DIABETIC RETINOPATHY IN THE KATHERINE REGION OF THE NORTHERN TERRITORY

by

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Declaration

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university or tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference is made in the text.

I give consent to this copy of my thesis, when deposited in the University Library, being available for loan and photocopying.

15th of January 2003

Nandor Jaross

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Abstract

Diabetes has reached epidemic proportions among Aboriginal Australians (Daniel et al 2002). This thesis presents results from the Katherine Region Diabetic Retinopathy Study (1993 –1996). These results provide the first detailed information on the basic epidemiology of diabetic retinopathy and impaired vision in an Aboriginal diabetic population.

Results of the Katherine Region Diabetic Retinopathy (KRDRS) show that while on the basis of overall prevalence alone, diabetic retinopathy may not seem as major a complication in Aboriginal communities as in the general Australian diabetic population, the highest reported prevalence of clinically significant macula edema (CSME) in Australia, and vision-threatening retinopathy (VTR) prevalences similar to or higher than in the Newcastle Study of Diabetic Retinopathy (NSDR) (Mitchell 1980) suggest otherwise. This is especially so if prevalences are adjusted for previous laser treatment.

On the basis of the annual incidence rate the progression of diabetic retinopathy may seem to be slower than in the non-Aboriginal community in Australia, however the much higher incidence of VTR compared to the NSDR suggest otherwise. More so, since the time since diagnosis of diabetes in subjects and the observation period in the KRDRS was much shorter than in the NSDR (Mitchell 1980, 1985, 1990).

The prevalence of blindness in the Aboriginal diabetic population of the KRDRS was as high as in the non-Aboriginal Australian population approximately 30 years older (Newland 1996, Taylor 1997, Wang 2000). The major cause of impaired vision, monocular and binocular blindness was cataract, just as it was in Taylor's report 25 years ago in an Aboriginal

Australian population (Taylor 1977). Data from the KRDRS also show that impaired visual acuity, irrespective of whether ocular pathologies other than diabetic retinopathy are present, is a poor indicator of diabetic retinopathy, maculopathy, CSME or VTR.

On the basis of results from the KRDRS annual screening for diabetic retinopathy in Aboriginal communities is recommended. The method of screening selected must allow case identification to be followed by treatment. The individual needs of Aboriginal communities together with the spectrum of ocular pathology must also be considered.

Analysis of the KRDRS study populations in 1993 and 1996 shows that the burden on ophthalmic services will increase. The geographic and cultural environment of the people and the epidemiology of the disease in Aboriginal communities present health service planners and clinicians with substantial challenges related to surveillance, treatment and follow-up.

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List of abbreviations

ABS	Australian Bureau of Statistics
ADF	Australian Defence Forces
AMS	Area Medical Service
AHW	Aboriginal health worker
AITEC	Aboriginal Islander Top End Eye Center
AQEHS	Adelaide Queen Elizabeth Hospital's Study
BDR	background diabetic retinopathy
BMES	Blue Mountains Eye Study
BMI	body mass index
BSL	blood sugar level
cont	continued
CSME	clinically significant macular edema
DHS	Commonwealth Department of Health and Family Services
DM	diabetes mellitus
DMO	district medical officer
DRS	The Diabetic Retinopathy Study
ETDRS	The Early Treatment of Diabetic Retinopathy Study
FDA	Food and Drug Administration
freq	frequency
HbA1c	haemoglobin A1c
IDDM	insulin dependent diabetes mellitus
IDI	International Diabetes Institute
IOP	intraocular pressure
IRMA	intraretinal microvascular abnormalities
KDH	Katherine District Hospital
KRDRS	Katherine Region Diabetic Retinopathy Study
MEU	Mobile Eye Unit
max	maximum value

min	minimum value
MVIP	Melbourne Visual Impairment Project
NHMRC	National Health and Medical Research Council
NIDDM	non-insulin dependent diabetes mellitus
NPDR	nonproliferative diabetic retinopathy
NSDR	Newcastle Study of Diabetic Retinopathy
NT	Northern Territory
NTAEHC	Northern Territory Aboriginal Eye Health Committee
NTEHP	National Trachoma and Eye Health Program
No	number
OATSIH[1]	Office for Aboriginal and Torres Strait Islander Health
OATSIHS[1]	Office for Aboriginal and Torres Strait Islander Health Services
OGTT	oral glucose tolerance test (abbreviation in table 1 as per "Proposed categories for diabetes prevalence studies in Australian Aborigines. International Diabetes Institute (1998))
PATS	Patient Assisted Travel Scheme
PDR	proliferative diabetic retinopathy
RDH	Royal Darwin Hospital
PEH	past eye history
PMH	past medical history
PRP	panretinal photocoagulation
po	per os
pt	patient
RDH	Royal Darwin Hospital
QLD	Queensland
RAAF	Royal Australian Air Force
RACO	Royal Australian College of Ophthalmologists

[1] Please note the name change of the organisation in the references.

RN	registered nurse
SCHS	Sydney Children's Hospital Study
SD	standard deviation
THS	Territory Health Services
USA	United States of America
va	visual acuity
WA	Western Australia
WESDR	Wisconsin Epidemiology Study of Diabetic Retinopathy
YPLL	years of potential life lost
%	percent

Chapter 1

1. Introduction

Diabetic retinopathy is a feared and virtually unavoidable complication of diabetes mellitus. In its most severe form it leads to blindness which may well be bilateral.

We know that strict glycemic control may slow the progression of diabetic retinopathy, but management of this condition depends on detailed knowledge of the epidemiology of the disease in the population, adequate screening for the underlying disease and an effective management policy.

Most prerequisites for the control of diabetic retinopathy are missing in the Northern Territory of Australia.

This thesis reports the objectives and results of the Katherine Region Diabetic Retinopathy Study (KRDRS), the most ambitious project to date that addresses the epidemiology of diabetic retinopathy in Aboriginal communities of the Katherine Region in the Northern Territory.

Chapter 2

2. Objectives of the study

The Katherine Region Diabetic Retinopathy Study (KRDRS) was carried out in the Katherine region of the Northern Territory as part of the Territory Health Service's (THS) eye health program. Therefore, in the Katherine region clinical service provision and a health education program were supplemented by clinical research.

Primary objectives:

1. To describe diabetic eye disorders in Aboriginal people living in the Katherine region by

    - documenting the prevalence and incidence of diabetic eye disease in the known diabetic population of the region
    - monitoring the progression of diabetic retinopathy.

2. To describe the visual acuity in the known diabetic population of the Katherine region and describe the effect of various ocular pathologies (both diabetic and non-diabetic eye disorders) on vision.

3. On the basis of the findings of the KRDRS, to address the issue of service delivery in the Katherine region of the Northern Territory.

The following hypotheses are proposed:

1. Diabetic retinopathy in Aboriginal communities of the Katherine region of the Northern Territory is at least as prevalent as in non-Aboriginal Australians.
2. Diabetic maculopathy and clinically significant macula edema (CSME) are more prevalent in Aboriginal than in non-Aboriginal diabetics.
3. The prevalence of vision threatening retinopathy (VTR) is at least as high in Aboriginal than in non-Aboriginal diabetics.
4. VTR presents shortly after diagnosis due to the high prevalence of CSME.
5. The prevalence of diabetic retinopathy is higher in subjects living in a non-traditional environment.
6. The prevalence of diabetic retinopathy is as high in major centers as in smaller communities.
7. The progression of diabetic retinopathy is at least as fast in Aboriginal as in non-Aboriginal diabetics.
8. The progression of diabetic retinopathy is at least as fast in major centers as in smaller communities.
9. The visual acuity in the Aboriginal diabetic population of the Katherine region is affected by diabetic retinopathy as well as by other ocular pathologies.
10. CSME is not inconsistent with good vision.

Secondary objective:

That meeting the NHMRC guidelines (1991) on best practice for research in Aboriginal communities, combined with clinical practice based on the principles behind the guidelines, will lead to a long term beneficial effect on the eye health of the study subjects.

Chapter 3

3. Literature review

3.1 The epidemiology of diabetes in the Aboriginal and Torres Strait Islander community

There have been numerous reports on diabetes in Aboriginal communities from the early 1960s. The diagnostic criteria vary among these studies, but some conclusions may be drawn:

1. Aboriginal people have a higher prevalence of diabetes than non-Aboriginal Australians with a much younger average age of onset. (Wise, 1970; Bastian, 1979; O'Dea, 1982; Cameron, 1986; O'Dea, 1988; Williams, 1987; Guest, 1992, O'Dea, 1993; Gault, 1996)

2. The peak prevalence of NIDDM (Non-Insulin Dependent Diabetes Mellitus) is about 40 years, 30 years younger than in Europeans. (Glatthaar, 1985)

3. The reported prevalence of NIDDM in Aboriginal communities varies. The most significant difficulty in analyses of epidemiological studies is the loss of direct comparability as a consequence of the use of different methodologies and defining criteria for the screening and diagnosis of diabetes. In attempting to deal with this, the report from the International Diabetes Institute proposed four categories reflecting the key methodologies used (Table 1) (International Diabetes Institute, 1998). If only studies with category I - II are taken into consideration, O'Dea found the prevalence of diabetes in the

Kimberley region of Western Australia to be 12.5% in Aboriginal men and 6.3% in women above the age 35; there were no diabetics among those younger than 35 (O'Dea, 1982).

Category	Characteristics of study
Ia	Population-based representative sample, 75 g OGTT in all subjects, using WHO 1985 criteria and reporting results by sex and 10-year age groups.
Ib	Population-based representative sample, 75 g OGTT in all subjects, using WHO 1980 criteria and reporting results by sex and 10-year age groups.
II	Population-based representative sample, 75 g OGTT in all subjects, using WHO 1985 or 1980 criteria and reporting results by sex and two age groups.
III	Population-based representative sample, 75 g OGTT in all subjects, using WHO 1985 or 1980 criteria and reporting results by sex only.
IV	Population-based representative sample, using non-WHO screening procedures or self-reported or clinical diagnosis of diabetes, with or without sex and age stratification.

Table 1 Proposed categories for diabetes prevalence studies in Australian Aborigines (International Diabetes Institute, 1998).

In this result selection bias may have played a part, though O'Dea et al have taken all possible measures to minimize it. In this study of 162 Aborigines from the Wunumbal tribal group who were registered officially as being resident at Kalumburu, 71 were under 15 years of age and were not included in the study. Of the target population 15 years and over, 67 were surveyed. The 24 people not surveyed included 7 who were (1993) over 60 years and 15 who were under 30 years (most of whom were absent at the time). There is no account given for the drop out of two individuals. There were no known diabetics among those not surveyed. In a rural community of

the Northern Territory, O'Dea found the prevalence of diabetes was 6.1% in men younger than 35; there were no diabetics found among women in this age group (O'Dea, 1990). There was no information in the paper that would explain the latter finding. In those aged above 35 she found the prevalence of diabetes was as high as 23.5% in men, and 22.9% in women. Van Buynder found that the prevalence of diabetes in three different Aboriginal communities was 14%, 12% and 27 % respectively (Van Buynder, 1991). The latter community was a "desert community"; the other two were coastal communities. O'Dea found in a Central Australian westernized community the prevalence of diabetes in the age group younger than 35 was 0.9% in men, 5.3% in women (O'Dea, 1993). In the age group older than 35, the prevalence rates were 30.6% and 28.8% respectively. Gault reported a prevalence of diabetes in the 15-34 year age group of 2% in men and 6% in women (Gault, 1996). In the age group above 35 these prevalence rates were 19% and 13% for men and women respectively.

