Letters to the Editor

Hokianga Hospital (1935 and 1998), Rawene, New Zealand

There is increasing concern regarding the development of antibiotic resistance in many common bacterial pathogens. Well known examples are methicillin resistance in Staphylococcus aureus and penicillin resistance in Streptococcus pneumoniae. Ellis-Pegler challenges us to try and preserve "these amazing agents" for as an long as possible.[1]

What was life as a doctor like prior to the antibiotic era? What will life in the future hold for us if we allow antibiotic resistance to increase? I have taken the opportunity to look at the record of admissions to Hokianga Hospital in Rawene, New Zealand for three month period in 1935. In antibiotic terms this was the year Domagk's work with prontosil was published, Flemming's penicillin mould was still awaiting Howard Florey's hand of genius, while Sulphonamides become available in 1936.

Hokianga Hospital is an isolated country hospital serving a rural population, a high percentage of whom are Maori. The period examined was from on 1st July 1935 through to 30th September 1935. This is winter time in Hokianga, and is usually a time of frequent heavy rain, the roads would have turned to mud and slush and travel by boat on the harbour was the preferred transport.

The Surgeon Superintendent of Hokianga at the time was the legendary George McCall Smith, who was often the sole practitioner for 40 miles. At that time an operation at Rawene was five guineas, it was two guineas for a confinement and ,ten shillings and six pence for anti-tetanus serum. Wages on the relief schemes at that time were seven shillings a day for a single European man. (Maori paid four pence less).[2] No Maori woman had a baby in the hospital in the study period. Some of the patients who were admitted during this period are still alive today and are still patients of our practice. Other names are representatives of long standing families in the area.

Table 1 shows the categories of admissions during the months of July, August and September in 1935 and 1998. Table 2 shows the details of inpatient deaths in 1935 and the equivalent period in 1998.

In 1935,there were 22 admissions for influenza in the three month period, of whom seven died. Six out of these deaths were Maori. The average age of the fatal cases was

Table 1. Types of admissions

	1998 July to September		1935 July to September	
Cases	**Number**	**Frequency (%)**	**Number**	**Frequency (%)**
Infections	60	28	44	30
Trauma	15	7	28	19
Sundries	21	10	12	8
Cardiac	20	9	1	1
Respiratory	27	13	5	3
Endocrine	10	5	0	0
Gastro-Intestinal	4	2	0	0
Neoplasia	12	6	1	1
Musculoskeletal sundry	8	4	0	0
Side effect drug/alcohol	7	3	0	0
Obstetric, Gynae	24	11	26	18
Routine surgical	0	0	23	16
Psychiatric	6	3	5	3
Total	**214**	**100%**	**145**	**1**

Table 2. Cause, place and age of death

Ethnicity*	Gender	Month of death	Age	Place	Cause
Mortality 1988					
m	M	July	41	Home	Myocardial Infarct
m	F	July	61	Home	Cancer Liver
e	M	July	38	Home	Carcinoid Tumour
e	F	July	69	Home	Cancer Uterus
m	M	July	51	Hospital	Cancer Liver
m	F	August	90	Home	Adenocarcinoma Uterus
m	F	August	89	Home	Pneumonia CCF
m	M	August	54	Hospital	Myocardial Infact CCF Diabetes
m	M	September	19	Hospital	Motor Vehicle Accident
e	M	September	85	Home	Myocardial Infarct
m	M	September	81	Home	Bronchial Cancer
m	M	September	64	Home	Myocardial Infarct
Mortality 1935					
m	F	July	2 weeks	Hospital	Septic Umbilicus
m	M	October	44	Hospital	Post Laparotomy?
m	F	August	12	Hospital	No diagnosis
m	M	July	27	Hospital	Influenza
e	F	September	62	Hospital	Rheumatism
m	M	August	69	Hospital	Jaundice
m	F	August	39	Hospital	Influenza
e	F	August	81	Hospital	Senile Decay
m	M	September	15	Hospital	Influenza
m	M	August	18	Hospital	Influenza
m	M	August	45	Hospital	Influenza
m	M	August	13	Hospital	Influenza
e	F	September	17	Hospital	Influenza

Ethnicity: m = Maori e = European

24 years old, with a range of 13 to 45 years. No record of post-mortem findings is available. Of the survivors, their average length of admission was 33 days. Deaths outside the hospital are not recorded. On searching the archives of the local newspaper, I could find no reports of an influenza outbreak being reported at this time. This suggests that these deaths were not regarded as untoward, but rather as part of normal life and death in 1935.

What was the influenza that killed these young people? In the New Zealand Government gazettes, that reported the 1919 influenza pandemic, there are summaries of post-mortem reports, which refer to fulminant pneumonia, with the predominant bacterial organism present being identified as H. influenzae. The average length of stay for them was 33 days. This prolonged stay suggests a slow recovery, the development of chronic conditions such as empyema and bronchiectasis would have been highly likely.

Considering my 22 years at Hokianga, I can remember only one nine year old dying from generalised staphylococcal septicaemia. However we regularly admit fit young adults with pneumonia who respond quickly to antibiotics and are discharged within a few days. The Xray usually showed lobar pneumonia, which responds to intravenous benzyl penicillin. Many other minor cases have been treated as outpatients.

In the present day a large percentage of our admissions are for cardiac conditions and diabetes. It is of note that only one admission out of 150 in the three months of 1935, was for a cardiac condition and none for diabetes.

