New Zealand Institute of
Medical
Laboratory
Science

ISSN 1171-0195

Volume 61 Number 2 August 2007

Established in 1946

New Zealand Journal of Medical Laboratory Science

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

South Pacific Congress
Auckland, New Zealand
AUGUST 21st - 24th 2007

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The Journal is abstracted by the Cumulative Index to Nursing and Allied Health Literature, Index Copernicus, Excerpta Medica/EMBASE, Australian Medical Index, and the Thomson Gale Group. The Editor and Deputy Editor are members of the World Association of Medical Editors (www.wame.org) and the Editor is currently a Board Director of WAME.

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New Zealand Journal of
Medical Laboratory Science
Volume 61 Number 2
August 2007
ISSN 1171-0195

Case studies

For debate

Regular features

In this issue

In this issue are two case studies. The first, presented by Rajani Gutha and Malati Tangirala is a case of a male presenting with a metastaic adenocarcinoma of unknown origin with a very high level of serum prostate specific antigen (PSA) being the only indicator of malignancy. This man was treated for prostate cancer and had his PSA followed over many years. His fluctuating PSA levels over time correlated with his clinical status at those time points.

The second case study presented by Barbara Hoy was of a 64-year old male in whom the only significant laboratory finding was an artefactually low HDL cholesterol. This led the laboratory according to an established protocol to do more tests which resulted in a diagnosis of multiple myeloma.

Campylobacteriosis is a serious public health problem in New Zealand. In order to tackle this severe epidemic it is essential that effective interventions are found to protect the New Zealand population. For that to happen it is imperative that the main primary source or sources of this infection are identified. In this issue Michael Baker and Nick Wilson argue, and present epidemiological evidence, that fresh poultry is the most important source of human Campylobacter infection in New Zealand and that fresh poultry needs to be removed from the human food chain to control the epidemic. Warrick Nelson and Ben Harris argue that although fresh poultry plays a role in human Campylobacter infection, other sources and vectors, such as excrement from cows, should be considered. As well as presenting their case, both sets of authors have had the opportunity to comment on each others articles before publication and their responses accompany the two articles. Let us know, as letters to the Editor, which side of the argument and why you support one or the other.

As from this issue the three Universities (AUT, Massey and Otago) are given their own column in which they can inform the readers with news from their Schools of Medical Laboratory Science. Inside are contributions from AUT and Otago. It is hoped that this will be a regular feature and we look forward to hearing from the three Universities who supply our medical laboratory science graduates.

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med·bio

Med-Bio offers an award for the best article in each issue of the *New Zealand Journal of Medical Laboratory Science*. All financial members of the NZIMLS are eligible. The article can be an Original, Review or Technical Article, a Case Study or a Scientific Letter. Excluded are Editorials, Reports, or Fellowship Treatises. No application is necessary. The Editor and Deputy Editor will decide which article in each issue is deemed worthy of the award. If in their opinion no article is worthy, then no award will be made. Their decision is final and no correspondence will be entered into.

Serial serum prostate specific antigen measurements over time in a patient presenting with a metastatic adenocarcinoma of unknown origin

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[2]Department of Biochemistry, Nizam's Institute of Medical Sciences, Hyderabad, India

Abstract

A 60- year male presented with a mass in the lower abdomen and was referred to the Biochemistry department, Nizam's Institute of Medical Sciences, Hyderabad, India for serum prostate specific antigen (PSA) estimation. The referring physician had made the following differential diagnoses: non-Hodgkin's lymphoma, prostate carcinoma, and cancer with unknown primary. A very high serum PSA was the only indicator of prostatic malignancy, histopathology being inconclusive.

The patient was treated for prostate cancer. The PSA concentration declined to normal levels as the patient responded to treatment. He was in remission for seven years but later developed bone metastases. In total the patient was followed up over a period of eight years with serial PSA measurements. The PSA correlated well with the clinical stage of the patient during stable course and recurrent stage of the disease.

Key words: prostate specific antigen, prostate carcinoma, unknown primary cancer

N Z J Med Lab Sci 2007; 61 (2): 31-2

Introduction

Prostate cancer is the second most common malignancy in men globally. It is known to have increased prevalence with advancing age. Prostate specific antigen (PSA) was discovered in 1979 and by the late 1980's its prognostic importance and role in monitoring prostate cancer was established (1,2). However, the role of PSA in screening remains controversial (3-6). In this paper a case study is presented in which PSA established the diagnosis of metastatic prostatic cancer and provided information on the course of the disease and response to treatment, between 1995 and 2003.

Case history

A 60- year old male presented with a mass in the lower abdomen and was referred by the physician to the biochemistry department at the Nizam's Institute of Medical Sciences Hyderabad, India for serum PSA estimation. Serum PSA was measured with a one step double monoclonal antibody enzyme immuno-adsorbent assay (ELISA), using the Enzymun-Test Kit PSA on the Boehringer Mannheim ES300 immunoassay batch analyser. The reference interval for PSA in our laboratory was 0.3 to 4.0 ng/ml.

Ultrasound of the abdomen had suggested bilateral iliac and pelvic lymph adenopathy. A CT scan confirmed huge pelvic and lower para aortic lymphadenopathy. The physician made three differential diagnoses: non-Hodgkin's lymphoma, prostate carcinoma, and cancer with unknown primary. The serum PSA analysis revealed a very high value of 825 ng/ml, although histopathological examination showed only metastatic adenocarcinoma with unknown primary. In April 1995, the patient underwent bilateral orchidectomy as an appropriate treatment for prostatic cancer. In May 1995 and August 1995 PSA values were 34 ng/ml and 22ng/ml respectively.

A further decline in PSA to 4.2 ng/ml and then to 0.8 ng/ml was noticed between October 1995 and April 1996. However, in August 1996 the patient presented with pain near the left pelvic girdle and the PSA had increased to 32 ng/ml. He was treated with Flutamide (a potent anti-androgen). From November 1996 to January 2002 normal PSA levels (2.8ng/ml, 0.4 ng/ml, 0.5 ng/ml, 0.8 ng/ml, 0.6 ng/ml and 0.08 ng/ml) were observed and the patient was clinically stable during this period (Figure 1).

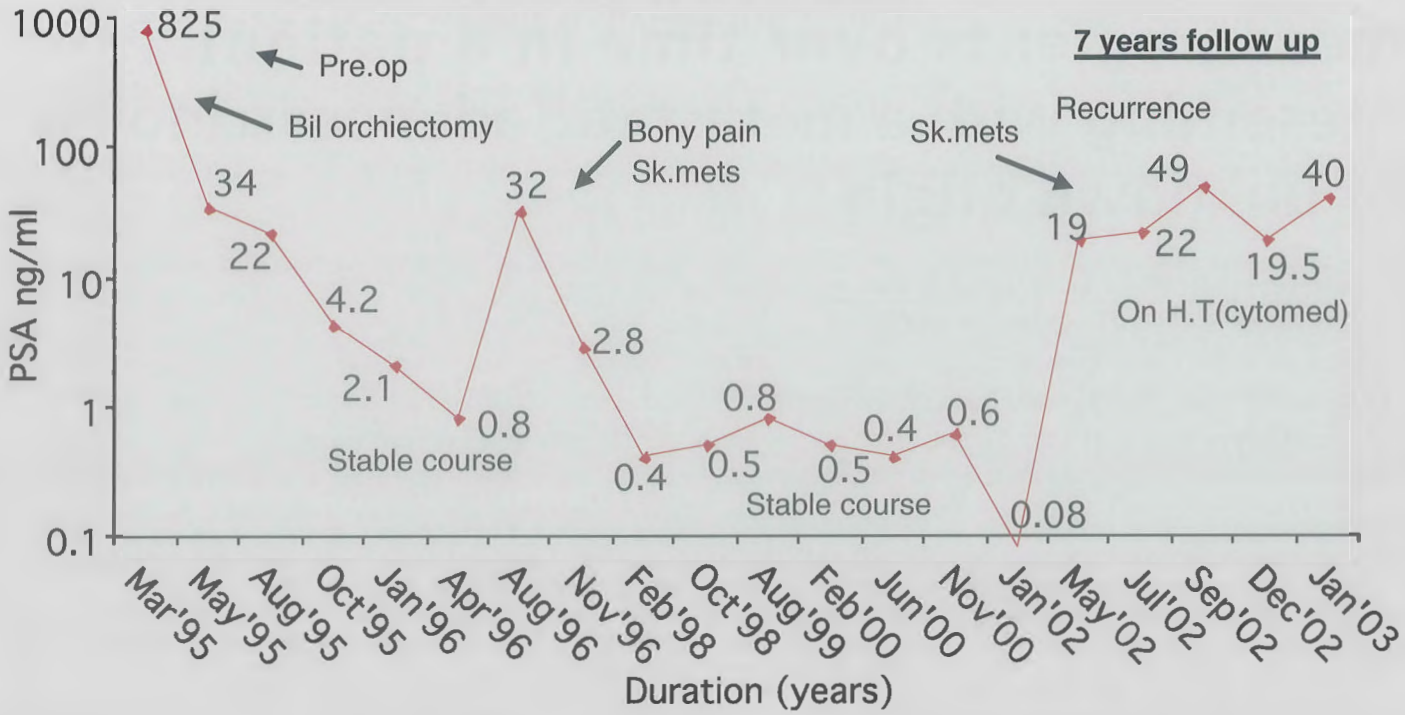
In May 2002 the patient complained of weakness and leg pain and the PSA had increased to 19 ng/ml. He was started on Cytomid (an anti-inflammatory and anti-androgen). A bone scan revealed multiple skeletal metastases. During July 2002 and September 2002 a progressive rise in PSA levels was observed (22 ng/ml and 49 ng/ml). The total alkaline phosphatase (ALP) was borderline elevated at 132 U/L while the ALP bone fraction was elevated at 77%. ALP was estimated on a Merck Micro lab 200 semi auto analyzer using the manufacturer's reagents. The reference interval for ALP is 38 to 126 U/L. ALP iso-enzymes (bone and liver) were separated by electrophoresis and the fractions were quantitated by scanning of the electrophoresed gels with a Helena densitometer at 595nm, using the ALP isoenzymes kit supplied by the manufacturer. The reference interval for the bone fraction is 23 –67%.

The patient had swelling in the inguinal region and bone pain. The patient was subjected to 25 exposures of radiation therapy as well as hormone therapy consisting of Cytomid. In December 2002 his PSA slightly declined to 19.5 ng/ml. In January 2003 swelling in the inguinal region was reduced but the PSA rose to 40 ng/ml along with a rise in total ALP (641 U/L) and bone ALP fraction (80%).

Discussion

Although the patient's prostate cancer could have been detected earlier by a screening program, there is a considerable debate regarding the necessity or even desirability of screening to detect prostate cancer in cases of asymptomatic individuals (3-6). PSA is considered as an ideal

Figure 1. PSA levels over 8 years



tumor marker for monitoring prostate cancer and assessing response to treatment (7,8). A study by Malati and colleagues during a screening programme in India on South Indian males aged between 40 to 80 years emphasized the importance of a multidisciplinary approach using digital rectal examination, ultrasound and PSA measurement but did not recommend for or against screening (9).

This case study clearly indicates the importance of PSA estimation as a preoperative marker as well as a marker for follow-up during different stages of the cancer such as remission, stable course of the disease, and recurrence. This patient's clinical presentation was unusual and histology was unhelpful in reaching a diagnosis. The high PSA confirmed the presence of prostatic cancer and was very useful in monitoring successful treatment and the course of the disease over a seven year period.

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The diagnostic footprints – a case study

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Medlab, Hamilton

Abstract

The only significant laboratory finding in a 64-year old male was an artefactually low HDL cholesterol. Laboratory protocols were followed step by step, leading to a diagnosis of advanced multiple myeloma. In this case study, where no laboratory tests were initially requested that could have led to this diagnosis, false low HDL cholesterol was a diagnostic tool rather than a reagent problem.

Key words: high density lipoprotein (HDL), total protein, immunophoresis, protein electrophoresis, multiple myeloma, monoclonal gammopathy.

N Z J Med Lab Sci 2007; 61 (2): 33-5

Case History

A 64-year old male presented to his GP with nausea, weight loss, and generally feeling unwell. His GP ordered a range of blood tests covering most organ systems: kidney function, electrolytes, thyroid function, glucose, ferritin, B12 and folate assays and a complete blood count.