In a recent paper Daniel reported the prevalence of diabetes in a voluntary sample drawn from 15 highly remote communities of central, northern and north western Australia (Daniel, 2002). The climatic conditions in these communities ranged from the central desert areas to the tropical, monsoonal regions of the north west Australia. With the exception of people indigenous to the Cape York and Torres Strait areas these communities represent the spectrum of remote settings in which Aboriginal people reside in Australia. In accord with the agreement reached with these communities, the people in this report were de-identified. The representativeness of the sample surveyed was established relative to the national Australian indigenous population. The population surveyed was between 15 and 94 years old. The authors found that in this large

sample of 2626 subjects, who were representative of the Aboriginal Australian population, the prevalence of diabetes, when adjusted for age and gender, was 14.8%.

The above data show that diabetes is more prevalent in Aboriginal communities than among non-Aborigines. Welbourn estimated an Australia-wide (non - Aboriginal) diabetes prevalence of 3% for men and women over 25 years of age, based on self-reported diabetes in the 1989-1990 Australian Bureau of Statistics (ABS) National Health Survey (Welbourn, 1995). Assuming a ratio of 1:1 for diagnosed and undiagnosed diabetes, a national prevalence of around 6% in adults was predicted (Zimmet, 1995). In Aboriginal Australians the best estimates indicate a likely overall prevalence of diabetes between 2 and 4 times higher than this, but with considerable variability among communities.

4. Published data support the notion that diabetes is an important cause of mortality in Aboriginal people. In the Northern Territory diabetes contributed to 3-5% of excess deaths in Aborigines aged 15-65 (compared with the general population), and 2% of years of potential life lost (YPLL) before age 65 (Cunningham, 1996). An additional 6 -10% of excess deaths and 5 - 10 % YPLL before age 65 were attributed to ischaemic heart disease. Ischaemic heart disease and a number of other causes of death (eg kidney failure) are likely to be related to diabetes. These figures may underestimate the effect of diabetes on mortality in the Aboriginal Australian population due to misclassification. Philips found that 44% of death certificates for Aboriginal people, previously diagnosed with diabetes, did not mention diabetes as a direct or indirect cause of death (Philips, 1995). Unfortunately, there are no specific data regarding the risk of diabetes on mortality in the

Australian Aboriginal population, but there are some data available from the Pacific region, which may be applicable to the Australian Aboriginal population (International Diabetes Institute, 1998). In Nauruans, diabetes was associated with an increased mortality risk of 4.5 fold in men, and 4 fold in women, compared to non diabetics (Zimmet, 1988). The relative risk of death of diabetics compared to non diabetics in Melanesian Fijians was 1.7 in men and 2.0 in women, and in Fijian Indians 4.2 in men and 3.2 in women (Collins, 1996).

5. There are only two studies reporting the incidence of diabetes in Aboriginal communities. McDermott reported an overall incidence of diabetes of 10.3/1000 person-years among homeland residents and 19.3/1000 person years among those living in centralized communities[1] (McDermott, 1998). Daniel during an eight year follow-up study found that the diabetes incidence rate was 20.3 cases/1000 person-years, with BMI (body mass index) specific rates of 10.7 - 47.2 cases/1000 person-years (Daniel, 1999). They concluded that BMI specific diabetes incidence rates in Australian Aboriginal people are among the highest in the world. Diabetes incidence in the lowest BMI category (10.7cases/1000 person-years) is two to five times greater than corresponding rates for non-Aboriginal populations.

From the literature review it is evident that diabetes is a major health problem in Aboriginal communities. The prevalence of diabetes is likely to increase in these communities as more people are exposed for longer to the adverse environments that have precipitated high rates in Aboriginal and Torres Strait Islander persons.

[1] Homeland denotes the land where Aboriginal people traditionally lived before the 1950s and 1960s before being moved to centralised communities. This is the area where some smaller or larger family groups moved back after the 1970s.

3.2 Overview of diabetic retinopathy

3.2.1 Diabetic retinopathy in Aboriginal Australian communities

There have been very few studies on the prevalence of diabetic retinopathy in Aboriginal communities, and only some of the results have been published so far.

The first report on diabetic retinopathy came from Edwards et al (1976). This team carried out eye examinations in 361 Aboriginal adults in South Australia. There were 26 known diabetics in this sample. He examined fundi through dilated pupils. Microaneurysms and haemorrhages were not observed, nor were any other signs of diabetic retinopathy, despite a diabetes duration of greater than 15 years in some cases.

The National Trachoma and Eye Health Program (NTEHP) did not systematically identify all Aboriginal people with diabetes, nor did it specifically perform dilated retinal examinations on all known diabetics. Only 59 (0.1%) of the 62116 Aboriginal people screened by the NTEHP were identified as having diabetes, and only 32 (0.05%) had diabetic retinopathy (The Royal Australian College of Ophthalmologists, 1980).

Stanton studied 1218 diabetics in and around country towns of WA (Stanton, 1985). This diabetic population (who volunteered to attend the survey) consisted of 1084 Caucasians and involved, in the authors terms, 134 "part Aborigines". Among this population Stanton et al reported that "retinopathy was at least as common in Aborigines as in Caucasoids" and gave a prevalence rate of 12% for the presence of "obstructive" retinopathy. Analysis of the tables in the study shows that

in this Aboriginal population the overall prevalence of retinopathy and the prevalence of retinopathy in males and females were 31%, 20.2% and 35.9%, respectively. This compares unfavourably with the overall prevalence rate of 8% in the Caucasian population examined.

Philips found that 7 of 24 diabetic patients in the renal unit at Alice Springs had vision-threatening retinopathy (Philips, 1995). The total number of retinopathies in this diabetic population was not reported.

McKenzie in Bourke, NSW found that in a diabetic sample of 54, 10 (19%) had diabetic retinopathy; the number with vision-threatening retinopathy (VTR) was not provided (McKenzie, 1995).

Markey reported that out of the 140 people with diabetes in the Darwin Rural Chronic Disease Register only three were documented as having retinopathy (Markey, 1996). He also quotes the findings of another report (the health record audit of diabetics in the Top End) that revealed that out of 141 people with diabetes 63% had not seen an ophthalmologist in two years and, of those who had, 30% (16/52) had documented retinopathy. The chronic disease register of the Miwatji Aboriginal Corporation (East Arnhem) quoted in the report above states that 8.4% of their diabetic patients (6/72) had diabetic retinopathy.

Diamond in a very well designed study assessing the viability of non-mydriatic fundus photography for identification of diabetic retinopathy in rural WA, found that 22.6% (74/328) of eyes examined with combined diagnostic techniques had diabetic retinopathy and 10.1% (35/328) of eyes had vision threatening retinopathy (VTR) requiring laser treatment (Diamond, 1998).

The review of eye health in Aboriginal and Torres Strait Islander communities gives a summary of numerous unpublished data (Taylor, 1997). In 1995/96 in a volunteer sample of 21 diabetics in Victoria 14% had retinopathy, the VTR was not reported. In1996 in a large diabetic sample of 1059 in the Torres Strait and Cape York there were 91 eyes with vision-threatening retinopathy, the total number of patients with retinopathy was not provided.

There are no longitudinal data at present on diabetic retinopathy in Australian Aboriginals. Taylor in his report to the Minister Health and Family Services gave an excellent summary of the challenges of the management of diabetic retinopathy in remote communities (Taylor, 1997). He states that

- "the health service's staff in remote communities often does not understand the management of diabetic retinopathy once it has been detected"
- there were patients sent to ophthalmologists for treatment, but "were not treated, and rebooked for a second costly trip shortly thereafter"
- "compliance with the current recommendations for treatment was poor, largely due to the lack of adequate portable equipment." This results in very inconvenient trips to major centers which is only made more stressful by the lack of adequate organized help during and after the trip. (The author experienced that requests for support to be given to diabetics with poor vision and language difficulties were not always met due to the limitations of the Patient Assisted Travel Scheme (PATS).)
- in QLD and NT Katherine region there are portable lasers available and treatment could be given in the communities.(Taylor suggested that in these regions there may be a greater compliance with the treatment.)

- "often there is little follow-up of patients after initial laser treatment and also little possibility of this in remote communities, which contributes significantly to the poor management of diabetic retinopathy in the communities"
- "databases of patients with diabetes are irregularly kept, although most clinics had some form of listing."

London and Guthridge explored the concepts and beliefs of members of a "remote Aboriginal community" about diabetes (London 1998). While it was encouraging that one person mentioned the possibility of eye complications, it was evident that more diabetes education was needed in that community/region. This paper unfortunately does not reveal which region this community belongs to. Since Guthridge was working in the Darwin Rural District it is most likely that the study was carried out in the same region. It would be useful to know the exact location of this "remote Aboriginal community", since between 1992 and 1996 the Katherine region received more attention to eye disorders than other regions. During that time the region had a number of programs including cataract camps, block surgeries, Mobile Eye Unit (MEU) field trips, first Azythromycin treatment trial for eradication of Chlamydia Trachomatis and the use of portable lasers in communities, therefore the awareness of eye disorders including diabetic eye complications must have been higher in the Katherine region than in other health districts.

There are no reliable data on diabetic retinopathy at present in the Aboriginal Australian population. Unfortunately, none of the papers associated with Edwards' study informs us of the total number of known diabetics in the region; from the response rate it is likely that selection bias is present in the study (it is quite remarkable that they did not find any retinopathy in this diabetic population) (Edwards, 1976; Wise,

1976). In the other studies the results are questionable due to selection bias or bias in the reporting. Even the most carefully designed study by Diamond, to evaluate the use of non-mydriatic fundus photography for identification of diabetic retinopathy in rural communities in Western Australia, gives cross-sectional information only about an "Aboriginal population" (Diamond, 1998). While it was not the primary purpose of this study, it still does not provide information about the total number of diabetics (screened and not screened) in the five selected communities, nor does it inform us of the make-up of the population (traditional or non-traditional Aboriginal population).

From this review it is clear that there are no reliable data on the prevalence and incidence of diabetic retinopathy in Aboriginal diabetics.

3.2.2 Screening for diabetic retinopathy in Aboriginal Australian communities

Approximately 20 years ago it was observed that diabetic patients at risk of loss of vision from severe retinopathy, who might benefit from laser photocoagulation, were not receiving appropriate ophthalmologic care (Witkin, 1984). Based on models incorporating epidemiologic and clinical trial data, it was estimated that early detection and timely photocoagulation treatment of proliferative diabetic retinopathy and CSME would result in a considerable amount of person-years of sight saved, would be cost effective and would reduce human suffering and lost productivity due to blindness (Dasbach, 1991).

There have been numerous publications on screening for diabetic retinopathy from the 1990s. However, in the Australian ophthalmic literature, as early as 1981 Mitchell (on the basis of his clinical

experience) recommended the combined use of direct ophthalmoscopy with other methods (retinal photographs or equivalent) for the screening of diabetic retinopathy (Mitchell, 1981). These recommendations were supported by the Retinopathy Subcommittee of the Australian Diabetes Society for Diabetes Australia (Diabetes Australia, 1988).

In the years following these recommendations it become increasingly clear that diabetics in remote areas were unable to access the same level of service as their city counterparts. Harper confirmed these recommendations and also raised the possibility of using fundus cameras alone for screening of diabetic retinopathy (Harper, 1995). There are numerous publications in the ophthalmic literature suggesting the use of fundus camera alone for screening of diabetic retinopathy (Pugh, 1993; Evans, 1997; Karagiannis, 1996; Diamond, 1998). However, the balance between screening sensitivity, patient benefits and cost effectiveness for diabetic retinopathy had not been studied until Javitt and Dasbach used data from the Wisconsin Epidemiology Study for Diabetic Retinopathy (WESDR) to model the cost of preventing diabetes related blindness in patients with IDDM and NIDDM against savings in disability payments in the US (Javitt, 1990; Javitt, 1994; Dasbach, 1991).

