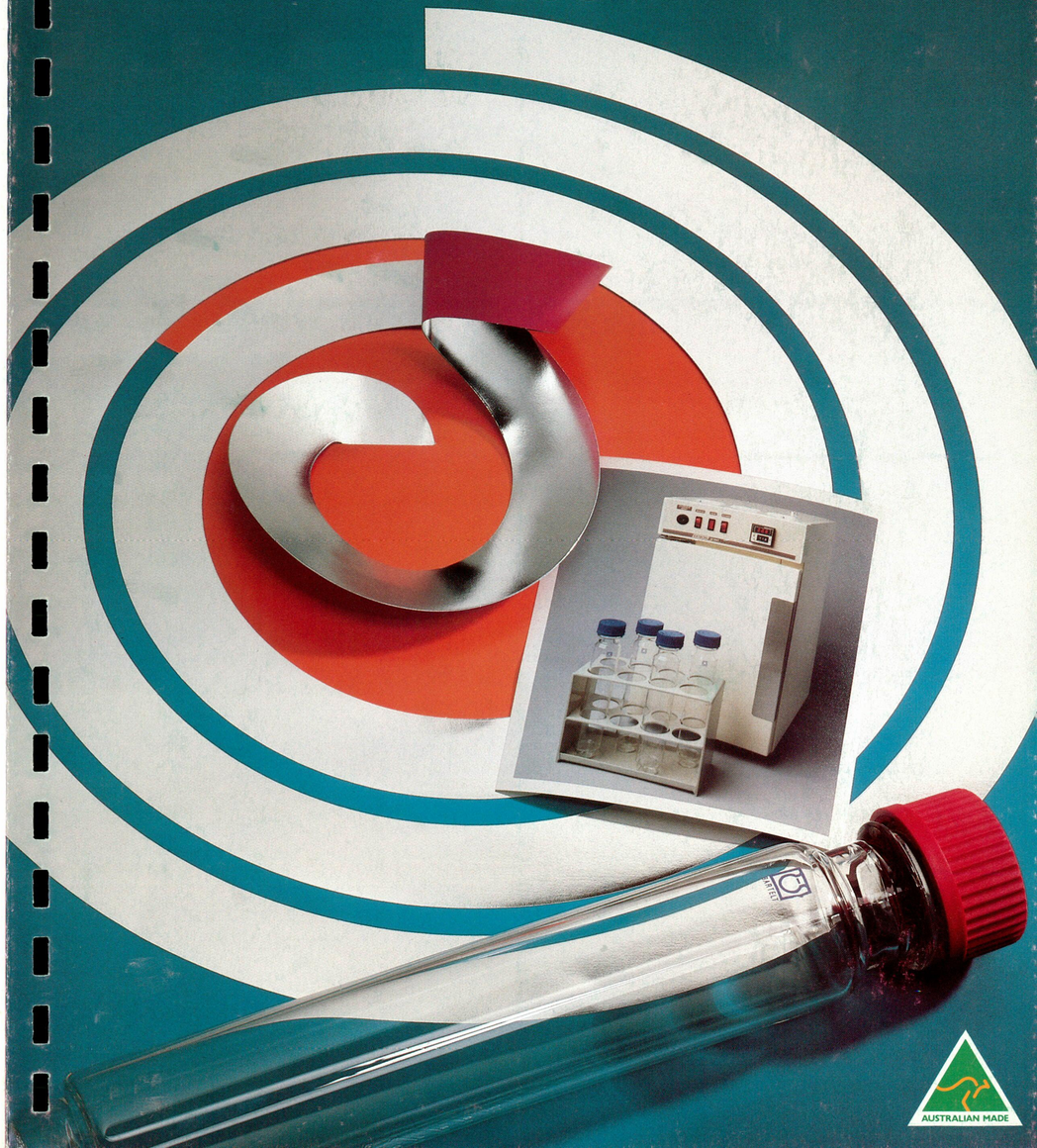
MELBOURNE, VICTORIA, AUSTRALIA, 3081
Telephone: (03) 459 8822. Fax: (03) 457 5906. TOLL FREE 008 334 862.

MINI-HYBRIDISATION INCUBATOR HI-2001



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