Parameter Haematology	Initial result	Reference interval	
Haemoglobin	**106**	130-175	g/L
RBC	3.8	4.0-6.0	$x10^{12}$/L
Haematocrit	0.33	0.37-0.50	
MCV	85	80-99	fL
MCH	28	27-34	pg
Platelets	405	160-400	$x10^{9}$/L
WBC	5.1	4.0-11.0	$x10^{9}$/L
Neutrophils	3.9	2.0-7.0	$x10^{9}$/L
Lymphocytes	0.8	1.0-4.0	$x10^{9}$/L
Monocytes	0.3	0.2-1.0	$x10^{9}$/L
Eosinophils	0.1	0.0-0.7	$x10^{9}$/L
ESR	27	1-30	mm/Hr

A few macrocytes and target cells.

Biochemistry			
Cholesterol (Total)	6.58	4.28-5.46	mmol/L
Triglycerides	2.45	0.62-2.59	mmol/L
LDL Cholesterol	5.00	2.46-3.96	mmol/L
HDL Cholesterol	**0.47**	0.85-1.85	mmol/L
Chol/HDL Ratio	14.0	<5.5	

HDL cholesterol result is unreliably low due to interfering proteins/ drugs.

Laboratory Protocol

If an HDL cholesterol is <0.5 mmol/L look at the reaction monitor and history of the patient. If the patient is not a known myeloma case, and the reaction monitor is abnormal, then a total protein is added and the specimen sent to immunology for protein electrophoresis.

Reaction Monitor

We use the Roche product HDL-C plus 2nd generation (1). The leaflet states: In very rare cases gammopathy, in particular type IgM (Waldenstrom's macroglobulinaemia), may cause unreliable results.

Test principle : Homogeneous enzymatic colorimetric test.

- Sample and addition of R1 buffer. In the presence of magnesium sulfate, dextran sulfate selectively forms water-soluble complexes with low density lipoprotein [LDL], very low density lipoprotein[VLDL], and chylomicrons which are resistant to polyethylene glycol [PEG] modified enzymes.
- Addition of R2 [PEG-modified enzymes/4amino-antipyrine/ buffer] and start of reaction. The cholesterol concentration of HDL-cholesterol is determined enzymatically. In the presence of peroxidase, the hydrogen peroxide generated reacts with 4-amino-antipyrine and HSDA to form a purple-blue dye. The colour intensity of this dye is directly proportional to the cholesterol concentration and is measured photometrically.

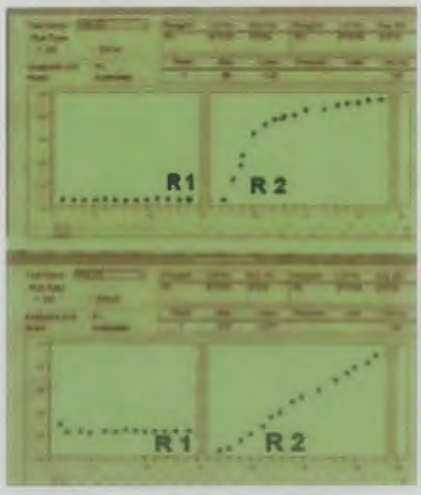

Figure 1. Normal HDL cholesterol (top). Patient's abnormal HDL cholesterol reaction monitor. Note loss of cholesterol between R1 and R2

Electrophoresis

In our laboratory a total protein of >80 g/L and/or a globulin is >40 g/L, without a known patient history, is also a criterion for protein electrophoresis. In this patient the total protein was 93 g/L and serum globulin was 56 g/L. Electrophoresis showed a discrete band in the gamma region comprising 31.5% of total. Immunofixation diagnosed the gamma band as IgG Kappa paraprotein. At this stage the doctor was contacted for explanation and further testing.

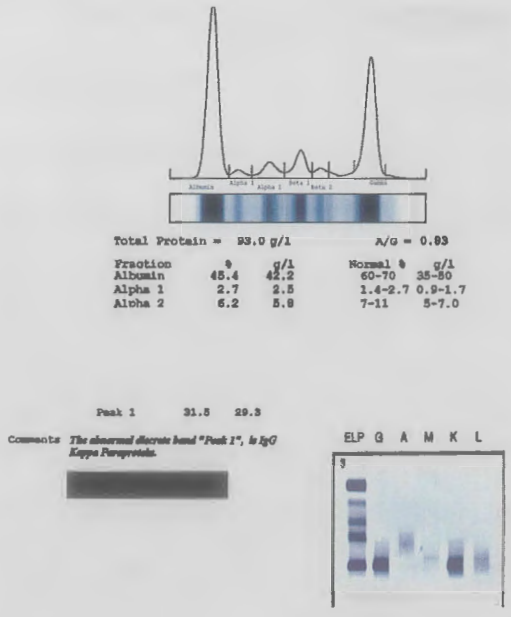

Figure 2. Serum protein electrophoresis

Laboratory Protocol

A bone marrow may be indicated when there is a significant monoclonal band present. The bone marrow on this patient showed a marked number of myeloma cells comprising 85% of total white cells. Many of these plasma cells were binucleated with some multinucleated, forming plasmacytomas. A diagnosis of advanced multiple myeloma was made.

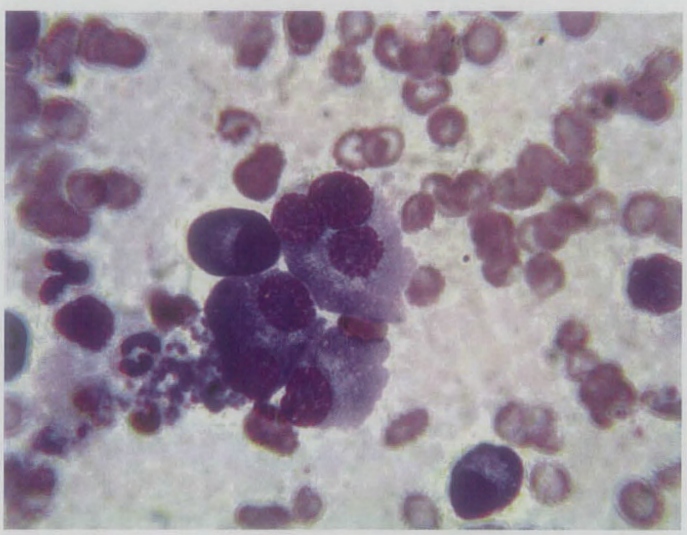

Figure 3. Bone marrow showing multinucleated plasma cells

Discussion

Multiple myeloma is a blood cancer in which plasma cells replicate uncontrollably and accumulate in the bone marrow (2). Plasma cells are derived from B lymphocytes whose normal function is to secrete antibodies or immunoglobulins that help fight disease. Immunoglobulins are composed of four protein chains, two long "heavy" chains and two shorter "light" chains. Myeloma cells also secrete immunoglobulins, but since the cells are monoclonal, i.e. derived from a single plasma cell, they all produce the same immunoglobulin protein (IgG, IgA, IgM or IgD) in large amounts. The monoclonal IgM does not help protect the body from infection, but can build up in organs such as the kidneys, causing serious damage over time. In some cases myeloma cells secrete immunoglobulins that contain only the light chains, these are secreted in the urine and are known as Bence Jones proteins (2).

Multiple myeloma can cause many complications. This constellation of signs and symptoms is commonly referred to as CRAB. These, and other complications are described below.

Complications (CRAB) (2).

Calcium elevation. Bone destruction results in release of calcium into the blood causing weakness, fatigue, nausea, loss of appetite and confusion.

Renal dysfunction. Excess proteins and high blood calcium can cause renal damage. Impaired renal function is a common complication, affecting about 40% of patients in the course of the disease.

Anaemia. Accumulation of myeloma cells in the marrow can interfere with production of normal cells. These deficiencies can cause chronic anaemia, increased susceptibility to infections and excessive bleeding.

Bone Disease. The most troubling symptom of myeloma is bone pain. Osteolytic lesions and inhibition of new bone formation makes bones highly susceptible to fractures. Fractures of the vertebrae can result in increased pressure on the spinal nerves, causing numbness, tingling, pain or muscle weakness in the lower extremities.

Other complications include hyperviscosity syndrome caused by excessive amounts of protein production. This gives rise to bleeding from the nose and mouth, blurred vision, stroke like symptoms and congestive heart failure. Hyperviscosity syndrome can be treated with plasmapheresis which removes excess proteins from the blood.

Treatment

There is no cure for multiple myeloma but an aim of prolonging the survival rate. The key objectives are: kill tumour cells, control tumour growth, control pain and other disease related symptoms, allow patient to have active and good quality life.

Treatments are: chemotherapy, and autologous stem cell transplant which involves collection and transplantation of patients own stem cells. The median survival rate is 3-4 years.

This disease is the second most commonly diagnosed blood cancer after non- Hodgkin's lymphoma. There is a slightly higher incidence in males and the median age is 65. Most people have no known risk factors other than age (3).

Conclusions

In spite of an absence of other abnormal laboratory results initially, this patient was diagnosed with the serious disease, Multiple myeloma, using a low HDL cholesterol as a initial tool to follow the laboratory protocol steps through to diagnosis.These protocols are summarized as follows:

- HDL <0.50 mmoL, check graph and patient history
- Add total protein, if >80 g/L and/or globulin is >40 g/L, add protein electrophoresis
- Immunofixiate if a monoclonal band is present
- Bone marrow may be indicated if IgG, IgA, or IgM is significantly increased.

Acknowledgements

Peter Hale, whose knowledge and expertise set the laboratory protocols and this journey to diagnosis. Doug Napier, for Immunology, for results and discussion.

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Is chicken meat the most important source of human Campylobacter infection in New Zealand?

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It is undisputed that chicken meat plays a role in human campylobacteriosis. However, the claim that *"there is overwhelming epidemiological and laboratory evidence that fresh chicken is the dominant source of human infection"* (1) is open to question. For chicken as the prime source theory to be accepted, the considerable anomalies associated with this would have to be explained away. Other sources and vectors of campylobacteriosis in New Zealand should at least be properly considered. For instance, the largest reservoir of campylobacter in New Zealand is likely to be the 5.2 million dairy cows (an increase of 79% in 25 years – www.stats.govt.nz), producing excrement equivalent to at least 70 million people. This excrement is deposited into our waterways, either directly by cows accessing streams, or indirectly from washing off our clean, green pastures.

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Summary of anomalies to chicken as only source of campylobacter infections

- Campylobacter infections have very marked seasonal cycles, but chicken consumption does not.
- The degree of seasonality varies, roughly less seasonal in the North to greater inter-seasonal variation in the South (2).
- Where poultry production has stopped (e.g. Belgium 1999 Dioxin crisis), campylobacter rates have only dropped up to 40%, leaving at least 60% non-poultry source unexplained.
- These campylobacters cannot grow at all under 30°C, less than most peak spring and autumn temperatures, so poor food storage multiplication cannot be an issue (unlike other food poisoning bacteria).
- Each 1°C ambient temperature increase results in a 5% increase in campylobacter infections up to 14°C (3) – but the bacteria do not multiply below 30°C, so what cause and what vector?
- Over 80% of human cases are sporadic cases, not outbreak clusters – sporadic incidents have sporadic causes.
- When outbreaks have occurred, the sources have been traced to contaminated water, unpasteurised milk, processed meats and only sometimes chicken.
- New Zealand campylobacter infection rates increased 1995-1999, but dropped dramatically in 1999 and 2000, unlike chicken production and consumption which increased steadily.
- Molecular typing shows the same strains in chickens, humans, animal wastes, water and flies – which came first?
- Food handlers and preparers of "filthy chicken" are not at increased risk of infection.

Introduction

Campylobacter jejuni is typically found to cause about 80% of campylobacteriosis cases. At least 10% are thought to be from C. coli, with a few cases from other species.

Common features of campylobacteriosis are:

- a marked seasonal variation in cases with a small winter/spring peak and a major summer peak.
- very high rates in infants and the age group 20-40 years.
- higher rates in males, generally across all age groups.
- at least 80% of cases are sporadic rather than outbreak in nature (unlike other food poisoning causative agents).
- relatively low levels of incidence from the 1980s, but growing erratically through to the present.
- the infectious dose is very small, possibly as low as 500 cells (4-6), and
- a very strong and persistent association with chicken consumption.

These features are commonly reported for many countries. New Zealand shows similar trends, but notably at very much higher rates - European and North American rates are of the order of <100/100 000 while New Zealand is >400/100 000 (7).

Campylobacters are also a significant cause of Travellers' Diarrhoea. Foreign travel is such a significant source of campylobacteriosis that some countries record campylobacteriosis separately according to whether it was acquired domestically, or during or after travel. About 25% of cases in the UK are estimated as travel related (8) and up to 38% in Norway (9).