They found that among the strategies tested annual examinations of IDDM patients with semiannual examinations of those with retinopathy was the more sight saving and nearly as cost effective as less frequent examinations. A 60% or greater screening sensitivity maximized cost-effectiveness in screening for diabetic retinopathy. The model was relatively insensitive to variations in screening sensitivity over 60%, suggesting that there may be very little advantage in adding routine fundus photography to screening examinations for detecting very early

retinopathy in IDDM patients. Increasing the sensitivity from 60% to 100% provided a diminishing additional benefit due to the frequency of screening and the likelihood that retinopathy cases missed at one visit would be detected at the next. However, the cost benefits of the screening began to decline considerably at sensitivities below 60%.

In patients with NIDDM it was found that changing the frequency of screening for patients with no retinopathy or mild non-proliferative diabetic retinopathy (NPDR) from 1 to 2 years had little detrimental effect on sight-years saved, but reduced screening costs. In patients who developed moderate NPDR or more advanced retinopathy, sight-years saved were then sensitive to the screening interval. It is also interesting that while in people taking insulin the annual screening program costs were recovered, in NIDDM patients not taking insulin the screening cost were not recovered by avoiding the costs of blindness. (Dasbach, 1991). Their analysis however did not take into account the "analytic perspective of the federal budget nor did they consider savings associated with treatment of clinically significant macular edema" (Javitt 1994). After correcting for these Javitt et al found that substantial federal budgetary savings may result from effective screening and prompt treatment of retinopathy in patients with type II diabetes (Javitt, 1994).

In the publications recommending the sole use of fundus camera for screening of diabetic retinopathy the sensitivity of screening with a fundus camera met the 60% sensitivity required to maximise cost-effectiveness and benefits (Pugh, 1993; Evans, 1997; Karagiannis, 1996, Diamond, 1998; Javitt 1990,1994).

A change in the screening schedule for the Australian population was recommended by the National Health and Medical Research Council

(NHMRC) (National Health and Medical Research Council 1997). It took into consideration that in subjects with IDDM, significant retinopathy is rare in people aged less than 15 years with a disease duration of less than five years. In older IDDM patients with a disease duration of less than 5 years, retinopathy is almost always NPDR. For IDDM with onset before puberty, eye examinations could therefore be delayed until the onset of puberty. For IDDM after puberty, eye examinations should commence at the time of diagnosis. In subjects with NIDDM diabetic retinopathy is present in 10 –15% of patients at the time of diagnosis of diabetes and in Klein's study (1992) 2% of subjects had vision threatening retinopathy (all cases were clinically significant macula edema [CSME]). Therefore, for all patients diagnosed with NIDDM the NHMRC recommended screening for diabetic retinopathy as soon as practicable after diabetes is detected (National Health and Medical Research Council 1997).

Constable reports on the use of digital imaging systems (a portable Nidek fundus camera and a prototype monocular indirect ophthalmoscope constructed at the Lions Eye Institute) for screening of optic disc cupping, glaucoma and clinical signs of diabetic retinopathy (Constable 2000). They carried out five series of experiments to "explore progress, in the adaptation to community screening for blinding eye disease, of digital imaging devices and technology for storage and transmission". In relation to diabetic retinopathy the authors found that digital images did not compare favourably with those obtained by gold standard photographs. Only 24% of the digital images were graded as good quality by three ophthalmologists, and only 56% as acceptable quality for diagnosis of minute diabetic lesions such as microaneurysms. At the same time, 93% of the photographs were graded as good quality images for the diagnosis. The overall agreement between digital images and photographs was less than satisfactory

(kappa < 0.30). The method of subject selection or the sensitivity and specificity of the digital imaging was not reported in the study.

Lin reported the sensitivity and specificity of single – field nonmydriatic monochromatic digital fundus photography with remote image interpretation for diabetic retinopathy screening (Lin 2002). They found a highly significant agreement (kappa = 0.97, P = 0.0001) between the degree of retinopathy detected by a single nonmydriatic monochromatic digital photograph and that seen in seven standard 35-mm color stereoscopic mydriatic fields. The sensitivity of digital photography compared with colour photography was 78% with a specificity of 86%. Agreement was poor (kappa = 0.40, P = 0.0001) between mydriatic ophthalmoscopy and the seven-field standard 35-mm colour photographs. Sensitivity of ophthalmoscopy compared with colour photography was 34%, with a specificity of 100%. The authors conclusion that "single-field nonmydriatic monochromatic digital photography with remote interpretation is a sensitive and specific means for the detection of retinopathy in high-risk populations," while somewhat overstating its utility based on their data (Klein and Klein 2002) is consistent with those from other studies showing that digital photography may provide a useful alternative to film-based detection of retinopathy (Lin, 2000; Tennent; 2001; Bursell, 2001; Fransen, 2002). The use of digital imaging for the screening of diabetic retinopathy may be a valuable tool, there are several issues however that need to be considered before embarking on a screening program using digitised retinal images (see discussion).

In Australia, the Retinopathy Subcommittee of Diabetes Australia produced guidelines for the screening of diabetic retinopathy in 1988 (Diabetes Australia, 1988), later followed by the guidelines of the NHMRC (National Health and Medical Research Council, 1997).

However, compliance with these recommendations was poor (Harper, 1995) or "suboptimal" in the community (Bamroongsuk, 2002). The Melbourne Visual Impairment Project (MVIP) found that 50% of people with diabetes could not recall having a dilated fundus examination in the past 2 years and that one-third of people with diabetes could not recall ever having had a dilated fundus examination (McCarty, 1998). Similarly, in Aboriginal communities in Australia, compliance with these recommendations was also poor (Markey, 1996). Markey quoted the findings of the health record audit of diabetics in the Top End that revealed that out of 141 people with diabetes 63% had not seen an ophthalmologist in two years (Markey,1996).

The current guidelines of the Office for Aboriginal and Torres Strait Islander Health suggest annual screening for diabetic retinopathy in Aboriginal communities using fundus camera and immediate referral to ophthalmologists of subjects with VTR (OATSIH, 2001). According to these guidelines non - urgent referral is warranted for subjects with visual acuity less than 6/12 and no diabetic retinopathy, with unexplained visual loss or with ungradable photos. While the OATSIH guidelines are specific in their recommendations, data from the Australian medical literature show that there was a breakdown in the application of the screening principles for diabetic retinopathy in the wider Australian community, as well as in the Aboriginal Australian population. There were very few publications that address some of the issues relevant to screening for diabetic retinopathy in Aboriginal Australians (Karagiannis, 1996; Keeffe, 1997; Diamond, 1998.

From the literature it is also clear that there was no systematic screening of diabetic Aboriginal populations for diabetic retinopathy before the KRDRS. This is also confirmed in the Taylor report which

summarizes all published and unpublished information on diabetic retinopathy in Aboriginal communities (Taylor, 1997).

3.2.3 The community impact of diabetic retinopathy in Aboriginal Australians

To be able to estimate the community impact of diabetic retinopathy in Aboriginal Australians, the following information is required:

- the prevalence of diabetic retinopathy in Aboriginal communities
- the incidence of diabetic retinopathy and the rate of progression in a known diabetic population of Aboriginal Australians
- the prevalence and degree of visual impairment from diabetic retinopathy in Aboriginal Australians.

Regarding the prevalence of diabetic retinopathy there are numerous publications (see chapter 3.2 - Diabetic retinopathy in Aboriginal Australian communities) but there have been no longitudinal studies carried out so that the incidence and the rate of progression of diabetic retinopathy in Aboriginal Australians could be determined.

As did the NHMRC (National Health and Medical Research Council, 1997) it has to be concluded that there are no records regarding the distribution of visual acuity or even the prevalence of blindness in a diabetic Australian population (Aboriginal or non-Aboriginal), so quantification of the impact of this disease on the community cannot be made.

3.2.4 Diabetic retinopathy in non-Aboriginal Australians.

There are five large studies that provide information about diabetic retinopathy in the non-Aboriginal Australian population. These include the Newcastle Diabetic Retinopathy Study (Mitchell (1980,1985,1990), the West Australian Study (Knuiman, 1986), the Sydney Children's Hospital Study (Fairchild, 1994), The Adelaide Queen Elizabeth Hospital Study (Phillipov, 1995), the Blue Mountains Eye Study (Mitchell, 1998) and the Melbourne Visual Impairment Project (McKay, 2000). The KRDRS is most comparable to Mitchell's study, since his study (even though it was not strictly population based) most likely included all known diabetics from the target population. The other studies were not population based or were restricted to limited age subsets.

It is therefore important to summarize the findings of Mitchell's study since it will be used to compare the prevalence and progression of diabetic retinopathy with the KRDRS(Mitchell, 1980; Mitchell, 1985). This comparison will show if the characteristics of diabetic retinopathy differ in Aboriginal communities compared with the non-Aboriginal Australian population. There are, however some discrepancies in the reporting in Mitchell's second and third publication (Mitchell, 1985; Mitchell, 1990) when the papers refer to the "initial survey" already described in his first publication (Mitchell, 1980). In his first report Mitchell describes the overall prevalence of diabetic retinopathy, the prevalence of proliferative diabetic retinopathy, maculopathy and "threatened maculopathy" in the Newcastle diabetic clinic and in the Singleton Census area (Table 3 of his publication) (Mitchell, 1980). Table 3 of his later publication (Mitchell, 1990) presents the same data. In his first publication he writes "Macular involvement occurs in stages 2

and 3 as the result of exudation or loss of macular capillaries. Oedema and hard exudates originate most frequently from leakage into the lateral macular region and from the perifoveal capillary bed." (Mitchell, 1980). There are, however no definitions given to "threatened maculopathy". It is not clear if it refers to CSME, or whether maculopathy is just about to occur. In a later publication presenting the same data maculopathy was defined as "central visual loss due to macular dysfunction" (Diabetes Australia, 1985). It does not cover the terms for clinically significant macula edema (CSME) since it has been shown that CSME is not inconsistent with good visual acuity. Also, macular capillary closure while it may result in central visual loss, may not necessarily cover the definition of CSME. Therefore, I will refer to Mitchell's data from his first publication (Mitchell, 1980) but since the author used the term "maculopathy" as part of the category of VTR in his later publication, these figures are also presented in this thesis. It should be remembered that these figures could be biased according to our current understanding of VTR.

The detailed presentation of Mitchell's results in this thesis is important, since (despite the limitations in the terminology) his study is the most comparable to the KRDRS in Australia. Moreover, his study formed the basis of the screening schedule proposed for diabetic retinopathy in Australia (Mitchell, 1980; Mitchell, 1990; Diabetes Australia, 1988) until the NHMRC guidelines were published in 1997 (National Health and Medical Research Council 1997).

In the Newcastle diabetic clinic, Mitchell found the prevalence of ophthalmoscopic retinopathy, proliferative diabetic retinopathy, maculopathy, "threatened maculopathy" and vision threatening retinopathy (VTR) as 49%, 7%, 10% and 2%, respectively

(Mitchell, 1980). The same findings for the Singleton census area were 36%, 3%, 6% and 3% respectively.

In his later publication Mitchell modified his figures giving a prevalence for ophthalmoscopic retinopathy, proliferative diabetic retinopathy, vision-threatening maculopathy and VTR of 49%, 7%, 10% and 13% in the Newcastle diabetic clinic. The comparable findings for the Singleton Census area were 36%, 3%, 7% and 8%, respectively (Mitchell, 1985).

Mitchell also reported the progression of diabetic retinopathy (Mitchell, 1985; Mitchell, 1990). He found that the annual progression rate from no retinopathy to any retinopathy was 8% and from no retinopathy to VTR was 0.4%. Of those with mild background diabetic retinopathy (BDR), 8% progressed annually to a worse grade diabetic retinopathy and 4% to VTR. The progression of maculopathy and CSME were not reported in detail.