In 1998 and 1935 the admissions for infectious diseases were strikingly similar (28% versus 30%). On the other hand there has been a large increase in admissions for neoplasia, non-infectious respiratory diseases (asthma) and for cardiac illness. Routine surgical admissions are not represented (the operating theatre being closed in 1989).

In 1935 eight out of the total of 13 deaths were from infections and that most of these were in relatively young people. To lose a young healthy patient to infectious disease is a rare event in a modern day doctor's career. If we return to the situation that existed in 1935, then our profession would have to carry a share of the blame. It is not often that medical science regresses, but the increasing development of microbial resistance must be considered a large step backwards

References

1. Ellis-Pegler RB. Antimicrobial resistance - can we, should we do anything about it? *NZ Med J* 1999, 112:349-51

2. Kemble-Welch. Smith of the North.Reed Publishers

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Insulin resistance: implications for Maori

Insulin resistance and it's association with hypertension, dislipidaemia, central obesity, Type II diabetes and ischaemic heart disease is increasingly well recognised and the prevalence of these diseases among Maori is high. Hyperinsulinaemia is the the link between these illnesses and the recognition of low carbohydrate, high protein diets as appropriate for such patients offers new avenues for health promotion and treatment of illness and loss of weight.

Insulin Resistance: Any disturbance in the binding of insulin to insulin receptors or in the receptors responses, then insulin resistance follows and higher levels of insulin are required for the given level of glucose. Insulin resistance is thought to be due to both genetic and environmental factors. For instance, there is a high rate of Type II diabetes amongst Maori that is thought to be genetically determined and, in addition, a diet high in foods with a high glycaemic index will lead to high insulin levels. High stress may also be contributory, as cortisol and sympathetic outflow increase, circulating glucose increases and therefore, insulin levels. Hyperinsulinaemia ensues.

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Hypertension: Traditionally we know the cause of 10% of cases of hypertension, the other 90% are labelled, essential hypertension. Insulin resistance, as evidenced by hyperinsulinaemia, has been found in approximately 50% of these cases. Hyperinsulinaemia increases sodium reabsorption, induces sympathetic nervous stimulation and it increases the levels of Insulin Growth Factor 1, causing smooth muscle hypertrophy.

Dislipidaemia: Hyperinsulinaemia has been found to derange lipids, raising the triglyceride and LDL and decreasing the HDL fraction of the total cholesterol profile. Insulin controls the rate limiting, HMG-CoA step in cholesterol production, the step that the statin group of drugs inhibits.

Central Obesity:. Hyperinsulinaemia increases lipid production, in particular, triglycerides as a product of glucose breakdown. Thermogenesis has also been shown to be impaired in hyperinsulinaemia thus increasing the efficency of weight gain and adiposity The fat distribution of someone with hyperinsulinaemia is apple shaped, ie central obesity.. Children that are overweight or obese

have been shown to be up to 50 times more likely to have hyperinsulinaemia.

Type II Diabetes: If insulin resistance is present, then the pancreas needs to synthesize more insulin to overcome that resistance and maintain normal glucose homeostasis. Hyperinsulinaemia ensues. When the pancreas is unable to keep up with demand, hyperglycaemia results.

Ischaemic Heart Disease: The combination of obesity, hypertension, hypercholesterolaemia and diabetes is well recognised as contributing to the development of IHD. For Maori, who also have a high prevalence of smoking, this combination is disastrous and accounts for much excess morbidity and mortality.

The advent of the high carbohydrate, low fat diet that has been pushed for the past 25 years as the diet of choice, has seen a significant increase in these illnesses and accounts for much Maori excess mortality and morbidity. Such dietary advice coupled with a particular genetic makeup in a low socio-economic environment have contributed to widespread hyperinsulinaemia with its sequelae. We all know that Maori diet - high in the saturated fatty acids and refined carbohydrates that have been specifically implicated. A significant proportion of my Maori patients have this group of diseases, commonly known as Syndrome X.

The advent of the high protein, low carbohydrate diet advocated by the Atkins diet, the Zone Therapy diet, the Protein Power Programme, Dr Chang's Super Fat Burning Diet and many others is therefore very interesting and reportedly has helped many people. The biochemistry is very simple -there is not enough glucose in such diets to induce hyperinsulaemia and protein and fat do not stimulate insulin. Rather, protein stimulates glucagon which mobilizes stored body fat as energy. The patients on these diets are told they can have as much protein and vegetable as they like to eat, but find that they are not as hungry and so typically will eat less. The evidence is conflicting whether muscle mass is maintained on this regime, but the theory is that if you eat more protein, then the need to rely on muscle protein, for gluconeogenesis is lessened, if necessary at all.

A diet higher in protein, lower in carbohydrate content, more particularly low in highly processed carbohydrates together with low saturated fatty acids would therefore seem to be the diet of choice for many Maori and non – Maori alike. There has been little headway gained in the health status of Maori. Lifestyle change has always been difficult to implement in any society and so it remains.

But if you can tell someone who has a weight problem with other features of Syndrome X that you have a diet where they can eat as much as they like (though steering them away from refined carbohydrates) which also may help to lessen their blood pressure, cholesterol and blood sugar measurements and possibly their need for some medications then it's a winner. In Ngati Porou Hauora we have begun such a programme and will be reporting on it at the next Te Ora Hui a Tau.

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HE KUPU HOU

he takirikiri	*a neuralgia*
he pakapaka	*blistered*