These *Campylobacters* do not multiply in foods. In fact they cannot multiply at all at temperatures under 30°C, nor outside of animals or birds (other than very carefully contrived laboratory conditions). Thus they are quite different to many other "food poisoning" bacteria in that the infectious dose is delivered entirely as contamination of the food, not multiplication in foods. Furthermore, symptoms follow the establishment of infection only, and not ingestion of toxins commonly produced by bacteria eg *Staphylococcus*. Even where foods are at a temperature sufficient to allow *Campylobacter* growth (optima are at 37°C and 42°C, typical body temperatures of mammals and birds respectively), no trials have demonstrated growth in food (10). Growth occurs in the intestinal tracts of birds and mammals – hence jejunum and coli species names. *Campylobacter* are readily killed at relatively low cooking temperatures – i.e.70°C (11). They are also only present on the surface of meats (12-13), the only clearly demonstrated exception is liver (14-15). Processed meat products (eg sausages, patés, burgers) therefore mix surface meat contamination into the bulk of the product.

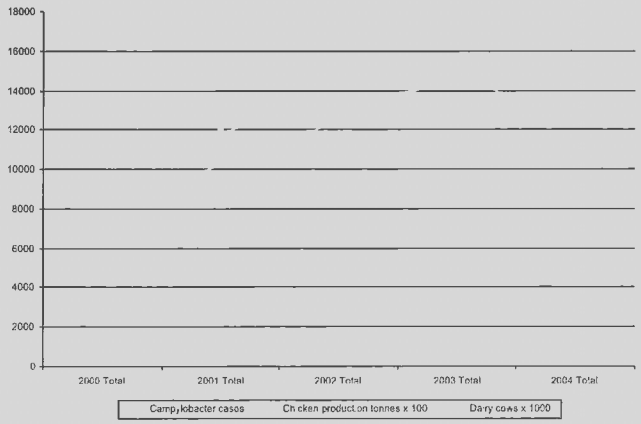

Figure 1 Annual campylobacteriosis cases with fresh chicken production (tonnes x 100) and dairy cow numbers (cows x 1000).

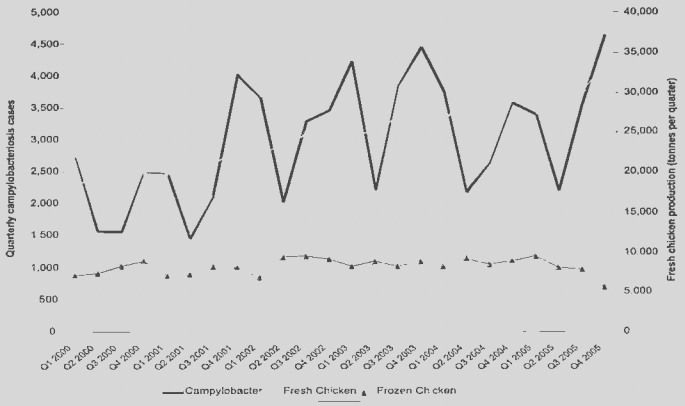

Figure 2 Campylobacteriosis cases and chicken production. There is clearly no correlation at all between these trends at this level.

New Zealand evidence

Laboratory evidence in New Zealand is particularly poor as regards chicken as the source of human cases. Routine clinical stool samples are typically identified to genus level only, using a selective medium for *C. jejuni* and *C. coli*. When methods suited to identifying other species are used, they are often found in clinical cases (16). Perhaps the advent of multiple species PCR tests now being used in research will assist in clinical diagnosis too[18].

Research laboratory evidence, while indicating a significant commonality between chicken and clinical strains, does not indicate chicken as source, but merely that similar strains are present in both sets of samples. Which came first – the chicken, the egg, the cow, the water, the fly? An early survey showed that some types were indistinguishable between clinical strains and those from chickens and untreated water. However, the strains most commonly isolated were also common in dairy and beef cows, sheep, water and chickens (17-18). Subsequent testing has confirmed the presence of common human isolates in sheep liver (19) and beef, sheep, pork and chicken sources (20).

Epidemiological studies in New Zealand come up with a bewildering array of risk factors. Chicken certainly features prominently, especially raw or undercooked chicken (21). This large case-control study, the most extensive in New Zealand to date, has been criticised for not fully considering other potential sources, such as animal and water-related risks (22). A further problem for this sort of study is the reliance on memory to determine food events and other activities that could affect the resulting risk profile (23). Risk assessments based on reservoir identification have not helped for *Campylobacter* as they are so widely spread, outbreak data are not representative of a largely sporadic disease, and case-control studies are problematic due to recall bias and long exposure windows (24). The bias towards blaming chicken can be even more blatant, for example a public health questionnaire to obtain information on the source of campylobacteriosis helpfully prompts *"Campylobacter infection is most commonly associated with undercooked or left-over chicken meals"* (25).

There have been many literature reviews conducted in New Zealand recently. While all mention the chicken/human disease link, none appears able to apply a percentage of cases to chicken specifically, nor do they specify chicken as the definitive major source for campylobacteriosis in New Zealand (26-30). On the contrary, many other sources are acknowledged, particularly dairy and sheep as probable direct and indirect sources. An older study raised the strong potential for domestic pets as a significant risk factor (31), a risk factor receiving renewed interest internationally (32-35). One study even observed that, at least some New Zealanders *"live in an environmental sea of Campylobacter"* with no significant overlap in types isolated from humans and raw chickens (17).

Outbreaks, while commonly considered to be less than 20% of campyobacteriosis cases, are rather better understood. Common identified sources in these cases are water, unpasteurised milk and processed meats (especially those not cooked after processing and/or that contain liver). Chicken as an outbreak source is still sufficiently uncommon to justify reporting, for example recent events in Australia, Denmark and Japan (36-38). Outbreaks in New Zealand (reviewed in reference 30)) are largely related to water sources, and foods from commercial or other catered events, only some of which included chicken.

Clearly there is no consensus amongst New Zealand campylobacteriosis researchers of any "overwhelming" *evidence to indicate that regulation of chicken contamination will control New Zealand's campylobacteriosis epidemic*", although it will probably go some way to reduce it.

The marked seasonal pattern of campylobacteriosis cases does not correlate with chicken consumption. The annual growth in cases may correlate with an annual growth in fresh chicken production (1), but this is clearly negated by the quarterly data (Figs 1 and 2). Where seasonal patterns of chicken colonisation have been monitored, they occur at the same time, or slightly after, campylobacteriosis cases (39), strongly suggesting a common source and dispersion agent. Curiously, seasonal campylobacteriosis infection does correlate well with numbers of short term visitors to New Zealand (Fig 3 of reference 40), but the New Zealand notification data does not have this case detail, unlike several overseas countries which show the strong link.

Rates of disease in New Zealand of various age groups are remarkably constant (www.nzpho.org.nz report server). The highest rates occur in the under five years group, and the 20-29 years group, across both males and females. It seems unlikely that these age groups will be consuming and/or preparing more chicken meals than other age groups.

Overseas evidence

If the New Zealand data is somewhat sporadic and gives no clear picture of the source of campylobacteriosis, the Iceland and Belgium events are often cited as being definitive against chicken.

Iceland revised its law in 1996 to allow the sale of fresh as well as frozen uncooked chicken meat. The following years showed a massive increase in campylobacteriosis cases, peaking in 1999. Rates dropped subsequently, associated with a major campaign by chicken producers to reduce contamination of retailed chicken meat products and public health campaigns aimed at food and personal hygiene (41). Iceland data must however be considered with some care – not any aspersion

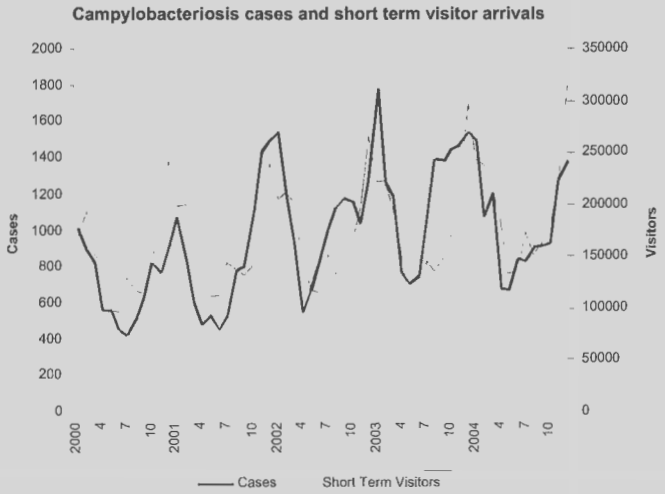

Figure 3 The uncanny correlation between New Zealand campylobacteriosis cases and tourist arrivals with a Spearman's Rank Correlation of 0.68.

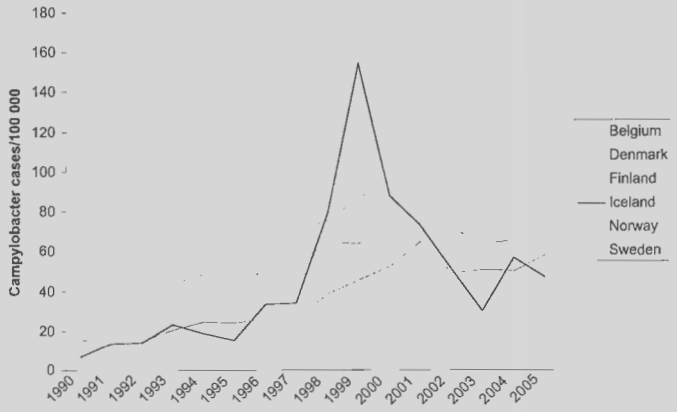

Figure 4 Rates of campylobacteriosis for a range of temperate European countries. Note the trend in rising rates, generally up to 2001.

on the source, but simply that the population is so small (total 300 000 i.e. less than Christchurch) that a few extra individual cases will have a large impact on the rate calculation. The peak of 1999 was only 426 cases – about a third of the number of cases reported in the Canterbury District Health Board area. In spite of this, other circumstantial evidence, for example the increased consumption of chicken and the growth of the fresh chicken component from <5% in 1996 to 60% by 1999, is strongly indicative of the role of chicken in Icelandic domestically acquired campylobacteriosis (42). However, this occurred within a background of smaller, but similarly timed, peaks in campylobacteriosis in many other European countries. Although campylobacteriosis reports have dropped since intervention measures have been implemented, they only dropped to a level roughly double that pertaining prior to pre-intervention 1996 (Fig 4, data.euro.who.int/CISID).

During the 1999 "Dioxin crisis" in Belgium, all Belgian-produced chicken products were withdrawn from the market for a six week period. An approximately 40% reduction in campylobacteriosis reports occurred during those six weeks, returning to trend following the return of chicken meat to market (43). However, many other food products were affected, not just raw chicken meat. May 29th 1999 (week 21) was the date for withdrawal of Belgian-produced poultry and eggs (Belgium is a net exporter of poultry products). June 2nd (week 22) saw the withdrawal of products extended to processed foods containing chicken and egg, and the withdrawal extended again two days later to include processed pork and beef products. Bearing in mind an incubation period of 2-7 days, it is interesting to note that campylobacteriosis reports only dropped from week 23, the week following the withdrawal of processed foods, rather than the week following the withdrawal of chicken. The 40% decline in campylobacteriosis rates cannot therefore be attributed solely to people not eating chicken, and even if it does, the majority 60% of cases are still left with an unexplained non-poultry source. These events also still leave the possibility of disease associated with the style of eating rather than indicating the chicken itself is the source. Indeed, the removal of processed foods eaten without further cooking, and replacement by beef or fish products for chicken in fast food outlets, could indicate a role for food-associated disease (44).

A number of other studies raise concerns about the overwhelming expectation that campylobacteriosis comes from chicken consumption. For example, Neal and Slack (45) determined for the Nottingham Health District, UK that foreign travel accounted for 25% of cases, and 15% of cases could be attributed to causes such as contact with puppies, eating chicken and drinking milk from pecked bottle tops, leaving the other 60% unexplained. Note also that eating chicken is only a component of the explained 15% of cases. A large study of the Helsinki, Finland region (46) found a fairly large overlap between PFGE genotypes between humans and chickens, but often not occurring at the same time. Their conclusion was that both humans and chickens probably acquire *Campylobacter* infection from a common source, rather than indicating chicken as a direct source for humans (or vice versa).

Similarly, a study in Quebec, Canada (47) also showed a high diversity of PFGE genotypes in chickens, but with single growing houses tending to have single strains. Comparison with human sources indicated only 20% of isolates were related to chicken strains. The Czech Republic has campylobacteriosis rates approaching those of New Zealand, rising from 22/100 000 in 1996 to 296/100 000 in 2005 (data.euro.who.int/CISID). Chicken infection patterns are again similar to Finland and Canada with each flock infected with a single clone (48). However, using both PFGE and PCR/RFLP methods, chicken and human genotypes overlapped by only 6%. The authors concluded that *"chicken meat does not represent as important a source of campylobacteriosis as was previously believed"*.