Mitchell also found that for the development or progression of retinopathy a slightly higher rate was seen in diabetics with an onset at the age of 40 or over compared with those with a younger age at onset (Mitchell, 1985). The annual rate of progression of retinopathy ranged from 5.8% (for diabetics with an onset under the age 40 and a duration less than five years) to 9.1% per year (for diabetics with an onset at the age of 40 and a duration of 10 years or more). He also found that maculopathy developed twice as commonly as proliferative retinopathy, particularly in older diabetics. Proliferative signs on the other hand developed more frequently in diabetics with a younger age at onset (the rate for patients with an onset under the age of 40 was twice that for the group with an onset at the age of 40 and over). Incidence rates for the new development of background diabetic retinopathy (BDR) in previously unaffected subjects showed a peak late in the first decade of

diabetes in the young-onset group, while in the older-onset group, the peak occurred in the second decade of diabetes. On the other hand, for progression from early to more advanced or proliferative stages of retinopathy, typically the rate peaked in the second to third decade of diabetes.

On the basis of his results Mitchell proposed an evidence-based screening schedule for diabetics (Mitchell, 1980; Mitchell, 1985). He suggested that if diabetes is diagnosed before age 30, retinal examination should start no later than 5 years after diagnosis. This view is still held if diabetes is diagnosed in prepubertal children. However if diabetes is diagnosed after puberty, the eye examination should commence at the time of diagnosis (American College of Physicians AD and American Academy of Ophthalmology, 1992; Retinopathy working party, 1991). Mitchell also suggested that screening for diabetic retinopathy should start immediately at diagnosis if the age at diagnosis was greater than 40, since approximately 18% of subjects diagnosed in the age group 40 - 49 has diabetic retinopathy at the time of diagnosis. His initial findings were confirmed by further studies (Klein, 1992; Mitchell, 1998).

The concordance of disease state between the two eyes has not been reported in any Australian studies, but the MVIP gives some indications about concordance in subjects with clinically significant macula edema (CSME) and vision threatening retinopathy (VTR) (McKay, 2000). This study that five of nine subjects with proliferative diabetic retinopathy (PDR) and five of 12 subjects with CSME were affected bilaterally.

These studies allow comparison of diabetic retinopathies in the Aboriginal Australian with the non-Aboriginal Australian population.

3.3 Overview of visual acuity in the Aboriginal Australian population

In discussing visual acuity in an Australian community the different definitions used in existing studies must be acknowledged. Before describing visual acuity in the Aboriginal Australian population and comparing to the non-Aboriginal Australian population, the definitions used in various reports will be provided.

Taylor and the National Trachoma and Eye Health Program (NTEHP) defined blindness as a best corrected visual acuity of 6/60 or less (Taylor, 1977; RACO, 1981). If only one eye was affected, the blindness was classified as monocular, if both eyes were involved the blindness was classified as binocular.

Later Stocks and Newland used both the Australian and WHO criteria to describe visual acuity (Stocks, 1994; Newland, 1995; Stocks, 1997).

Binocular blindness:

Australian criterion: Best corrected visual acuity < 6/60 in the better eye
WHO criterion: Best corrected visual acuity < 3/60 in the better eye.

Monocular blindness:

Australian criterion: in one eye of the subject the uncorrected visual acuity is less than 6/60 and the visual acuity is 6/60 or better in the other eye.

WHO criterion: in one eye of the uncorrected subject the visual acuity < 3/60 and visual acuity is 3/60 or better in the other eye

The NTEHP found that the prevalence of blindness was 1.49% in Aboriginal people compared to 0.16% of non-Aboriginal people (Royal Australian College of Ophthalmologists (RACO), 1980). The causes of blindness were substantially different for Aboriginal and non-Aboriginal people. Corneal disease was responsible for 50% of the blindness seen among Aboriginal people, including 41% due to trachoma. In contrast, only 7% of the blindness among the non-Aborignal people examined was due to corneal disease. Cataract caused 39% of blindness among Aboriginal people, and 23% among the non-Aboriginal people. Retinal disease caused 52% of blindness among non-Aboriginal people, but only 6% among Aboriginal people.

In 1989 – 1990, using the definition for legal blindness in Australia, the prevalence of blindness was 1.45% among Anungu Pitjantjatjara residents (Stocks, 1994). The NTEHP found the prevalence of binocular blindness was 2.3% in the Red Center (RACO 1980). If this overall prevalence of blindness in the Red Center were representative of the situation in the Anungu Pitjantjatjara communities, the 1989-1990 survey suggests that the prevalence of blindness in these communities has not changed significantly over the previous ten to fifteen year period. The difference in prevalence estimates from Stocks (Stocks, 1994) and the NTEHP (RACO, 1980) might be even less marked if it is considered that the 2.3% prevalence figure for binocular blindness in the RACO's report also includes those subjects who had a visual acuity of 6/60 and who are missing from the figures given by Socks (Stocks, 1994). The causes of blindness in 1989 – 1990 were similar to those found by the NTEHP (RACO, 1980), with trachoma and cataract each

responsible for around 40% of blindness. The prevalence of binocular blindness was 2% in females compared with 0.8% in males in Stocks' study (Stocks 1994); in the NTEHP the prevalence of binocular blindness was not reported by sex in detail, but it was found that there was a higher prevalence of binocularly blind females in the 60+ age group that was statistically significant only in the Red Center (RACO, 1980). In the Aboriginal population in the 60+ age group the prevalence of blindness was 22% in the NTEHP (RACO, 1980) compared with 14% in Stock's study (Stocks, 1994).

In the NTEHP the prevalence for monocular blindness was 2.7% among Aboriginals compared with 1% in the non-Aboriginal population. The distribution of monocular blindness was in 3.1% in males and 2.3% in females (RACO, 1980). Stocks reported monocular blindness according to the WHO criteria (Stocks, 1994) and comparison of their figures is difficult with those from the NTEHP (RACO, 1980). It is still evident that even if the prevalence of monocular blindness was less than that reported by the NTEHP, the burden of monocular blindness is still significant in the 60+ age group.

However, Stocks overestimated the blindness in the Pitjantjatjara land (Stocks, 1994). In both the NTEHP (RACO, 1980) and in Stocks' survey (Stocks, 1994) an active search for the blind was made and therefore a more accurate estimate of blindness could be obtained if the source population was used in the denominator. This method assumed that non-participants had (on average) better visual acuity than participants. This was confirmed when no additional blind individuals were found following attempts to identify the visual acuity of non-participants using community medical records and local health worker knowledge. A random sample of Aboriginal people would have provided a better

estimate of blindness but this was impractical in Aboriginal communities.

Stocks, using the source population in the denominator, found that in the Anungu Pitjatjatjara the prevalence of poor vision decreased from 7.7% in 1980 to 4.1% in 1990 (Stocks 1997). The respective figures for blindness were 1.6% and 0.9%. However, despite the overall decrease in blindness in the population they found that in the 60+ age group the prevalence of blindness was still very high (10.4% overall, 6.7% in males and 13.6% in females). They concluded that further improvement in the eye care of the rural population, in particular in the elderly, needed to occur.

Both Taylor and Stocks pointed out that almost all blindness in their Aboriginal study populations was preventable (Taylor, 1977; Stocks, 1994).

Several studies in Australia and overseas have indicated the high frequency of undercorrected refractive errors in the older community. Investigators from the Baltimore Eye Survey reported that 54% of their participants improved their vision by at least one Snellen line with refraction and 7.5% improved by three or more lines (Tielsch, 1990). The Melbourne Visual Impairment Project found that 10% of subjects presenting in the study improved their vision by one or more lines after refraction (Liou, 1999). This represented 57% of the subjects with visual acuity of 20/20 minus two letters. The Blue Mountains Eyes Study found that refraction improved visual acuity by one or more lines in 45% of participants and by three or more lines in 13% of subjects (Attebo, 1996). There has been no description of refraction on vision Aboriginal Australians.

In Chapter 8 of this thesis the visual acuity findings in the KRDRS study population will be reported. This study aims to determine whether diabetic retinopathy is the major cause of decreased vision among Aboriginal diabetics or whether those ocular conditions previously described by Taylor, the NTEHP and later Stocks are the major cause of impaired vision (Taylor, 1977; RACO, 1981; Stocks, 1994).

Chapter 4

4. Study description

4.1 Study type

The KRDRS was a longitudinal study of known Aboriginal diabetics in the Katherine region of the Northern Territory. It was intended to examine subjects for diabetic retinopathy in 1993 and in 1996.

4.2 Description of the study population

4.2.1 General overview

The study population consisted of all known Aboriginal diabetic patients who were residents at the time of community visits in Katherine region. This study population was drawn from a total regional population of 14990, of which 6290 were Aboriginal persons. 5618 of the Aboriginal residents were living in remote areas. The majority of the known Aboriginal diabetics of the region were living in Katherine and three other major centers Borroloola, Kalkaringi and Lajamanu. A considerable effort had to be made so that diabetics in smaller communities were followed up with equal determination to avoid selection bias in the study population.

The study population was recruited using information from several sources:

- the chronic disease register of Katherine region of the Northern Territory

- diabetics known by the nursing staff of the community
- information gathered from the Aboriginal Health Workers regarding the whereabouts of the diabetic patients

The strategy of recruiting an entire defined population using multiple overlapping population lists minimises the possibility of selection bias.

The "chronic disease register" was the innovation of the DMOs (District Medical Officers) in Katherine region. A team of DMOs found it very difficult to follow Aboriginal patients with chronic diseases due to the mobility of the population and the frequent change of DMOs in the region. Therefore, a database was created that contained vital information on all chronically ill patients. This database was regularly updated after the scheduled medical visits in the community. Before a visit by the KRDRS to the communities, the DMOs provided the community health centers and study staff with a list of the known diabetics from the community.

Due to the mobility of the Aboriginal population, there may have been diabetic Aboriginal people living in communities who only came for a visit for a certain period of time from another community or from another region. The DMOs may not have known these Aboriginal diabetics, but the nursing staff was aware of them. These patients came to the health center occasionally "to have their sugar checked" or for other minor ailments. Since the DMOs did not see them it was unlikely that they were placed on the chronic disease register. Therefore, the second source of vital information was the nursing staff of the communities.

The third source of information was the Aboriginal health workers (AHW). The whereabouts of some known diabetics in the communities was often uncertain. Sometimes diabetics decided to live at outstations and their whereabouts was not known to the health centers. The information from

the AHWs was essential in locating these diabetics; this way all diabetics in the communities could be accounted for.
To overcome the difficulties of spreading information in the communities the local media were also utilized to announce the time and place of the following week's visit of the Mobile Eye Unit. (For detailed description see Chapter 4 section 5.)

4.2.2 Subclassification of the study population in 1993 and in 1996

While consideration of the entire study population provides information on the prevalence of retinal complications of diabetes in 1993 and in 1996, it is only by subclassification of the populations that we can understand the dynamics of eye disease over time and hence an appreciation of the burden of disease.

The following subgroups are described within the study population:

a. core population: This group consisted of those subjects in whom at least one of the retinas could be examined in 1993 *and* in 1996. These subjects provided the basis for a longitudinal study.

b. non-core population of 1993: This group consisted of those subjects in the 1993 study population who could not be re-examined in 1996. These subjects were those whose lack of attendance in 1996 might have been a source of information bias in a longitudinal study.

c. non-core population of 1996: Those subjects in the study population of 1996 who were "newcomers" to the study, regardless of their time of diagnosis of diabetes. These may be further broken down to the

- newly diagnosed group: Those subjects in the non-core population of 1996 who were diagnosed with diabetes between 1994 - 1996.
- non-newly diagnosed group: Those subjects in the non-core population of 1996 who were diagnosed with diabetes before 1994. (These subjects were not seen in 1993.)

Figures 4.1 and 4.2 show the subgroups in relation to the study populations in 1993 and in 1996. The non-core group is shaded; in the non-core group of 1996 the two different shades represent the two subgroups of the non-core population of 1996: the group diagnosed with diabetes before 1994 and the one diagnosed with diabetes between 1994 and 1996.

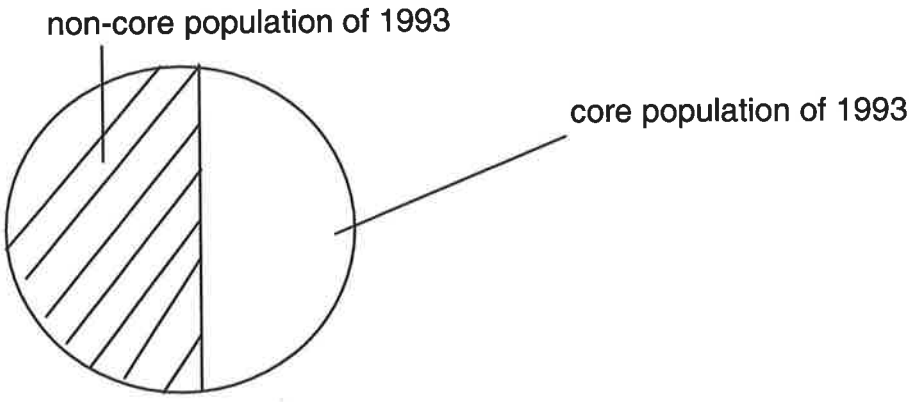

Figure 4.1 Subgroups of the study population in 1993

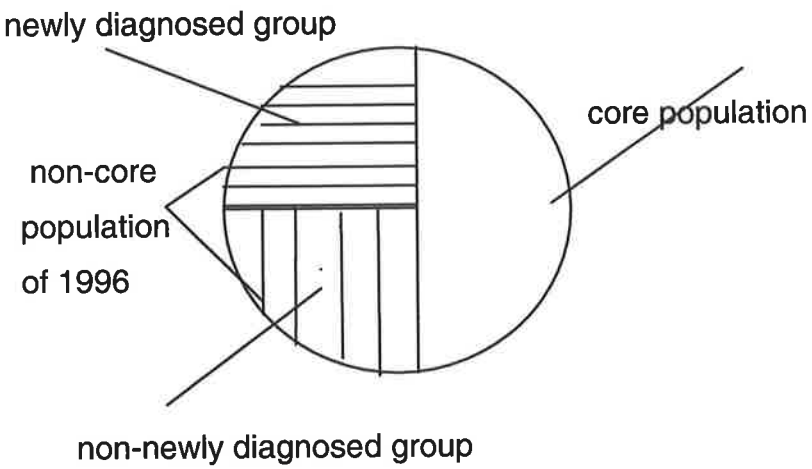

Figure 4.2 Subgroups of the study population in 1996.

4.3 Description of the study area

The "Top End" of the Northern Territory (NT) lies between longitudes 129 - 138 degrees E and latitudes 11 - 19 degrees N. It comprises 4 health districts - Darwin rural, Darwin urban, East Arnhem and Katherine regions. It covers an area of 517044 km^2 and has a population of 117290.

Health care is provided through community clinics, Katherine Hospital and Gove District Hospital and urban general practitioners with both NT government and independent health agencies servicing Aboriginal communities.

Clinical experience shows that Katherine region is similar to other Top End communities with respect to the high prevalence of eye disease, particularly in Aboriginal communities and including conditions, which, if untreated, will lead to blindness.

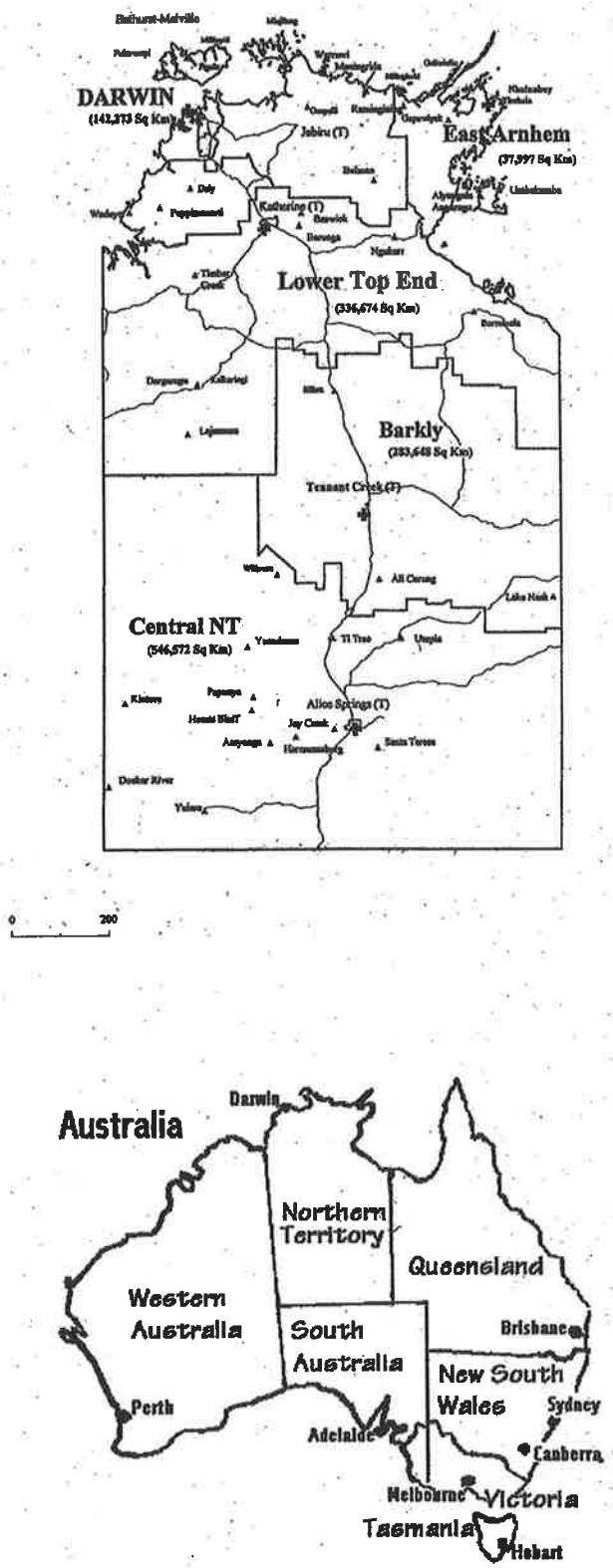

Fig 4.3 The Lower Top End in relation to Australia (scale in kms for the top map).

Katherine region of the Northern Territory lies between longitudes 129 - 138 degrees E and latitudes 14 - 18 degrees N and covers an area of 336674 ^{km2}. (Fig 3) The climate is subtropical, though the Southern border of the region reaches the edges of the Tanami desert. The dry season lasts between May and November, however some of the roads may still be blocked in May or already blocked in October. During heavy rain the roads can suddenly be blocked and the settlements become approachable by plane only, or, if the airstrip is closed, by helicopter.

The major roads in Katherine region are the Stuart Highway, the Victoria Highway and the Carpenteria Highway. The first two are two-lane bitumen roads; the latter is in some parts two lanes, in some parts one-lane bitumen road. The other roads to the communities are mostly dirt with one lane only.

These characteristics have major significance for planning any clinical activities in this region. The roads are dangerous and travelling time always has to be taken into consideration when planning work in the bush. When prioritizing work, allowances have to be made for the climatic conditions and basic survival skills must be taught to those travelling on their own into these communities.

4.4 Glossary of terms

The following terminology is used throughout the rest of this thesis:

4.4.1 Diagnostic criteria of diabetes

Health records of the study subjects were examined and diabetes was accepted as a diagnosis on the basis of the WHO criteria (1985):

- typical symptoms plus unequivocal elevation of random venous glucose (greater than or equal to 11.1mmol/l), or
- fasting (overnight) venous plasma glucose greater than or equal to 7.8 mmol/l, or
- following ingestion of 75gm oral glucose, venous plasma glucose greater than or equal to 11.1 mmol/l at 2 hours.

4.4.2 Diabetic retinopathy

Diabetic retinopathy may be defined as the presence of typical retinal microvascular changes in a person with diabetes. However, none of the individual lesions are specific for diabetes, as they may occur in other disease processes such as hypertension, hyperviscosity and radiation. It is the pattern, symmetry and evolution of the lesions that characterises the appearance of diabetic retinopathy.

Nonproliferative diabetic retinopathy (NPDR): the retinal pathology is at the level of the retina itself.
Mild NPDR: at least one microaneurysm.

Moderately severe NPDR: extensive intraretinal haemorrhages and/or microaneurysms and/or cotton wool spots, venous beading, or intraretinal microvascular abnormalities (IRMA) definitely present, but not as severe as "severe" NPDR.

Severe NPDR: the 4-2-1 rule (*either* intraretinal haemorrhages in all four quadrants, *either/or* venous beading present in two quadrants, *either/or* IRMA in one quadrant).

Proliferative diabetic retinopathy: changes are present outside the level of the retina. They consist of fibrovascular tissue and/or retrohyaloid haemorrhage and/or vitreous hemorrhage.

Diabetic maculopathy: a particular type of background diabetic retinopathy where the background changes are localized to the macular area.

Clinically significant macular edema (CSME):
One or more of the following:

- retinal thickening at or within 500 micron from the center of the macula
- hard exudates within 500 micron of the macula associated with adjacent retinal thickening
- a zone or zones of retinal thickening one disc area in size at least part of which is within one disc diameter of the center of the fovea.

Vision threatening retinopathy: proliferative diabetic retinopathy and CSME.

4.4.3 Definition of Aboriginality

The definition of an Aboriginal or Torres Strait Islander person which serves as the basis for this study was given in a High Court judgement in

the case of Commonwealth v Tasmania (1983) 46 ALR 625. In this decision "Australian Aboriginal" refers to a person "of Aboriginal descent, albeit mixed, who identifies himself as such and who is recognized by the Aboriginal community as an Aboriginal". The three components of this definition are descent, self identification and community acceptance. The Australian Bureau of Statistics has also accepted the above definition (ABS 2002).

For the purpose of these studies, Aboriginality was accepted on the basis of the patient viewing him/herself as such.

4.4.4 Definition of "traditional" and "non-traditional" environment

Traditional community: An Aboriginal community where the vast majority of diabetic and non-diabetic Aboriginal people have their meal/food from the local store but a considerable portion of their food derives from fishing and hunting.
Non-traditional community: Aboriginal community where the vast majority of people (diabetic and non-diabetic Aboriginal people alike) have their meal/food purchased at the local store or provided by other aged care facilities.

4.4.5 Definition of major centers and smaller communities

The distribution of diabetics in the region, and the relatively better access to services in some, warrants the introduction of the term "major centers". This term refers to those communities where there were more than 20 known diabetics and where access to services was better than in the rest of the communities. The term "major centers" includes Borroloola, Kalkaringi, Lajamanu and Wurli-Wurlinjang Health Center. Since equal access to services is always an issue in the ophthalmic care of people in

the Katherine Region, a description of diabetic retinopathy in terms of major centers may provide data to suggest better resource allocation.

4.4.6 Definitions relating to visual acuity

Subject vision refers to the visual acuity of the better eye of the subject. It can be measured without correction, referred to as "uncorrected subject vision" or with correction, referred to as "best corrected subject vision".

Impaired vision refers to a visual acuity less that 6/18. If measured at the subject level it means that the visual acuity of the subject is worse than 6/18 in the better eye; if measured in each eye, it refers to the respective eye. It too can be measured with and without correction.

Blindness refers to a vision less than 6/60. Monocular blindness refers to a situation when only one, but not both eyes are blind; in binocular blindness this is the case in both eyes. Again, acuity can be measured with and without glasses. In this thesis the Australian blindness criteria are used. (See Chapter 3, section 3)

4.5 The Mobile Eye Unit (MEU)

4.5.1 Set up of the unit

The MEU was an essential element of the NT Eye Health Program and KRDRS. Clinical services and research in Katherine region would not have been possible without a mobile eye health unit that took services to remote areas. This was a new concept that was enthusiastically supported by health professionals in the Northern Territory. The unit was set up and successfully trialled in 1993. Funding for the operation of the unit came

from various sources (NT Aboriginal Eye Health Committee, Territory Health Services, ATSIC, Commonwealth Department of Health, and from fundraising from Sunrise Rotary Club and Driver Primary School and from private donations).