Cross contamination and elimination?

Another anomaly is that handling and preparing chicken does not appear to be a risk factor for disease. Since anything up to 100% of raw chicken can be contaminated, it seems strange that food preparation is not a risky business. Analysis of genetic strains suggests that very small outbreaks are likely being reported as sporadic (49-50), possibly indicating cross-contamination in domestic kitchens. Tests indicate that *"during food preparation bacteria become widely disseminated to hand and food contact surfaces"* (51) and transfer rates from hands or kitchen utensils to ready-to-eat foods are up to 27% (52). Poultry is a common food item which is contaminated to a high level, with an easily spread and cross-contaminated bacterium requiring a low number of cells needed for infection, yet actual cases of campylobacteriosis are remarkably few compared to the number of chicken meals consumed.

Banning the retail sale of fresh, raw chicken meat has been suggested (1). Reduction of campylobacter numbers on chicken meat can occur during freezing. This is clearly the assumption in the Icelandic experience, both from the ascribed source of the epidemic and one intervention being freezing of meat from flocks testing positive before slaughter (41). Early tests appear to confirm that freezing results in fewer contaminated birds, for example 48% contamination of fresh and 4% of frozen chickens (53), although possibly better isolation tests suggest far higher numbers of frozen chickens are contaminated,

for example 94% of fresh and 77% of frozen chickens in a small Irish survey (54). A 0.5 to 3.4 log reduction in *Campylobacter* following commercial freezing over a 2-week period looks promising, although the authors noted that *"refrigeration and freezing are not a substitute for safe handling and proper cooking of poultry"* (55). A similar 3 log reduction was noted for contaminated ground beef (11). Apart from contamination in livers, chicken meats are contaminated on the surface. Some chemical treatments applicable for use at slaughter could prove promising to reduce contamination to undetectable levels (56).

If not chicken, what else can be the source and vector?

It has not been our intention to suggest that chicken meat is not a source of *Campylobacter*, but simply to show that there are significant difficulties associated with ascribing the bulk of the blame to chicken meat. There is certainly no doubt that campylobacteriosis can occur from consuming raw/undercooked chicken meat, or consuming other foods contaminated with raw chicken meat or juices.

Quite clearly, animal (including cows, sheep, pets and humans) and bird excreta are the ultimate source. For human infection to occur, we need both a source and a vector. Other factors, such as different pathogenicity or increased susceptibility (57), including possible immunity reactions (58) play a role in determining actual disease status following colonisation.

Common farm animals, especially cows, must be considered a major source. They are present in large numbers, over 9.5 million cattle in New Zealand, their faeces commonly carry *campylobacter* that is seldom treated to reduce bacteria. Raw excrement is generally deposited directly into the environment, or is purposely spread to both dispose of it and to fertilise pastures. Dairy cows alone (now 5.2 million) are producing effluent equivalent to at least 70 million humans that is not being treated before discharge into the environment (22). Cows and sheep are clearly major sources for environmental, and especially water, contamination in New Zealand (27,59). These would appear to be the true source of the *"environmental sea of Campylobacter"* quoted earlier. Further, dairy herds show a marked seasonal pattern of campylobacter shedding (60-61).

We also need to reconsider the vector(s). In New Zealand, it would appear that water and flies, however currently unfashionable compared to chicken consumption, are strong candidates as significant vectors. Sparrows are a very mobile vector for transferring campylobacter from rural to urban areas, although what part they might play in human infection is less clear (62). The reason for water is that significant sections of the New Zealand population are normally, or frequently, exposed to untreated drinking water supplies (including shallow groundwater wells), and even treated water can have detectable levels of *Campylobacter* contamination (63). This could also go some way to explain the correlation between campylobacteriosis and visitor numbers since many small water supplies service holiday and visitor attractions for both potable water and recreational/swimming activities.

Addressing the anomalies in the chicken story and considering other possible sources and vectors is unlikely to generate a *"miasma viewpoint"* likely to be *"paralysing and easily exploited by interest groups"*(64). We would hope to stimulate research into other sources, possibly more likely to offer a means of reducing campylobacteriosis infection rates more significantly.

Conclusions

Chicken meat is undoubtedly one significant source for *Campylobacter* causing human disease. Where detailed studies have been conducted to establish chicken as the direct source of disease, results indicate from 6% to possibly 40% of cases should be attributed to chicken consumption. Of greatest significance for New Zealand is the growing data indicating why New Zealand has such a high rate of disease, and the environmental sources driving the pattern of infection.

Poultry meat has gained an unenviable public perception as the essential cause of almost all *campylobacter* cases, and there are also many scientific and industry vested interests surrounding this. But, for this concept to have reasonable scientific credibility, all the serious anomalies listed would have to be credibly debased. We need to address some sacred cows before we put all our eggs into one basket if we are going to topple ourselves off the perch of leaders of the OECD for campylobacteriosis.

Acknowledgements

Thanks to the Institute of Environmental Science and Research for campylobacteriosis statistics, and to the Poultry Industry Association of New Zealand for statistics on chicken production, and to Statistics New Zealand for short term visitor and dairy cow numbers.

European country campylobacteriosis rates from data.euro.who.int/CISID/.

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Response to Nelson and Harris' article by Michael Baker and Nick Wilson

Reading the article by Nelson and Harris (1) one could easily come to the conclusion that they are arguing for the affirmative, that chicken meat is indeed the most important source of human *Campylobacter* infection in New Zealand. They repeatedly cite evidence showing the importance of fresh chicken meat as a source of this infection. Nowhere do they refer to a body of evidence that supports an alternative source as being more important in New Zealand.

The main proposition that Nelson and Harris appear to be arguing for is that chicken meat is not the only source of human campylobacteriosis. For example, their first subheading reads "Summary of anomalies to chicken as only source of campylobacter infections" (1). Of course we can only agree with this statement, given the numerous studies cited in both of our papers that describe these other sources. However, this statement was not the one we were meant to be debating so it makes the logic of their article rather bewildering. Nevertheless, we wish to address some of the particularly spurious arguments that are raised by these authors.

Environmental reservoirs vs sources

New Zealand is not short of mammalian and avian reservoirs for *Campylobacter* infection. But for such reservoirs to be important, they need plausible pathways that link them to regular human ingestion of infectious organisms, hence the need to focus on sources of infection. As we discussed in our paper, fresh chicken meat provides a well-documented pathway for such transmission in the New Zealand setting (2). It is heavily contaminated by chicken faeces during slaughter and processing. The organism survives well at refrigeration temperatures. Fresh chicken meat has been found to be heavily contaminated at the point of sale, far more than any other meat. The volume of sales has risen to the point where it is now the most commonly eaten meat and so consumers have many opportunities to be exposed to it. Preventing cross-contamination in kitchen settings is very difficult. Nelson and Harris have not presented evidence-based arguments that challenge the plausibility and important of this source and pathway.

Seasonality

Campylobacter infection has a seasonal component and the mechanisms for this seasonality have received considerable scrutiny (3). These mechanisms are likely to be driven by either seasonal variations in human behaviour linked to exposure, or seasonal variation in the prevalence of *Campylobacter* in reservoirs and sources (3). Barbecuing chicken, which is known to be a risk factor (4), is more common in summer months. Studies in other temperate countries have found both chicken flocks (5) and fresh chicken meat for sale (6) to be more heavily contaminated in summer. The fact that consumption of chicken itself is not especially seasonal is therefore not particularly relevant.

Sporadic vs. outbreaks

The observation that most campylobacteriosis cases occur 'sporadically' rather than as recognised outbreaks is also entirely consistent with the importance of chicken meat as a major source. *Campylobacter* infection requires only a small infectious dose. Many New Zealanders are likely to be regularly exposed to foods and surfaces that have been cross contaminated from chicken meat, both in their homes and in commercial food outlets. This combination of circumstances creates the conditions for large numbers of sporadic cases. To quantify the sources of sporadic disease usually requires an analytical epidemiological investigation such as a case-control study. As we have noted, such studies carried out in New Zealand point to chicken as the dominant source of infection (4,7).

Effects of host immunity

The observed patterns of *Campylobacter* infection are a product of both exposure and the human immune response. No single exposure, no matter how dominant, can explain the full pattern of disease distribution that we observe. Nelson and Harris appear to imply that the higher rates of disease in young children, for example, count against chicken as an important source whereas this distribution is almost certainly influenced by patterns of immunity as well as exposure. In developing countries where the levels of environmental exposure are very high, almost all cases are in young children and it is hypothesised that this results in some level of immunity that extends into adulthood (8).

Conclusion

The 'debate' about the sources of New Zealand's campylobacteriosis epidemic is not simply an academic matter. If we fail to act on the important sources then the human health and economic consequences will remain considerable (2). Consequently, we are alarmed that these authors appear stuck in the hypothesis-generating stage and continue focusing on a succession of minor sources, such as overseas visitors, without a strong evidence base for their assertions. For example, they are incorrect in stating that New Zealand's notification system does not record whether a case was travel associated (it is on the standard case report form). In 2006, only 6% of cases who were asked about travel reported that they had been overseas during their incubation period (9). The caption to their Figure 3, which talks about the "uncanny correlation between New Zealand campylobacteriosis cases and tourist arrivals..." is nothing more than 'pseudoscience'.

Fortunately, agencies such as the New Zealand Food Safety Authority have accepted that contaminated chicken meat is the main source of New Zealand's campylobacteriosis epidemic (10). There are even encouraging signs that the Poultry Industry Association takes the same view, based on their recent press releases.

Even so, we also support other approaches to controlling this disease including: improving drinking water quality; education about the risks of untreated surface water and unpasteurised milk; promoting good hand washing after touching animals, contaminated foods, and other potential sources; travel health advice; and general efforts to improve the microbial safety of all foods through 'paddock-to-plate' food safety programmes. However, none of these efforts is unlikely to have much impact compared with taking the essential steps to manage the dominant source of infection in New Zealand (11).

We also advocate more research on New Zealand's serious campylobacteriosis epidemic. Given its estimated cost, there is an economic justification for investing tens of millions of dollars a year in researching and controlling this hazard. But to get the best value for money, let's make this research highly focussed on dealing with the dominant source. This country is well positioned to carry out a controlled intervention trial of the impact of reducing contamination of chicken meat eg, by freezing and/or chemical treatments. Discussing how to design and run such a trial – now that would be a debate worth having!

Competing interests

There was no external funding for this work. One of the authors (MB) has provided technical advice to the NZFSA and the other (NW) has had two previous research contracts with the NZFSA in 2005.

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Chicken meat is clearly the most important source of human Campylobacter infection in New Zealand

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Abstract

Chicken meat is clearly the most important source of human Campylobacter infection in New Zealand. This source explains most cases and also provides the best opportunity for controlling our severe epidemic. The dominant importance of chicken meat as a source of human infection is supported by epidemiological, laboratory, intervention, and ecological evidence. The New Zealand Food Safety Authority has also identified poultry as the main source. The serious scientific debate has now shifted to the more productive task of finding the most effective interventions to protect the New Zealand population from this source of infection. Given that New Zealand's campylobacteriosis epidemic reached a new peak in 2006, regulatory authorities need to act now to control this epidemic. Highly contaminated fresh poultry needs to be removed from the human food chain.

Key words:

N Z J Med Lab Sci 2007; 61 (2): 44-7.

Introduction

We were somewhat reluctant to participate in this exchange of invited papers on the role of chicken as a source of human *Campylobacter* infection. Food safety scientists in New Zealand have now largely accepted the central importance of chicken meat as the main source of human infection (1). The important debate has now moved on to discussing the most effective ways of controlling this source, so in many ways this present topic is going over old ground (2).

Nevertheless, we have an easy task in presenting the evidence to demonstrate that *"Chicken meat is the most important source of human Campylobacter infection in New Zealand"*. This proposition is well supported by the available evidence. The New Zealand Food Safety Authority has concluded that *"...poultry accounts for just over half of the identifiable infections"* (1). To argue successfully against this proposition also requires identifying a single alternative source that is more important than fresh poultry.

We are taking this statement at 'face value' rather than dissecting it for semantic 'loop holes'. The only term that we think warrants some further discussion is the word 'important'. From a public health perspective, a source of infection could be important for two reasons: either it is responsible for a large proportion of disease burden, or it is particularly amenable to interventions. We would argue that chicken meat is important for both of these reasons.