The purpose of the unit was:

- to take the same ophthalmic services to remote areas of the Northern Territory that was available in central hospitals
- to gain understanding of the dynamics of eye disorders in Aboriginal communities
- to be involved in education of the communities regarding eye health
- to collaborate with other agencies in the provision of "cataract camps" (intensive block surgeries)
- to provide information to health administrators so that services could be planned.

The unit consisted of an ophthalmologist[1], an optometrist, an Aboriginal Health Worker (AHW) and a volunteer nursing sister. The optometrist carried out refractions so glasses could be provided immediately. The volunteer nursing sister was a vital link in communicating with the clinic and she also checked blood pressures, took urine samples, collected blood for further analyses and assisted at treatments. The AHW, with their local knowledge of the communities, ensured that those requiring attention came to the mobile clinics and they were also involved in patient care whenever they showed interest. The ophthalmologist carried out full eye examinations and was also responsible for taking retinal photographs of diabetics. He also assumed general responsibilities for the clinical activities.

[1] The author of this thesis.

For the initial series of community visits Norforce[2] assigned one of its members and provided a tent, generator, general survival advice and training.

Later the Mobile Eye Unit carried out its work with the team consisting of an optometrist, an ophthalmologist and occasionally a nursing sister. Sometimes local Aboriginal health workers were also involved in the work of the unit. In Wurli-Wurlinjang Health Center the entire activity of the unit was supported by their health workers.

The establishment of the unit was a milestone in ophthalmic service delivery and research in remote communities in the Northern Territory.

4.5.2 Access to communities

To ensure the ongoing support of the communities the following actions were taken:

<u>prior to visits</u>

- presentation of the plan to the nursing staff of the region prior to the start of the project
- confirmation of the need for a visit with the local District Medical Officer (DMO)
- discussion with the staff of the health center about the most suitable time for the visit and about the activities planned
- discussion with the Community Government Council

<u>at the time of arrival</u>

[2] Norforce is a unit of the Australian Defence Force stationed in the Northern Territory.

- visit to the Community Government Council, and, if invited, attendance at the council meeting
- visit to the community elders

during the visit

- attendance, if invited, at meetings, ceremonies or visits to sacred sites

upon leaving the communities

- provision of information about the community visit to all interested parties (detailed medical information to the health center and DMO, summary information to community councils)
- ensuring that promises were kept and community benefits were delivered.

4.5.3 Quality control and feedback

To ensure that the work of the MEU was performed to the standards of the Royal Australian College of Ophthalmologists, the support of Dr Masoud Mahmood, Director of Eye Services at the Royal Darwin Hospital was enlisted. Between 1993 - 1995, when laser treatment was not available outside of Darwin, nearly all of the diabetics requiring laser were reviewed by Dr Masoud Mahmood and/or the senior registrar from Sydney Eye Hospital. The cataract camps were also carried out in consultation with Dr Masoud Mahmood.

To improve the MEU's performance in the communities a review form was designed that had to be filled in by local clinic staff commenting on the individual and team performance.

In 1993 the community visits were scheduled so that by the end of the dry season all communities had been visited. Only communities where the job could not be completed at the initial visit were visited in the build-up season[3]. Table 3 shows the visits of the MEU in 1993. As table 4 shows, the visits of the MEU changed considerably by 1996. Even though the same communities were visited as in 1993 considerably more time was spent in Wurli-Wurlinjang Health Centre. Wurli-Wurlinjang Health Center became a regional center for Katherine region where follow-ups could be carried out, including laser treatments using a portable argon laser.

[3] Buildup season refers to the months (August – November) prior to the arrival of the monsoon when the humidity increases.

4.5.4 Communities visited

Date	Community visited
26/04 - 30/04	Barunga (patients from Beswick transported)
03/05 - 05/05	Bulman
16/05 - 21/05	Kalkaringi
24/05 - 27/05	Lajamanu
30/05 - 31/05	Mataranka
01/06	Duck Creek
02/06	Hodgson Downs
03/06	Hodgson River
06/06 -10/06	Ngukurr
14/06 -16/06	Borroloola
17/06 -18/06	Pine Creek
21/06	Timber Creek
22/06	Kildurk
23/06	Timber Creek
24/06	Bulla camp
25/06	Yarralin
27/06 - 03/07	Wurli - Wurlinjang Health Centre
04/10 - 08/10	Borroloola
11/10 - 16/10	Lajamanu

Table 4.1 Community visits of the Mobile Eye Unit 1993

Date	Community
20/8/96 22/8/96 (separate clinics)	Barunga Beswick
22/04 - 25/04	Kalkaringi
29/04 - 03/05	Wurli-Wurlinjang Health Centre (Katherine)
06/05 - 10/05	Lajamanu
03/06 - 07/06	Katherine District Hospital and
10/06 - 14/06	Wurli-Wurlinjang Health Centre
12/08 - 16/08	Lajamanu
21/8/96	Pine Creek
16/09 - 20/09	Wurli-Wurlinjang Health Centre
29/09 - 30/09	Timber Creek
01/10	Kildurk
02/10	Bulla Camp
03/10	Yarralin
13/10	Mataranka
15/10	Duck Creek
16/10	Hodgson Downs
21/10 - 22/10	Bulman
23/10 - 25/10	Ngukurr
18/11 - 19/11	Mataranka
20/11 - 21/11	Borroloola
25/11 - 28/11	Borroloola
02/12 - 13/12	Wurli-Wurlinjang Health Centre

Table 4.2 Community visits of the Mobile Eye Unit 1996

4.5.5 Clinics at the Renal Unit

During follow-up visits at the Wurli - Wurlinjang Health center the MEU received information that some people had moved to Darwin, the Territory capital, to undergo dialysis. For the KRDRS it was very important to follow these patients even after they had left their community. In Darwin it was very difficult to fit in-patients into routine eye clinics when they are dialysed several times a week. After discussions with the Renal Unit it was agreed to carry out ophthalmologic reviews at the renal unit.

4.6 Data collection

4.6.1 General overview

The data collection form used in the study is shown in Appendix(1). The layout of the form reflects the patient encounter during the visits.
A great deal of care was taken to identify the patient. This was done by the nursing sister or, in Wurli-Wurlinjang Health Center, by the Aboriginal health workers. The patients were then seen by the optometrist, who carried out refraction after which the ophthalmologist took a medical history and carried out an ophthalmic examination. The ophthalmic examination was carried out in a tent provided by Norforce or in an examination room. Every attempt was made to optimize the conditions required for the ophthalmic examination and for obtaining retinal photographs. While the patient waited for the dilating drops to work, blood and urine samples were collected and other diabetes-associated variables were measured.

4.6.2 Personal section

Data on the personal section were of primary importance in identifying the Aboriginal patients. This could only be done by meticulous filling in of all the information requested in the personal section. Name, date of birth, sex, community seen in, community patient lived in, date of examination, Medicare number and ethnicity were recorded. Unfortunately some patients were recorded under different names but knowing these patients personally and using the above data for identification made it possible to avoid having the same subjects with different identifiers in the final analysis.

4.6.3 Medical history section

The medical records of the patients and all relevant past eye history (PEH) and past medical history (PMH) were recorded and reviewed. In some health centres (usually places where the same nursing staff was employed for years and in Wurli-Wurlinjang Health Centre) the medical records were easily manageable. In other health centres it was an arduous task to find the information required for the study. At the time of the study, diabetes was diagnosed on the basis of various criteria. In every case the records were re-examined and the existence of diabetes was identified strictly on the basis of the WHO criteria 1985. When a diagnosis of diabetes was founded on the basis of a random BSL of less than 11.2 mmol/l, the diagnosis was confirmed by the DMO. The method used for the diagnosis of diabetes was also recorded together with the test values. The diabetic medications actually taken by the patient, not necessarily those

prescribed, were recorded. The presence of hypertension was also recorded.

4.6.4 Ophthalmological examination

4.6.4.1 Visual acuity

Uncorrected and best corrected visual acuity was measured with the Snellen chart. Refraction was carried out by the "Flying Optometrist"[4] of the Northern Territory. If the optometrist could not make the trip, a list was generated of all those people whose uncorrected visual acuity could be improved with pinhole and therefore would benefit from glasses. The optometrist carried out the test within 10 days after the list had been created, and provided information to the MEU.

4.6.4.2 Intraocular pressure (IOP)

The IOP was measured with a Keeler Pulsair airpuff tonometer.

4.6.4.3 Anterior segment

The anterior segment was examined using a slitlamp. The upper and lower lids were everted only in cases when the patient had specific complaints suggesting that he/she might have upper lid pathology or when

[4] The "Flying Optometrist" was one of the optometrists providing regular services (prescribing glasses) to Aboriginal communities in the Northern Territory. The "flying" term refers to his unique mode of transportation.

the patient had obvious eyelid pathology and/or corneal changes suggestive of a previous or present eyelid pathology. The conjunctiva, cornea, iris, pupil, lens were also examined.

4.6.4.4 Posterior segment:

a. The anterior vitreous was examined with a slitlamp, the posterior vitreous with the help of Heine Omega 200 indirect ophthalmoscope

b. The retina was examined with the combination of direct and indirect methods (see chapter 3.2.2 - Screening for diabetic retinopathy in Aboriginal Australian communities). Mydriasis was achieved using Tropicamide 1% and Phenylephrine 10% eye drops (1 drop each 3 times, 10 minutes apart, and wait 10-15 minutes after the last set of drops).

   The retina was first examined with slitlamp biomicroscopy using a Volk 90 D lens with yellow coating or, in case of extreme difficulties, when the examination with the above method was impossible, with the traditional direct ophthalmoscope (through dilated pupil). These were used to examine primarily the disc, the macular and paramacular area.

   In every case the retinal examination above was supplemented with indirect ophthalmoscopy using a Heine Omega 200 indirect binocular ophthalmoscope to be able to judge the periphery.

   Fundi were also examined with a Kowa Genesis hand held fundus camera and retinal photographs were taken of the retinas. The following fields were examined: 1. disc 2. area nasal to the disc 3. macula 4. area temporal to the macula (these are the most likely

areas where diabetic lesions could start), however if lesions were detected on the periphery of the retina, every attempt was made to photograph them as well. These photographs were subsequently de-identified and the degree of retinal disease graded independently by a retinal specialist and the study ophthalmologist who had given the original grade in the field. In case of a disagreement between the two readers, the slides were read by a third examiner, a Senior Retinal Specialist, and his opinion was accepted. If the slide was ungradable, the original field grading was accepted.

This process provided a means to assess interobserver agreement and to determine if grading by retinal photograph alone was consistent with grading by direct observation.

4.6.5 Collection of biochemical data

Blood has a special significance in Aboriginal culture. Explanation was given to Aboriginal patients for the need of blood specimens and an opportunity was also given to the patient to refuse permission. Refusal was extremely rare.

Blood was collated for HbA1c and full lipid studies, urea and creatinine. The diabetics were requested to come to the clinic fasted, but it was impossible to monitor whether they did so. I believe that in the vast majority of cases they did not comply with this request since Aboriginal people eat whenever they are hungry. Clinics usually started at 8am, and we organized transport of patients so that diabetics would be seen first, but it was only a "four wheel drive full" of patients whom we managed to get as soon as they got out of bed. Lion (1993) on the basis of her experience suggested that the only way to ensure that patients are fasted

is to go to their dwellings as early as possible, wake them up and take blood samples at that time. This was not practicable in the KRDRS.

Urine samples were tested with a dipstick for sugar, acetone and protein. The specimens were sent to a laboratory for urinary microalbuminuria - creatinine ratio. In the case of a menstruating female, the patient was asked to return to the health center after the period stopped.

4.7 Treatment issues of diabetic retinopathy in Aboriginal communities of Katherine, Darwin and Gove regions of the Northern Territory

The Diabetic Retinopathy Study (DRS), The Early Treatment Diabetic Retinopathy Study (ETDRS), The Diabetic Vitrectomy Study (DVS) gave a solid basis for the treatment of diabetic retinopathy in the general community. Specific medical and cultural issues need to be taken into consideration when treating diabetic retinopathy in Aboriginal communities.

These include:

- the vast majority of diabetes in Aboriginal communities is type II diabetes.
- diabetes in Aboriginal communities is poorly controlled (Stanton, 1985; Taylor, 1997)
- the difficulties of following – up patients in Aboriginal communities
- the concept of requiring laser treatment when eye sight is still satisfactory is hard to accept for the Aboriginal patients (personal field experience)

- the technical difficulties of laser treatment in Aboriginal patients.

In light of the above, and after discussion with senior colleagues, the DRS and ETDRS treatment guidelines were modified for the KRDRS as shown in Table 4. These recommendations are in agreement with the guidelines in the recent OATSIH publication (OATSIH, 2001) with the exception that the KRDS, on the basis of clinical experience, also suggested the focal treatment of leaking exudates outside the CSME definition.

DRS and ETDRS	Aboriginal communities
for eyes with proliferative diabetic retinopathy deferral of panretinal photocoagulation until the high risk characteristics develop did not significantly change the incidence of severe visual loss at the conclusion of the study	at the time of our work it was not possible to review the patients for more than a year or two, therefore in case of proliferative diabetic retinopathy ***even without high risk characteristics*** **immediate photocoagulation was carried out**
for eyes with macular edema and less severe DR the most effective strategy in preventing severe visual loss was immediate focal with delayed scatter only when more severe diabetic retinopathy developed, thereby reducing the risk of moderate visual loss by 50% compared to treatment deferral	this principle was strictly adhered to
for eyes with macular edema and more severe retinopathy the most effective strategy was immediate focal combined with immediate mild scatter. It has to be noted, however, that in this group the prompt focal and delayed scatter option was not evaluated.	In Aboriginal patients after the macular edema (usually in both eyes) was treated, a *very mild scatter was carried out* ***during the same week*** *if severe BDR was present.* If this could not be done, we were striving to complete the treatment fairly soon (hopefully at their **6-10 weeks review**)
for eyes with clinically NOT significant macular edema there was no statistically significant difference in the presence of severe visual loss at 3 years between the immediate focal treatment and the deferral group	clinically *not* significant macula edema was not considered for treatment
for eyes with clinically significant macular edema the immediate focal treatment significantly reduced the presence of severe visual loss	in case of CSME only focal treatment was carried out as per the ETDRS
the risk of neovascular glaucoma was reduced by performing PRP, achieving complete retinal reattachment and retaining the lens	in patients requiring vitrectomy we carried out as much PRP as it was possible before referring them further for vitrectomy
leaking exudates are not offered for treatment in the ETDRS	**leaking exudates were treated** (focal treatment only) outside the CSME definition since they were capable of producing the most bizarre, large areas of retinal edema, sometimes threatening the macula as well later

Table 4.3 Modified DRS and ETDRS treatment principles for diabetic retinopathy in remote Aboriginal communities.

4.8 Data management and Statistical analysis

Data from the KRDRS data collection form were entered into a database created in Microsoft Access 97. After checking the individual entries in Access the database tables were converted to Stata (release 7) datasets using Stat/Transfer.

Standard statistical methods were used to describe diabetic retinopathy in the 1993 and 1996 study populations. P values and 95% confidence intervals around prevalences and risk ratios are derived from log binomial models incorporating corrections for non – independence of subjects and eyes due to clustering. These results are asymptotic and should be treated with caution in the case of sparse data.

The three-year cumulative incidence and an average "annualised" incidence of diabetic retinopathy are described in subjects and in eyes. Exact 95% confidence intervals are based on the Poisson distribution when incidence is calculated for *subjects* who developed the condition. When confidence intervals are calculated for the incidence in *eyes*, adjustment for clustering of eyes within subjects should be made. Unfortunately, this estimation, based on maximum likelihood models, is valid only asymptotically and the majority (94%) of the incidences reported in this thesis are based on small numerators (say, $n \leq 10$). Therefore, confidence intervals around cumulative rates for conditions in eyes are estimated with adjustment for clustering only when the numerator of the incidence is greater than ten, otherwise the exact method (as for the incidence in subjects) is used, without regard to clustering. It is acknowledged that such intervals will be narrower than is actually the case, and caution must be exercised in their interpretation.

4.9 Ethical issues

The process of gaining ethical approval for the project began by writing a discussion paper entitled "Proposal for the investigation/care of diabetic eye disorders". This draft proposal was widely circulated for consultation to all sections of health care professionals involved in the care of diabetics and eye disorders: DMOs, specialist physicians, nutritionists, ophthalmologists, medical administrators, epidemiologists, Aboriginal organisations, NT branch of Diabetes Australia. The Northern Territory Aboriginal Eye Health Committee became a strong advocate of the project. Comments were received from health professionals within the Northern Territory and from interstate experts.

After the initial concerns of those involved in the management and care of diabetics and eye diseases were addressed, the final proposal was submitted for approval to the Joint Institutional Ethics Committee (JIEC) of the Royal Darwin Hospital and Menzies School of Health Research. The author was invited to make a presentation to the Aboriginal Subcommittee of the JIEC, but was not present at the discussion or voting.
The project received approval from the Joint Institutional Ethics Committee of the Royal Darwin Hospital and Menzies School of Health Research. The approval was reviewed and extended annually.

Chapter 5

5 Description of the study population

5.1 General overview

Suitable definitions of subpopulations within the KRDRS permit a longitudinal follow-up of subjects over the period 1993 – 1996, and a comparison of diabetes related variables in Aboriginal communities at the beginning and at the end of the study.

Some of the variables are known risk factors for DR (eg.: time since diagnosis of diabetes), other factors, such as age at the time of diagnosis, living environment and distribution of diabetics in the region may not be risk factors, but nevertheless changes in their distributions may have an impact on planning for the long-term care of a diabetic Aboriginal population.

The study population will be described in three sections:

In section 2 the overall characteristics of the study population in 1993 and in 1996 are described.

In section 3 the characteristics of the core group, non-core 1993 and non-core 1996 group will be described.

In section 4 the characteristics of the two "newcomer" subgroups of the non-core 1996 group will be described.
While these subgroups have already been described in Chapter 4 section 2.2, for ease of reference the diagrams that represent the composition of the study population in 1993 and in 1996 are again presented in Figs 5.1 and 5.2.

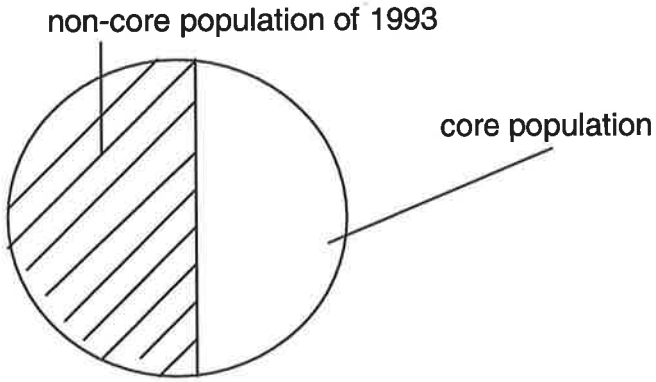

Figure 5.1 Subgroups of the study population in 1993.

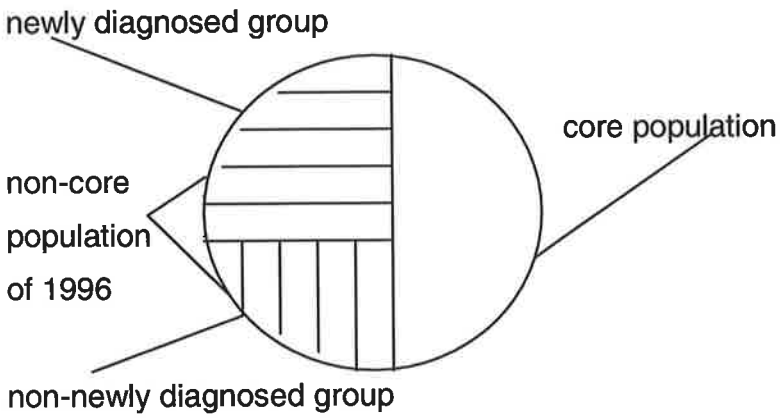

Figure 5.2 Subgroups of the study population in 1996.

5.2 Description of the diabetic population of Katherine region at the beginning and at the end of the study

There were 244 known diabetic Aboriginal patients in the Katherine region in 1993. Of these, eight subjects were on the diabetic list of the region, but seven were not residing in the region, and one person was "out bush"[1] at the time of the community visit. Two subjects refused permission to be examined. There were 23 subjects in whom the retina could not be examined in the right eye and 30 subjects where the retina could not be examined in the left eye, but there were only 21 subjects in whom neither the right nor the left retina could be seen. These 21 subjects were not included in the analysis investigating the prevalence and incidence of diabetic retinopathy in this population, but they are included in the description of the diabetic population of Katherine region. In summary, there were 234 diabetics in the study in 1993, 213 of whom had at least one retina examined.

In 1996, there were 272 known diabetic Aboriginal patients in the Katherine region. Of these, 24 subjects were on the diabetic list of the region, but before the visit three of them died from heart disease, one from kidney disease, one died from an unknown reason, one subject was in hospital during the study visit, the whereabouts of one diabetic was unknown to the community, and 17 patients still on the diabetic list of the region were not residing in the region at the time of the visit. Five subjects refused permission to be examined. There were five subjects in whom the retina could not be examined in the right eye

[1] This term is used to refer to the traditional activity of the Aboriginal patients away from the community (eg.: fishing and hunting); a sign of good health.

and seven subjects where the retina could not be examined in the left eye, but there were only four subjects in whom neither the right nor the left retina could be seen. These four subjects were not included in the analysis investigating the prevalence and incidence of diabetic retinopathy in this population, but they are included in the description of the diabetic population of the Katherine region. In summary, there were 243 diabetics in the study in 1996, 239 of whom had at least one retina examined.

At both the initial (1993) and final review (1996) of the study population there were five subjects with insulin dependent diabetes mellitus (IDDM), the rest were diagnosed as non-insulin dependent diabetics (NIDDM). In both years the subjects with IDDM were living in remote communities (in 1993, two in Beswick, one each in Duck Creek, Lajamanu and Ngukurr and in 1996, two in Bulman, one each in Beswick, Lajamanu and Ngukurr).

Table 5.2.1 shows the distribution of diabetics by settlement at the beginning and at the end of the study. The number of diabetics under the care of the Wurli-Wurlinjang Health Center represents the Aboriginal diabetics looked after by the health center from Kalano community, Katherine and various "town-camps"[2] in Katherine. While there was a moderate increase (4.3%) in the total number of diabetics in the region by 1996, in Borroloola the number of diabetics increased by 52%. Two-thirds of the diabetics were located in four major settlements at the beginning and at the end of the study (Borroloola, Kalkaringi, Lajamanu, Wurli-Wurlinjang). The increase in the number of diabetics in Borroloola and the distribution of diabetics in the population have implications for planning intervention and, in particular, prevention strategies.

[2] Town camp refers to the more or less permanent habitat of various Aboriginal groups in town. It also covers those present on transit to ceremonies or meetings.