In this paper we briefly summarise the overwhelming evidence that supports our proposition, much of which we have presented before (2). This evidence is considered under the headings of epidemiological, laboratory, intervention and modelling, and ecological and biological. We then conclude by re-stating the importance of action to control this source.

Evidence about the sources of human campylobacteriosis in New Zealand

Epidemiological evidence

Much of the field of epidemiology is devoted to the goal of investigating causality and of assessing the relative contributions of different sources, exposures and risk factors to the observed disease burden (3). A measure commonly used to describe the contribution of difference sources or risk factors is attributable risk or population attributable risk (PAR) (4). PAR provides an estimate of the burden of disease that would be removed if that particular risk factor or exposure were eliminated. For example, in some populations of working age men the PAR of lung cancer from smoking is estimated to be 73% to 83% (5). PAR is usual estimated using epidemiological studies such as case-control or cohort studies. Ideally, such research is followed by intervention studies, preferably randomised controlled trials, that measure the impact of interventions that reduce or eliminate the risk factor or exposure of concern.

Two separate New Zealand case-control studies of sporadic campylobacteriosis have specifically implicated chicken as the dominant source (6,7). One of these was a large multi-centre study that found that chicken-related exposures could explain over half of the PAR, more than all of the other risk factors combined (7). Other New Zealand epidemiological studies of sporadic campylobacteriosis and of outbreaks, often with supportive laboratory evidence (8-11), are also consistent with an important role for chicken as a risk factor (12).

Case-control studies of sporadic campylobacteriosis conducted in several other developed countries have also identified consumption of poultry at home or in restaurants as an important risk factor (13-18). However, we cannot necessarily apply the PAR results from overseas studies to New Zealand. No overseas countries have disease rates that are as high as ours. As other developed countries have reduced local transmission sources, they have seen imported disease become relatively more important. This source now account for half of new cases in some countries (19). Case-control studies have limitations and may underestimate the contribution of cross-contamination, particularly for foods prepared outside the home where the consumer cannot know how they were handled (20).

Case-control studies inevitably identify a range of risk factors other than chicken. These exposures include contaminated food, water, environments and contact with infected humans or animals. The large multi-centre case-control study of campylobacteriosis carried out in New Zealand in 1994-95 identified significant associations between

disease risk and consuming other raw or undercooked meat or fish, unpasteurised milk, puppy ownership, having a rainwater source for home water, handling of calf faeces, and having sewerage problems at home. However, none of these sources on their own accounted for more than a few percent of the PAR (7).

Descriptive studies of the distribution of campylobacteriosis in New Zealand are consistent with a largely foodborne disease. Notification and hospitalisation rates are higher in cities than in rural areas, which counts against contact with contaminated environments, animals, or water as dominant sources (21). For those living in rural areas, non-poultry sources of infection (such as direct zoonotic infection from farm animals) are likely to be relatively more important than for those living in cities (22). However, the rural population accounts for only 14% of the total New Zealand population (21), and many of them will still consume and be infected from commercially produced chicken in the same way as those living in urban areas.

Laboratory evidence

Laboratory testing of fresh chicken shows that it is heavily contaminated with *Campylobacter*. Surveys of fresh chicken in New Zealand show that contamination is routine and appears to be at high levels (89% of 250 chicken meat samples collected in a national retail survey in 2003–4 were contaminated, a much higher proportion than was seen for other meats) (23,24). Laboratory data, when combined with epidemiological data as detailed above (8-11), support an important role for chicken as a risk factor in this country. Further typing studies are currently underway and these are likely to provide additional information on the similarities between organisms infecting humans and those isolated from a range of animal and environmental sources.

Intervention and modelling evidence

Ideally, New Zealand should now move to interventions which reduce *Campylobacter* contamination levels in poultry meat (2). Not only would such interventions reduced disease in the population, if carefully evaluated they could provide additional evidence to more precisely quantify the importance of fresh chicken meat as a source of human *Campylobacter* infection.

There is some comparable evidence from 'natural experiments' overseas that supports the important role of chicken as a source of human *Campylobacter* infection. In Belgium, poultry was removed from the market for four weeks in 1999 because of a scare over dioxin contamination. The incidence of *Campylobacter* infection in that population dropped by 40% from the expected rate, and then returned to 'normal' when poultry was reintroduced (25).

In 2000, Iceland introduced an intervention that included testing chicken flocks and only allowing those that were '*Campylobacter*-free' to be sold as fresh chicken. The remaining contaminated chicken could only be sold frozen. This intervention was followed by a substantial decline in reported campylobacteriosis (26,27).

The effects of interventions can also be assessed in a more theoretical manner by modelling. Quantitative risk assessment in Denmark suggests that a drop of about 100-fold in *Campylobacter* contamination levels of fresh chicken is enough to reduce the risk to consumers by about 30-fold (28). Freezing chicken, for example, has been found to markedly reduce levels of the organism by 0.5 to >2.5 logs, or approximately a 3 to >300 fold reduction in contamination levels depending on the methods used (27-32) so could be expected to result in a marked reduction in disease rates if introduced in New Zealand. Preliminary results from similar modelling carried out here shows that freezing poultry would considerably reduce the predicted number of human infections (33).

Ecological and biological evidence

The importance of fresh poultry as a source of human infection fits very well with what we know about the ecology of this organism in chicken populations and on fresh chicken meat, and with the changes in food production in New Zealand over the past 25 years.

Large scale production processes mean that poultry provides a good ecological niche for *Campylobacter* (34). This organism is part of the normal gut fauna of chickens, whether reared in sheds or raised organically (35). Highly mechanised slaughter and processing distributes infected gut contents onto the bird carcases, including the traumatised skin. Subsequent decontamination is difficult (36). Bulk distribution and re-packaging provide further opportunities for cross-contamination of other food products. Viable organisms persist at refrigeration temperatures (30,32). Retail chicken meat sold in New Zealand is heavily contaminated (23,24). Kitchen contamination studies show that preparation of meals with raw chicken results in cross contamination to hands, plates, chopping boards, utensils, and ready to eat food (37). This organism can be recovered from kitchen surfaces 24 hours later and is difficult to eliminate with simple cleaning with hot water and detergent (38).

The rise in campylobacteriosis in New Zealand over the last 25 years has coincided with a substantial increase in fresh chicken consumption, which rose from 4 kg/person in 1981 to 30 kg/person in 2005 (Figure 2). Campylobacteriosis incidence is highly correlated with this increase in fresh chicken meat production (Spearman's rank correlation coefficient for disease rates $= 0.96$, $p < 0.0001$). These data provide supportive evidence (in the context of the other evidence detailed above) for explaining the huge increase in campylobacteriosis in New Zealand over this period.

Remaining areas of uncertainty

There are still gaps in our knowledge about *Campylobacter* epidemiology and control. This is to be expected given its transmission characteristics. It is ubiquitous in the environment, it only requires a small infectious dose, and patterns of human immunity are poorly understood (34). Investigating and quantifying the sources of human disease has several methodological challenges (20). Despite these challenges, the scientific evidence that fresh poultry is the dominant source for the human population in New Zealand is overwhelming. This relationship is quite sufficient to explain the observed epidemiological patterns of disease that are seen, particularly the steady rise in incidence over the past two decades.

Conclusions

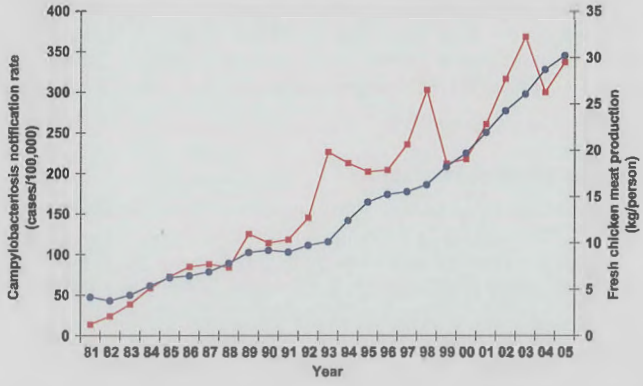
We were somewhat reluctant to participate in this exchange of invited papers on the role of chicken meat as a source of human *Campylobacter* infection. Our concern was that by participating we might imply that there is serious scientific debate about the importance of fresh chicken meat as the main source of New Zealand's campylobacteriosis epidemic. Nevertheless, the public health importance of this issue requires that the evidence-base be regularly reiterated to a wide range of audiences.

New Zealand's epidemic of campylobacteriosis reached a new peak in 2006 with 15,873 notifications and 969 hospitalisations, the highest totals ever reported in this country (39) (see Figure 2). This epidemic is resulting in preventable deaths, long-term disabilities and substantial morbidity as well as huge costs to our economy and reputation as a producer of safe food. Just as it would be irresponsible to delay action on climate change until all scientific doubt about the precise details were resolved, we think it would be irresponsible to delay action on contaminated chicken meat until there is total agreement on the precise size of its contribution.

Heavily contaminated chicken meat urgently needs to be removed from the human food chain in New Zealand. Switching to frozen poultry could provide a means to continue supplying this popular food in a safer form. Freezing could be combined with 'scheduled' processing where chicken meat from *Campylobacter*-free flocks, or those that have been shown to have low contamination levels, could possibly still be sold fresh. Meat from infected flocks could be treated by freezing or other methods to produce comparable reduction in contamination levels (eg, there are chemical decontaminants such as acidified sodium chlorite that show promise (40) but these agents need to demonstrate effectiveness and safety for large scale use). This is the approach favoured by some European food safety groups (41).

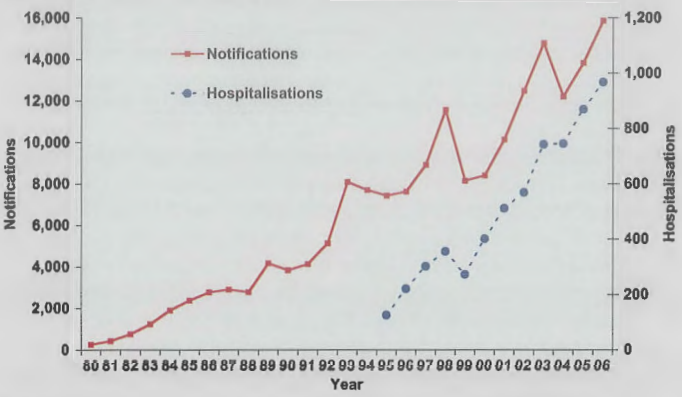
Our reason for participating in this discussion is to use the opportunity to urge swift decisive action by the New Zealand Food Safety Authority to control this foodborne epidemic. We also hope that the Poultry Industry Association of New Zealand (PIANZ) will support substantive action on this issue. PIANZ has previously shown itself very willing to seize on potential alternative theories about the sources of New Zealand's campylobacteriosis epidemic. We would be disappointed if they used this discussion in a similar cynical manner and thereby helped to perpetuate New Zealand's campylobacteriosis epidemic.

Figure 1. Campylobacteriosis rate (cases / 100 000) and annual production of fresh chicken meat (kg/person) in New Zealand, 1981–2005



Source: Statistics New Zealand quarterly survey of poultry producers. Note that because almost all NZ chicken production is destined for human consumption within NZ, and virtually no chicken is imported, these data correspond well to human consumption patterns (10).

Figure 2. Annual number of notifications (1980–2006) and hospitalisations (1995–2006) for campylobacteriosis in New Zealand



Source: Institute of Environmental Science and Research Limited (notifications) and New Zealand Health Information Service (hospitalisations, based on principal diagnosis).

Competing interests: There was no external funding for this work. One of the authors (MB) has provided technical advice to the NZFSA and the other (NW) has had two previous research contracts with the NZFSA in 2005.

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Response to Baker & Wilson's article by Warrick Nelson and Ben Harris

But where is the actual evidence?

The paucity of actual data presented for the chicken as prime source scenario simply does not prove that chicken, contaminated as it may be, must be the most important source for human illness. Studies of case histories, including the significant questionnaire bias of the helpful 'have you eaten any chicken' prompts help negate their objectivity. Chicken is a commonly eaten food by most NZ residents anyway. Also ignored under the 'ecological and biological evidence' heading is the much, much larger than poultry reservoir, campylobacter reservoir in other animals (cattle, sheep, pets, etc) which may well have a multiple vector spread to humans – eg water, flies, sparrows – which better explains annual seasonality cycles, large North Island vs South Island differences, variable dairy cow shedding rates and 5% increases in infection rates up to 14 degrees Celsius (noting campylobacter does not multiply below 30 degrees).

Simply stating and restating a firmly held belief does not change this belief into a fact. Risk assessments and case-control studies are important tools, but do not provide evidential proof, especially where there is significant and credible competing evidence. It has been very tempting to take the simple route that chicken meat is frequently contaminated with campylobacter, humans eat chicken meat, ergo chicken is the source. New Zealand's very high incidence of campylobacteriosis requires far more than simply referring to risk assessments and finger pointing at a single industry. New Zealanders deserve a higher standard of investigation for such a significant disease.

The two seemingly prime evidence case control studies quoted 'implicated' chicken as the dominant source and 'could explain' poultry as source and later acknowledge 'case control studies have limitations' – we agree.

In response to NZFSA 'poultry accounts for just over half the identifiable infection' – but in well over 90% of cases a source is never identified. The NZFSA, liberally quoted, clearly have their doubts. They, plus the Ministry of the Environment, have just secured a $735,000 grant to look further. Why would they need to if they were sure, or should they return the money? From the NZFSA 11 April 2007 press release, abridged, concerning this grant (our emphasis and bracket comment added):

NZFSA Risk Analyst Peter van der Logt and Sue Powell, MfE General Manager says: The work will help us better understand the links between human health and the environment. For example, we will look at how Campylobacter moves from animal waste on farms into human food and water supplies and how best to prevent this.

Poultry is recognised as a likely primary pathway and is believed (evidence?!) to be responsible for about half New Zealand's reported cases of campylobacteriosis. Animal contact and person-to-person transmission also play a significant role.

NZFSA's Science Manager, Andrew Pavitt, says: Campylobacteriosis is a very complex disease and uncertainty about the relative contribution of the various transmission pathways has prevented a comprehensive risk management programme from being developed. We hope this project will lead to an improved understanding of the movement of Campylobacter in the environment. This information can be used by farmers and environmental managers to reduce the passage of this bacterium in our waterways says Sue Powell. The project aligns closely with the outcomes sought in the Sustainable Water Programme of Action, and will be directly relevant to the proposed national environmental standard for sources of human drinking water.

Correlation between annual campylobacteriosis rates and chicken consumption, with a lack of correlation on a quarterly basis, neither proves nor disproves chicken as a source. The distinct seasonality in campylobacteriosis rates indicates that at least one other factor is involved, assuming that chicken consumption is directly correlated with risk for campylobacteriosis. Following this assumption led us to suggest a food associated risk with another vector involved (flies were postulated) in our earlier paper (1). We suggested that chicken (and particularly chicken eaten away from home) is frequently fingered as the source of campylobacter infection because of the extensive finger contact during consumption, and that the campylobacter contamination was introduced on the dirty hands of the eater, rather than campylobacter on the raw chicken before cooking.

The 'intervention and modelling evidence' headline wants interventions now, and were detailed, which 'could provide' evidence. Also freezing chicken 'could be expected to result in a marked reduction in disease'. This is not evidence, rather a hope, which future studies may or may not support.

Observing that chicken, contrary to almost overwhelming opinion, is not the most important source for campylobacter in humans does not imply, nor require, an alternate single source. We are simply concerned that research in this field has been hijacked onto a one-track path with no positive results yet.

We agree, action on poultry contamination is likely warranted, but there are too many serious unexplained anomalies not to research our proven to be heavily contaminated environment as a source. Chicken may eventually prove to be the most important source for human campylobacter cases, but the current evidence simply does not support this supposition. Rather, as better identification techniques are developed, the evidence against chicken is mounting. Extended metaphors may be fun, but this disease is unpleasant, expensive and can have serious consequences. This is no cock and bull story.

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Conflicts of Interest: None, including NZFSA or MOH.

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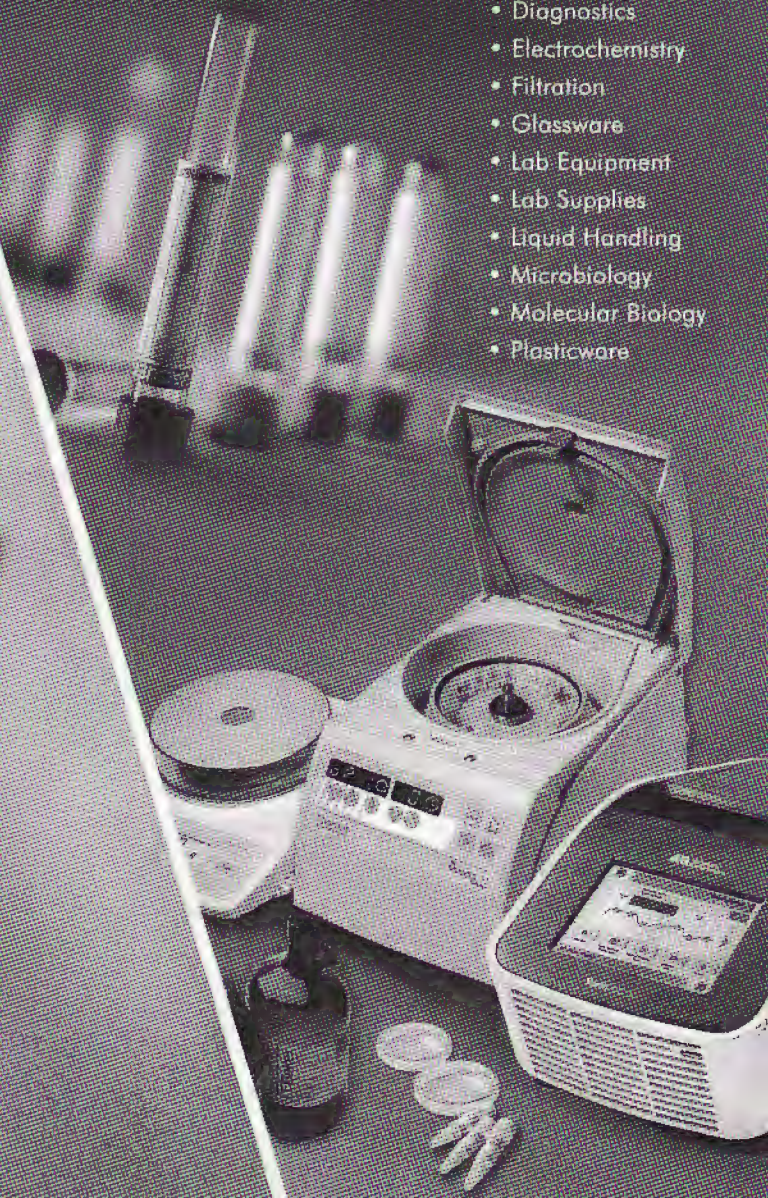

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Journal-based questionnaire

Journal-based questionnaire for the August 2007 issue

Below are 10 questions based on this issue of the Journal. The answers can be found anywhere, thus read the entire Journal.

Answers are to be submitted through the NZIMLS web site only (www.nzimls.org.nz). Make sure you supply an email address and that it is correct. The site will remain open until 5pm on Friday 19 October. You must get at least **8** questions right to earn 5 CPD points. Note the revised number of correct answers required, it is now at least 8, it used to be at least 7.

1. The screening programme for prostate cancer for South Indian males emphasizes the importance of which three approaches.
2. How was the serum PSA measured in the first case study.
3. What is the protocol in Medlab Hamilton if a HDL cholesterol of <0.5 mmol/L is found.
4. PSAs considered as an ideal tumor marker for.....
5. What is Medlab Hamilton's criterion for protein electrophoresis.
6. In the Diagnostics footprints article what were the bone marrow findings and what diagnosis was made.
7. What is Warrick Nelson and Ben Harris' view on the likely source and vector for Campylobacter in New Zealand.
8. What is the main Campylobacter species found in campylobacteriosis cases and at what percentage. What other organism has been implicated.
9. By how much did the incidence of Campylobacter infection in the Belgian population drop by from the expected rate when poultry was removed from the market in 1999.
10. The Campylobacter organism is part of the normal gut fauna only in chickens reared in sheds. False or True.

Questions and answers for the April 2007 journal-based questionnaire

1. What is the new prize worth for the best case study published in the Journal during the whole year
   $200
2. How many years has the Journal been continuously published as of the end of 2006
   60 years
3. Name two of the three ways that Fellowship of the Institute can be obtained
   By examination in 2 parts: 2 written exams and a dissertation
   By thesis: in the style of a Master of Science by thesis requirements
   By publications: min. 7 peer-reviewed, at least 4 as 1st author
4. Candidates applying for Fellowship by examination may be exempted the Part 1 examination if they are holders of an approved post graduate qualification. True or false
   True
5. What is still the gold standard for malaria diagnosis
   Direct microscopic observation of blood films
6. What is more sensitive for malaria diagnosis, the thin film or thick film, and why
   Thick film
   Larger volume of blood examined (sensitivity 40 parasites/µl or 1 parasite/200 WBC)
7. Parasites are always found in peripheral blood smears from patients with malaria. True or false
   False, but malaria pigments maybe seen in circulating phagocytic leucocytes
8. What method allows the detection of drug resistant parasites and mixed infections
   PCR using single nucleotide polymorphism (SNP) analysis
9. What are the two advantages of rapid methods for the diagnosis of malaria
   They are quick to perform
   They have high sensitivity
10. What are the three disadvantages of rapid methods for the diagnosis of malaria
    Their relatively high cost
    Inability of some tests to distinguish malaria species
    Manufacturing variation

British Journal of Biomedical Science Abstracts

Creagh S. Lucey B. Interpretive criteria for mupirocin susceptibility testing of Staphylococcus spp. using CLSI guidelines. *Br J Biomed Sci* 2007; 64: 1-5.

Mupirocin is an antimicrobial agent commonly used to treat staphylococcal infection or to eliminate persistent carriage. To date, interpretive criteria have not been established to define susceptibility or resistance when performing mupirocin susceptibility testing. In this evaluation, using CLSI guidelines, a total of 502 staphylococci comprising 219 methicillin-sensitive *Staphylococcus aureus*, 222 methicillin-resistant *S. aureus* and 61 coagulase-negative staphylococci are tested by broth microdilution, disc diffusion and E-test. Disc diffusion using 5 microg mupirocin discs was found to be a reliable method to distinguish susceptible and resistant strains. Minimum inhibitory concentration (MIC) determination was required to differentiate low-level and high-level resistance to mupirocin. E-test was found to be an accurate alternative to broth microdilution for the routine determination of MIC values of staphylococci to mupirocin. Broth microdilution and disc-diffusion results were plotted on a scattergram, and error rates were calculated. No errors were found using susceptibility criteria of < 4 microg/mL (MIC) and > 19 mm (zone diameter).

Wishart K, Loughrey A, McClurg RB, Goldsmith CE, Millar BC, Rao J, et al. Lack of horizontal gene transfer of methicillin-resistance genetic determinants from PBP2a-positive, coagulase-negative staphylococci to methicillin-sensitive Staphylococcus aureus using transcutaneous electrical nerve stimulation (TENS). *Br J Biomed Sci* 2007; 64: 6-9.

Previous research shows that approximately half of the coagulase-negative staphylococci (CNS) isolated from patients in the intensive care unit (ICU) at Belfast City Hospital were resistant to methicillin. The presence of this relatively high proportion of methicillin-resistance genetic material gives rise to speculation that these organisms may act as potential reservoirs of methicillin-resistance genetic material to methicillin-sensitive *Staphylococcus aureus* (MSSA). Mechanisms of horizontal gene transfer from PBP2a-positive CNS to MSSA, potentially transforming MSSA to MRSA, aided by electroporation-type activities such as transcutaneous electrical nerve stimulation (TENS), should be considered. Methicillin-resistant CNS (MR-CNS) isolates are collected over a two-month period from a variety of clinical specimen types, particularly wound swabs. The species of all isolates are confirmed, as well as their resistance to oxacillin by standard disc diffusion assays. In addition, MSSA isolates are collected over the same period and confirmed as PBP2a-negative. Electroporation experiments are designed to mimic the time/voltage combinations used commonly in the clinical application of TENS. No transformed MRSA were isolated and all viable *S. aureus* cells remained susceptible to oxacillin and PBP2a-negative. Experiments using MSSA pre-exposed to sublethal concentrations of oxacillin (0.25 microg/mL) showed no evidence of methicillin gene transfer and the generation of an MRSA. The study showed no evidence of horizontal transfer of methicillin resistance genetic material from MR-CNS to MSSA. These data support the belief that TENS and the associated time/voltage combinations used do not increase conjugational transposons or facilitate horizontal gene transfer from MR-CNS to MSSA.

Chaturvedi R, George S, John A. Preventive and protective effects of wild basil in ethanol-induced liver toxicity in rats. *Br J Biomed Sci* 2007; 64: 10-2.

In the present study, preventive and protective effects of *Ocimum gratissimum* in ethanol-induced hepatotoxicity are assessed in albino rats. A methanol extract of *O. gratissimum* leaves is prepared, with a yield of 3.5% (w/w) of the dry weight of leaves. Graded doses of the extract (10, 20, 40 and 80 mg/kg body weight), together with ethanol (5 gm/kg body weight) are administered orally to experimental groups for 30 days. Normal control rats receive distilled water only, while rats in an alcohol control group (AC) receive ethanol only for 30 days. *O. gratissimum* reduced the level of thiobarbituric acid reactive substance in all experimental groups (E1-E4). Alanine transaminase and aspartate transaminase levels fell in all experimental groups (E1-E4), but this reduction was significant only in groups E3 and E4 ($P < 0.05$), indicating inhibition of lipid peroxidation by free radicals generated after ethanol metabolism. Levels of antioxidants also increased. Ascorbic acid and glutathione levels increased in all experimental groups (E1-E4; $P < 0.05$ and $P < 0.01$, respectively). A significant increase in catalase ($P < 0.05$) was noted only in group E4, although an upward trend was noted in all experimental groups. This study shows that *O. gratissimum* prevents free radical damage to the liver and thus protects the organ from oxidative stress.

Larijani B, Shooshtarizadeh P, Mosaffa N, Heshmat R. Polymorphonuclear leucocyte respiratory burst activity correlates with serum zinc level in type 2 diabetic patients with foot ulcers. *Br J Biomed Sci* 2007; 64 :13-7.

Patients with diabetes mellitus (DM) are prone to infection, in part due to phagocyte dysfunction and impaired polymorphonuclear (PMN) leucocyte superoxide generation. Another frequently mentioned factor in the pathogenesis of infection in DM patients is altered zinc status. This study aims to evaluate the association between serum zinc level and PMN respiratory burst activity in patients with type 2 DM. Thirty-nine type 2 DM patients (19 with foot ulcers) and 20 healthy controls are studied. Respiratory burst activity is evaluated at baseline and in stimulated states by a nitro blue tetrazolium (NBT) reduction test. Serum zinc level is evaluated by atomic absorption spectrophotometry. Although not statistically significant, PMNs from diabetics with foot ulcers appeared to be slightly hyperactivated at the baseline state. The NBT index was significantly lower in DM patients with foot ulcers after stimulation. Mean serum zinc level was significantly lower in diabetics with foot ulcers compared to those without foot ulcers. A significant negative correlation between serum zinc level and NBT index at baseline was seen in patients with foot ulcers, but this changed to a significant positive correlation after stimulation. These findings may be explained by PMN hyperactivity at baseline and by respiratory burst dysfunction following stimulation in diabetic patients.

Keegan H, Ryan F, Malkin A, Griffin M, Lambkin H. Human papillomavirus prevalence and genotypes in an opportunistically screened Irish female population. *Br J Biomed Sci* 2007; 64: 18-22.

This study aims to evaluate human papillomavirus (HPV) prevalence and predominating genotypes in liquid-based cervical cytology samples from an Irish urban female population. In addition to use of routine cervical cytology testing, women are screened for HPV using the MY09/11 primers for the HPV L1 gene and primers for beta-globin amplification in a multiplex format. Overall, 996 women between the ages of 16 and 72 years (average age: 35) are included in the study and HPV prevalence was 19.8%. Cytology results showed that 88.9% were normal, 9% borderline or mild dyskaryosis, 1.1% moderate dyskaryosis and 0.9% severe dyskaryosis. Human papillomavirus prevalence in women under 25 was 31%, reducing to 23% in women in the 25-35 age group and to 11% in women over 35. Human papillomavirus prevalence increased with grade of cytology from 11.4% (normal) through 85.4% (borderline), 84% (mild), 100% (moderate) to 100% (severe dyskaryosis). HPV 16 (20%) and 18 (12%) were the most common high-risk types detected

in the study. Other common high-risk types were (in descending order) HPV 66, 33, 53, 31 and 58. HPV 66 was associated with the detection of borderline abnormalities by cytology. This is the first population-based study of HPV prevalence in the normal healthy cervical screening population in the Republic of Ireland.

Sharma A, Rajappa M, Saxena A, Sharma M. Antioxidant status in advanced cervical cancer patients undergoing neoadjuvant chemoradiation. *Br J Biomed Sci* 2007; 64: 23-7.

Cervical cancer is the most common cancer in Indian women. The aim of this study is to assess the alterations in the circulating lipid peroxide, antioxidant components and activities of defence enzymes in advanced cervical cancer patients, and to monitor the variations in their levels before and after neoadjuvant chemoradiation. Sixty patients with advanced cancer of the cervix (FIGO IIIa-IVb) are included in the study, along with 60 healthy controls. Blood samples are collected before the start of therapy (S1), two weeks after the second course of chemotherapy (S2) and two weeks after completion of tele/brachyradiation (S3). Single blood samples are taken from controls. Lipid peroxides, conjugated dienes, reduced glutathione (GSH), catalase (CAT) and glutathione-S-transferase (GST) are estimated using standard methods. Glutathione peroxidase (GPx) and superoxide dismutase (SOD) are assayed using commercially available kits. The pretreatment levels of plasma lipid peroxide were significantly elevated in cancer patients, while significantly lowered levels of GSH, GPx, GST, SOD and CAT were observed when compared to controls. After chemotherapy, the levels of lipid peroxidation showed a significant decline ($P < 0.05$), which became highly significant after chemoradiation ($P < 0.01$). Levels of GSH, GPx, SOD, GST and CAT showed a mild increase after chemotherapy. After chemoradiation, levels reverted to normal or near normal ($P < 0.01$). Low levels of antioxidants in the circulation of patients with cervical cancer may be due to their increased utilisation to scavenge lipid peroxidation as well as their sequestration by tumour cells. The observed increase in antioxidant concentration after therapy might be due to the death of tumour cells or the arrest of tumour growth by chemotherapeutic agents. The normalisation of these parameters may provide information about the efficacy of neoadjuvant chemoradiation. A larger patient cohort with a longer follow-up period for therapeutic response studies may yield more significant data.

Rughooputh S, Kachaliya S, Jetly D, Greenwell P. Cervical cancer and human papillomavirus among slum dwellers in India. *Br J Biomed Sci* 2007; 64: 28-31.

Almost half a million new cases of cervical cancer are diagnosed each year worldwide. Human papillomavirus is recognised as one of the leading causes and is associated with 90% of cases. However, other risk factors (e.g., age of first sexual contact, number of sexual partners, multiparity, diet, genetic predisposition and environment) are also associated with cervical cancer. The present retrospective study is performed on a cohort of women from the slums of a major Indian city. The patients are aged between 38 and 68 years (mean: 49.3 years) and are multiparous (mean number of children: 3.4). In this group, 61% have a history of miscarriages. Histological sections from cone biopsy are tested for the presence of high-grade human papillomavirus (HPV) using GP5+/GP6+ and MY09/MY11 primers and a set of beta3-globin primers. Only 33% of the cancer patients studied were positive for high-grade HPV DNA, suggesting that predisposition to cervical cancer in this cohort is not highly associated with HPV, and that other risk factors may increase the risk of cervical cancer.

Battle R, Clark B. Quantitative analysis of human leucocyte antigen expression during culture of Epstein-Barr virus-transformed cell lines using the dako QIFIKIT. *Br J Biomed Sci* 2007; 64: 32-4.

Pre-existing donor-specific human leucocyte antigen (HLA) antibodies in renal allograft recipients result in hyperacute and accelerated graft failure. These antibodies can be detected in flow cytometric assay systems using HLA-characterised Epstein-Barr virus (EBV)-transformed B-lymphocyte cell lines. Confident assay performance is predicated by the expression of HLAs on the EBV-transformed B-cell line surface. Surface HLA expression of three EBV-transformed B-cell lines that had previously been used as part of a potential organ recipient serum screening panel at St James' University Hospital, Leeds, are assessed for changes in the level of HLA expression over the nominal culture duration of eight days using the QIFIKIT (Dako, Denmark), a quantitative flow cytometry kit for assessing cell surface antigens. A comparison of the mean fluorescence intensity (MFI) of the known antigen levels of the beads via a calibration graph permits determination of the antibody binding capacity of the cell lines. Results showed that HLA expression is not consistent throughout the cell culture, with optimal expression occurring during day 2 of culture. Inconsistent HLA expression demonstrated during the cell culture means that no assumption of the level of HLA expression can be made, and that cell lines used as part of a screening panel should have their HLA expression in cell culture determined.

Nwose EU, Jelinek HF, Richards RS, Kerr PG. Erythrocyte oxidative stress in clinical management of diabetes and its cardiovascular complications. *Br J Biomed Sci* 2007; 64: 35-43.

Diabetes mellitus is a chronic disease in its own right and is also regarded as a cardiovascular risk factor as well as a cardiovascular disease, due to its ability to progress to a stage of cardiovascular co-morbidity. The pathophysiology of cardiovascular complications in diabetes is reported to involve hyperglycaemia-induced oxidative stress. The erythrocyte has an array of endogenous antioxidants involved in quenching oxidant production and the exponential chain reactions in diabetes. When the erythrocyte is oxidatively stressed, as demonstrated by depleted reduced glutathione and/or increased malondialdehyde in its cell membrane, the risk of diabetes progression and its cardiovascular sequelae, including atherosclerosis and coronary artery disease, is increased. Virtually all studies that determined erythrocyte malondialdehyde and glutathione in diabetes show consistently increased and reduced levels, respectively. Furthermore, cardiovascular complications of diabetes are reported to commence at the prediabetes stage. Current coronary artery disease screening programmes based on the presence of two or more risk factors are failing to identify those with increased risk of diabetes and cardiovascular complications, thereby limiting early interventions. Screening that includes erythrocyte oxidative stress determination may provide an additional marker for both preclinical and advanced disease. In this review, a concise description of the involvement of erythrocyte oxidative stress in diabetes mellitus and its cardiovascular sequelae is presented. Antioxidant action and interaction in the erythrocyte are also described, with emphasis on why current coronary artery disease screening markers cannot be regarded as erythrocyte oxidative stress markers.

Bocci V. The case for oxygen-ozone therapy. *Br J Biomed Sci* 2007; 64: 44-9.

Ozone is a very reactive gas that is toxic to the respiratory system. However, under controlled conditions, it can be therapeutically useful in several human diseases. An unfavourable combination of factors (ozone is one of the worst troposphere pollutants) and past misuse have led to misgivings about ozone therapy. However, basic and clinical work developed over the past 10 years has clarified the fundamental mechanisms of action of ozone in biology and medicine. Interestingly, judicious doses of ozone dissolved in blood trigger a cascade of well-defined chemical compounds acting on multiple cellular targets according to well-known molecular, biochemical and pharmacological pathways. Ozone therapy is proving to be very useful in age-related macular degeneration, ischaemic and infectious diseases, and in wound healing disorders, where conventional medicine has failed. Critical evaluation of the potential therapeutic utility of this simple, inexpensive medical application by national and international health authorities is warranted and may lead to clinical benefit for a large proportion of the world's population.

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News from the Universities Schools of Medical Laboratory Science

Auckland University of Technology

The AUT Division of Applied Sciences Awards Ceremony was held on Friday 11th May 2007. The Ceremony celebrates the success of the top graduating students within the Division. It was very well supported by professional bodies and industry companies. This year, the prizes were as follows:

NZIMLS Prize for the most outstanding Bachelor of Medical Laboratory Science student: **Helen Kazey**. A photo of Maree Gillies presenting the prize to Helen Kazey on behalf of the NZIMLS is shown on the next page. Helen came to New Zealand in 1999 and came to AUT to complete her BMLS. She graduated with very high marks in all subjects, and is also the winner of the top student in Haematology prize. She is now working at LabPlus in Haematology, training through all the sections. Helen finds Haematology work fascinating and challenging, particularly the morphology and its aid to patient diagnosis. She aspires to be a section leader in the future and likes to keep up to date reading journal articles. Helen's interests include reading and travel.

AACB Prize for the student with outstanding performance in Clinical Chemistry: **Jinny Ng.**

Roche Diagnostics New Zealand Ltd Prize for the most outstanding student in Immunology: **Bridget McMonagle**.

Sysmex Prize for the most outstanding student in Medical Microbiology: **Rebecca Reagan.**

Diamed Prize for the most outstanding student in Transfusion Science: **Keith Qassab**.

Beckman Coulter Prize for the most outstanding student in Haematology: **Helen Kazey.**

Diagnostic MedLab Prize for the most outstanding student in Cytology: **Laura-Jane Robertson.**

Medica Pacifica Prize for the most outstanding student in Histology: **Laura-Jane Robertson**.

Holly Perry
Programme Leader, BMLS
AUT

Otago University

I would like to thank Rob Siebers, Editor and the NZIMLS Council for creating a section in the Journal for the Universities to communicate with the wider membership of the NZIMLS. I would hope that this opportunity will provide a two way communication with the diagnostic pathology laboratories and will improve ways to communicate and respond to comments, enquiries and issues arising especially those relating to how our Medical Laboratory Science programme operates.

This year at Otago saw the new First Year Health Science (FYHS) Programme commence. A full academic year which all health science students take prior to applying for entry in to the Health Professional Schools. With the first semester over and the new second semester about to commence the programme has worked well ensuring that all health science students have a good grounding in the biological sciences including Physics and Chemistry as applied to biomedicine. The development of this new programme has created opportunities to review second year papers and has created more space in the second year medical laboratory science papers. This has allowed the development of areas such as professional practice and development, cultural and ethical issues in diagnostic pathology as well as new diagnostic pathology material.

This year also saw the introduction of a new Fourth Year paper - Diagnostic Molecular Pathology which replaced the Cytogenetics paper, and the appointment of a new lecturer, Dr Alison Fitches who is responsible for the paper. Whilst retaining the key elements of Cytogenetics the paper also covers the use of molecular technologies in disease diagnosis across pathology disciplines and has proven to be very popular.

A burgeoning issue for our programme at present is the placement of the Fourth Year students. A requirement for the degree and for subsequent registration is that the Fourth Year students need to complete two semesters in two different disciplines. In the past this has worked very well, however recently there has been increasing difficulty in finding diagnostic pathology laboratories willing to take students for placements. Whilst there are a number of external issues currently in diagnostic pathology, such as re-organization, changes in providers, workforce issues etc, the profession will need these graduates in the future. With an international shortage of medical laboratory scientists of about 34 percent, the lack of suitable training for our own graduates will become a significant issue for diagnostic pathology in the future.

Finally, as August draws near I will look forward to meeting colleagues at the South Pacific Congress in Auckland. The programme is looking good and will provide some interesting discussions on the science as well as the other issues the profession is facing.

Mike Legge
Director, Medical Laboratory Science Programme
University of Otago

Helen Kazey (on right) receiving the NZIMLS prize from Maree Gillies (on left).

Des Philip (left) receiving the IFBLS Past Presidents' Award from Noel White (right).

The IFBLS Past Presidents' Award

The IFBLS Past Presidents' Award is part of the IFBLS award programme at the IFBLS biennial World Congress. The award is in the gift of the three immediate Past Presidents of IFBLS. In 2006 these were Bill Younger (Canada), Martha Hjalmársdóttir (Iceland) and Noel White (Ireland). The award is given to an individual who the Past Presidents believe has made a unique contribution to the development of the profession both at national and international level.

Following correspondence and discussion between the three Past Presidents, the unanimous decision was that the 2006 award should be made to Desmond Philip from New Zealand. When Des' career is looked at it is easy to see why the decision was simple.

Employment

Des' career commenced as a 'bacteriological cadet' with the Auckland Hospital Board in 1946. After training at the Auckland Hospital he moved to Middlemore Hospital in1952, which in those days was still under the same Hospital Board as Auckland Hospital. Des went with two trainees under him, an Intravenous Solutions Department to oversee, no resident Pathologist (a visiting one came as necessary) and a hospital of 300 beds – 210 of which were orthopaedic, 30 medical, 30 surgical and 30 plastic surgery. When he left in 1988, after 42 years service, the hospital was under its own Health Board and the laboratory had grown to a full staff in excess of 150.

New Zealand Institute of Medical Technology

Des' involvement with things political was not long delayed! When he commenced work, the New Zealand Association of Bacteriologists had not been long formed and he was encouraged right from the outset to join. His boss was among the group that founded the Association and was its first Editor. He bought an old printing press and printed and collated the Journal in his basement. Des helped with the laborious one page at a time printing and then collating, packing and posting. Not long after he was at Middlemore he was encouraged to become concerned in the professional Association and was nominated for Treasurer, a post he held from 1959 - 1966. Des went on to serve as Vice President from 1966 - 1972. This was followed by a three year stint as President (1972 – 1975). Des left the Council in 1980 after 21 years as Treasurer, Vice President, President and Past President of the New Zealand Institute of Medical Technology.

New Zealand Medical Laboratory Technologists Registration Board

During that time Des was nominated by the Institute (NZ) to the Advisory Board on Laboratory Services which was responsible for the administration of the examinations leading to qualification. He served in this capacity from 1966 – 1972.

1973 saw Des still on the Board, now as a Ministerial nomination, when it became the Registration Board for Medical Technologists and he remained there until 1991, serving as Chairman from 1976 – 1991, a few years after he retired from laboratory work. All in all, in different guises, Des was a member of that Board for about 25 years.

One situation Des was not exactly comfortable in was when the Health Department nominated him to the State Services Commission as their adviser for the grading of laboratory positions throughout the health services of all NZ hospitals. Des always wanted to advance the technologist's cause but had to be scrupulously fair in comparing various positions – not always seen as such by disenchanted aspirants for upgrading!

IAMLT (now IFBLS)

A new world was opened up to Des by the grant, by Burroughs Wellcome, of their Travel Award for a New Zealand Technologist to travel to the IAMLT World Congress which was held in 1978 in Edinburgh. It was his first trip overseas and was a hugely enjoyable experience. While the Congress was a great success, Des was less than impressed by the organization and running of the General Assembly of Delegates (GAD). Des thought that he could contribute something to this, so he asked the New Zealand Institute to nominate him for Council of IAMLT. Des travelled to Durban in 1980 for the South African Congress. The Delegates elected him as a Councillor and thus commenced his years with IAMLT. One of his first tasks was to undertake a complete overhaul and revision of the Statutes, which at previous GADs had proved, on more than one occasion, to be contentious and difficult to interpret. It proved to be a long and arduous task. However, they came right after a number of years and for Des the whole thing proved worthwhile when a GAD was held without any amendments and not a single reference to the Statutes! Des was re-elected to Council in 1982 and in 1984 was elected Treasurer, which post he held for two years. Des served as Vice President from 1986 – 1988 and as President from 1988 – 1990. This was followed by two periods as Past President.

Another highlight of Des' time with IAMLT was as Editor of the Journal – *MedTec International (MTI)*. When the Association was formed a set of Statutes was drawn up in which it was defined that the Association would produce a Journal, the aims of which were twofold – namely that it would act as a 'newsletter' and that it would provide some sort of "prestige" to the Association. The growing diversity of disciplines within the profession, each with their own means of publishing prestigious scientific articles, meant that it was always difficult to source material suitable for publication.

When Des took over as Editor he tried a different approach and tried to make the publication 'thematic' in areas that were common to all the disciplines – e.g. ethics, management, equipment evaluation, etc. It was successful to a degree but difficult to find a range of suitable subjects. What it did do was provide a vehicle for the dissemination of WHO news.

After Des' time as Past-President the Council altered the constitution of Council to have the Editor as an appointment and not necessarily one of the Council members. He thus continued on Council as Editor of *MTI* from 1994 until he retired from the Editorship in 1997. A quick calculation will show that Des Philip served IAMLT for a total of 17 years –no mean contribution to the furtherance of the profession.

The above address was given by Noel White at the Awards Programme of the Opening Ceremony of the World Congress in Seoul, South Korea in September 2006. In excess of 1,200 delegates were present to hear of Des' achievements.

As Des was unable to be present at the World Congress, Council of IFBLS decided that the presentation of the Past Presidents Award should be made in person to Des in Auckland in March 2007, when Noel White would be in New Zealand on vacation.

At a luncheon, hosted by IFBLS, in Sails Restaurant on March 9th 2007, Noel White, on behalf of the Past Presidents of IFBLS, presented the award to Des. Also present at the lunch were Robin Allen, President NZIMLS, Dennis Reilly, Trish Reilly, Des' son and daughter and Siobhán White.

Department of error

Several errors occurred in the recent **"CPD recertification programme update 2007"** article in the April 2007 issue of the Journal (N Z J Med Lab Sci 2007; 61 (1): 8-11).

- The author of this article is Jillian (not Gillian) Broadbent.
- In the Table on page 10 under section 5. Presentation – oral: CPD points allocation for external presentation > 30 min should be 20, not 2.
- In the Table on page 10 under section 8. Scientific paper publication: CPD points allocation for primary author should be 20, not 10.

The Editors regret the errors.

NZIMLS Journal Prize

Council of the NZIMLS has approved an annual Journal prize for the best case study accepted and published in the Journal during the calendar year. The prize is worth $200.

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

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

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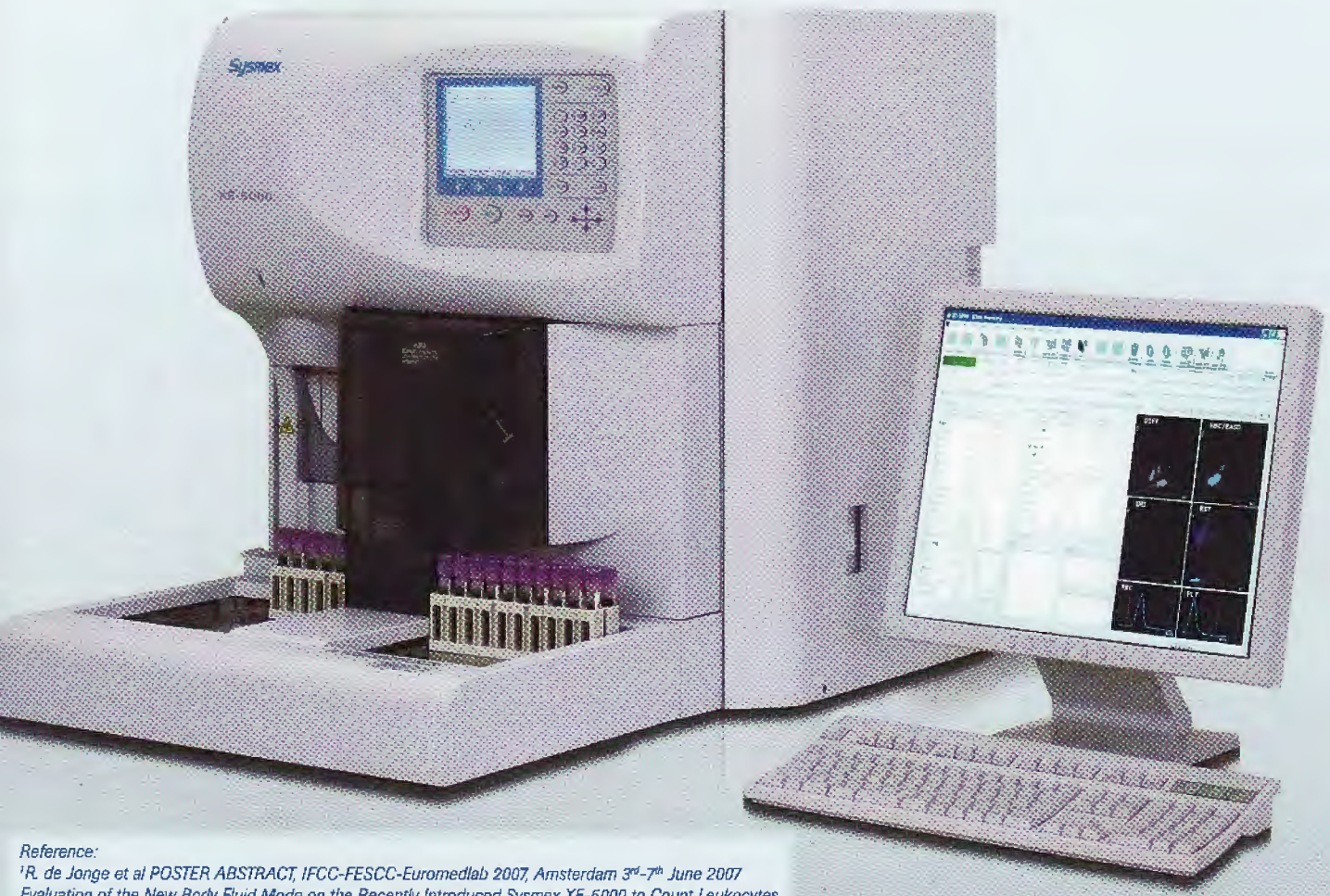

Reference:
[1]R. de Jonge et al POSTER ABSTRACT, IFCC-FESCC-Euromedlab 2007, Amsterdam 3rd-7th June 2007 Evaluation of the New Body Fluid Mode on the Recently Introduced Sysmex XE-5000 to Count Leukocytes and Erythrocytes in Cerebrospinal Fluid and Serous Fluids.

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