Settlement-live	year of ophthalmological examination 1993	1996
Barunga	13 5.56	16 6.58
Beswick	7 2.99	7 2.88
Binjari	3 1.28	6 2.47
Borroloola	26 11.11	38 15.64
Bulla Camp	4 1.71	7 2.88
Bulman	6 2.56	9 3.70
Duck Creek	2 0.85	5 2.06
Hodgson Downs/River	8 3.42	6 2.47
Kalkaringi	21 8.97	21 8.64
Kildurk	3 1.28	3 1.23
Lajamanu	51 21.79	48 19.75
Mataranka	5 2.14	5 2.06
Ngukurr	19 8.12	12 4.94
Pine Creek	3 1.28	1 0.41
Timber Creek	7 2.99	5 2.06
Wurli-Wurlinjang	52 22.22	51 20.99
Yarralin	4 1.71	3 1.23
Total	234 100.00	243 100.00

Table 5.2.1 Distribution of diabetics by settlement in Katherine region in 1993 and 1996. Entries are frequencies and percentages.

Table 5.2.2 shows the distribution of sex and living environment in 1993 and 1996.

	year of ophthalmological examination and environment					
	1993			1996		
	traditional environment			traditional environment		
sex	no	yes	Total	no	yes	Total
female	12	152	164	21	136	157
	7.32	92.68	100.00	13.38	86.62	100.00
	60.00	71.03	70.09	55.26	66.34	64.61
male	8	62	70	17	69	86
	11.43	88.57	100.00	19.77	80.23	100.00
	40.00	28.97	29.91	44.74	33.66	35.39
Total	20	214	234	38	205	243
	8.55	91.45	100.00	15.64	84.36	100.00
	100.00	100.00	100.00	100.00	100.00	100.00

Table 5.2.2 Distribution of sex by living environment in 1993 and 1996. Entries are frequencies, row and column percentages.

Table 5.2.2 shows that in both years there were around twice as many females as males (female/male ratio = 2.3 in 1993, and 1.8 in 1996). It also shows that the overwhelming majority of the study population was living in a traditional environment at the initial (1993) and final (1996) visits in the communities (91% and 84% respectively).

Table 5.2.3 shows the distribution of age by sex and environment in 1993 and 1996. In 1996 one traditional male subject's age was not recorded.

		year of ophthalmological examination and environment					
		1993			1996		
		traditional environment			traditional environment		
	sex	no	yes	Total	no	yes	Total
female	freq	12	152	164	21	136	157
	mean	45.3	50.1	49.8	48.1	49.3	49.2
	SD	5.6	15.2	14.8	12.7	14.7	14.4
	min	39.0	13.0	13.0	21.0	23.0	21.0
	max	56.0	91.0	91.0	82.0	94.0	94.0
male	freq	8	62	70	17	68	85
	mean	50.0	51.6	51.4	48.8	51.6	51.0
	SD	8.3	11.5	11.1	11.5	13.7	13.3
	min	39.0	22.0	22.0	30.0	16.0	16.0
	max	62.0	78.0	78.0	67.0	81.0	81.0
Total	freq	20	214	234	38	204	242
	mean	47.2	50.6	50.3	48.4	50.1	49.8
	SD	7.0	14.2	13.8	12.0	14.4	14.0
	min	39.0	13.0	13.0	21.0	16.0	16.0
	max	62.0	91.0	91.0	82.0	94.0	94.0

Table 5.2.3 Distribution of age by and environment in 1993 and 1996.

In 1993 in the non-traditional population the mean age of the women was nearly five years less than that among men (difference in means = 4.7 years), in the traditional population this difference was 1.5 years. In the non-traditional population the mean age was more than three years less (difference in means 3.4 years) than in the traditional population. In the 1996 study population, the mean age was less in all environment/sex strata compared to the 1993 cross-sectional results except in non-traditional females, where the mean age increased from 45.3 to 48.1 years. There was a modest change in the mean age of the 1996 study population compared with the 1993 cross sectional results (decrease by 0.5 years). In the 1996 study population the mean age of women and men decreased compared to the 1993 cross sectional results (differences of means = 0.6 and 0.4 years). By

the end of the study, in the non-traditional population, the difference in mean age between males and females decreased from 4.7 years to 0.7 years and in the traditional population the difference in mean age between males and females increased from 1.5 years to 2.3 years. In the beginning and at the end of the study the mean age of the non-traditional population was less than in the traditional population, however, by the end of the study this difference decreased from 3.4 years in 1993 to 1.7 years in 1996. These changes may reflect either new younger diabetics presenting in 1996 or the non-attendance of older diabetics in 1996, or both. It is worth noting that that the age of females in all subsets of environment and year is younger than that of males, and that the age of non-traditional subjects in all subsets of environment and year is less than that of traditional subjects.

Table 5.2.4 shows the distribution of the study population by age at the time of examination and gender in 1993 and in 1996 cross sections. While the overall proportion of the study sample under 40 was 22% in 1993 compared with 26% in 1996, the distribution of age by gender was different in 1996. We found, that in 1993 26% of females were under 40 compared with 29% in 1996. In males however the proportion of subjects under 40 increased from 13% in 1993 to 20% in 1996. This finding has a major implication in emphasizing the need to incorporate diabetes prevention in the men's health program.

Table 5.2.5 shows the age at diagnosis of diabetes in the study population in 1993 and 1996. In 1993, data of two traditional males and five traditional females were not recorded; in 1996 data of three traditional males and three traditional females were not recorded.

age at the time of examination	year and sex					
	1993			1996		
	female	male	TOTAL	female	male	TOTAL
10 - 14	1		1			
	100.00	.	100.00	.	.	.
	0.61	.	0.43	.	.	.
15 - 19	1		1		1	1
	100.00	.	100.00	.	100.00	100.00
	0.61	.	0.43	.	1.18	0.41
20 - 24	5	1	6	5		5
	83.33	16.67	100.00	100.00	.	100.00
	3.05	1.43	2.56	3.18	.	2.07
25 - 29	5	1	6	10	2	12
	83.33	16.67	100.00	83.33	16.67	100.00
	3.05	1.43	2.56	6.37	2.35	4.96
30 - 34	16	2	18	11	7	18
	88.89	11.11	100.00	61.11	38.89	100.00
	9.76	2.86	7.69	7.01	8.24	7.44
35 - 39	15	5	20	19	7	26
	75.00	25.00	100.00	73.08	26.92	100.00
	9.15	7.14	8.55	12.10	8.24	10.74
40 - 44	20	11	31	16	11	27
	64.52	35.48	100.00	59.26	40.74	100.00
	12.20	15.71	13.25	10.19	12.94	11.16
45 - 49	20	12	32	21	12	33
	62.50	37.50	100.00	63.64	36.36	100.00
	12.20	17.14	13.68	13.38	14.12	13.64
50 - 54	12	10	22	21	12	33
	54.55	45.45	100.00	63.64	36.36	100.00
	7.32	14.29	9.40	13.38	14.12	13.64
55 - 59	23	10	33	13	6	19
	69.70	30.30	100.00	68.42	31.58	100.00
	14.02	14.29	14.10	8.28	7.06	7.85
60 - 64	23	12	35	15	11	26
	65.71	34.29	100.00	57.69	42.31	100.00
	14.02	17.14	14.96	9.55	12.94	10.74
65 - 69	6	2	8	15	10	25
	75.00	25.00	100.00	60.00	40.00	100.00
	3.66	2.86	3.42	9.55	11.76	10.33
70 - 74	10	3	13	4	3	7
	76.92	23.08	100.00	57.14	42.86	100.00
	6.10	4.29	5.56	2.55	3.53	2.89
75+	7	1	8	7	3	10
	87.50	12.50	100.00	70.00	30.00	100.00
	4.27	1.43	3.42	4.46	3.53	4.13
TOTAL	164	70	234	157	85	242
	70.09	29.91	100.00	64.88	35.12	100.00
	100.00	100.00	100.00	100.00	100.00	100.00

Table 5.2.4 Distribution of the study population by age at the time of examination and sex in 1993 and in 1996.

In the study population in 1993 and in 1996, diabetes was diagnosed at an earlier age in the non-traditional than in the traditional population (differences of means 3.2 and 2.5 years).

Table 5.2.5 also shows that in the study population of 1996 the age at diagnosis of diabetes decreased in each subgroup, except in non-traditional females, where it increased by 0.4 (from 41.8 to 42.2) years.

		year of ophthalmological examination and environment					
		1993			1996		
		traditional environment			traditional environment		
	sex	no	yes	Total	no	yes	Total
female	freq	12	147	159	21	133	154
	mean	41.8	46.0	45.7	42.2	44.1	43.9
	SD	5.0	15.0	14.5	12.8	14.3	14.1
	min	36.0	12.0	12.0	21.0	14.0	14.0
	max	52.0	90.0	90.0	81.0	90.0	90.0
male	freq	8	60	68	17	66	83
	mean	44.9	46.7	46.5	42.6	46.5	45.7
	SD	5.4	12.5	11.9	9.5	13.7	13.0
	min	34.0	14.0	14.0	26.0	15.0	15.0
	max	51.0	72.0	72.0	62.0	72.0	72.0
Total	freq	20	207	227	38	199	237
	mean	43.0	46.2	45.9	42.4	44.9	44.5
	SD	5.2	14.3	13.8	11.3	14.1	13.7
	min	34.0	12.0	12.0	21.0	14.0	14.0
	max	52.0	90.0	90.0	81.0	90.0	90.0

Table 5.2.5 Age at diagnosis of diabetes by sex and living environment in 1993 and 1996.

Table 5.2.6 shows the time since diagnosis of diabetes in the study population at the time of the ophthalmic examination. In 1993 there were no data for five traditional females and two traditional males; in 1996 there were no data for two traditional males and three traditional females. There is one more traditional male subject in this table than in table 5.2.5, since the time

since diagnosis of this subject is known, but the age at diagnosis (and the age at the time of examination) of this subject is unknown.

The striking feature of table 5.2.6 is the short time since diagnosis of diabetes at the time of the ophthalmological examination. One might expect an increase in the "time since diagnosis of diabetes" by 1996 just because of the cohort effect from subjects examined in both years. However, only 108 of the 243 subjects in 1996 were seen in both 1993 and 1996. The modest increase in the time since diagnosis suggests that in the "newcomers" of the 1996 subpopulation (non-core population, see chapters 4.2.2 and 5.4) the "time since diagnosis of diabetes" at the time of the ophthalmological examination is still short.

		year of ophthalmological examination and environment					
		1993			1996		
		traditional environment			traditional environment		
	sex	no	yes	Total	no	yes	Total
female	freq	12	147	159	21	133	154
	mean	3.5	3.7	3.7	6.0	5.3	5.4
	SD	3.0	3.5	3.4	5.0	4.2	4.3
	min	1.0	0.0	0.0	0.0	0.0	0.0
	max	9.0	26.0	26.0	21.0	29.0	29.0
male	freq	8	60	68	17	67	84
	mean	5.1	4.6	4.6	6.2	4.9	5.2
	SD	5.1	5.1	5.1	5.0	5.1	5.1
	min	0.0	0.0	0.0	0.0	0.0	0.0
	max	14.0	25.0	25.0	17.0	27.0	27.0
Total	freq	20	207	227	38	200	238
	mean	4.2	3.9	4.0	6.1	5.1	5.3
	SD	3.9	4.0	4.0	4.9	4.5	4.6
	min	0.0	0.0	0.0	0.0	0.0	0.0
	max	14.0	26.0	26.0	21.0	29.0	29.0

Table 5.2.6 Years since diagnosis of diabetes at the time of ophthalmic examination at the beginning (1993) and at the end (1996) of the study.

Tables 5.2.7 and 5.2.8 show the time since diagnosis by five year intervals at the beginning and at the end of the study.

Tables 5.2.7 and 5.2.8 show that in 1993, 60% of subjects (12/20) were diagnosed with diabetes 0 - 4 years before the examination in the non-traditional environment compared with 66% (149/207) of subjects in the traditional environment. The respective findings for 1996 are 50% (19/38) and 53% (106/200). In 1993 66% (149/227) of the study population was diagnosed 0 - 4 years prior to the examination compared with 53% (125/238) in 1996. The proportion of subjects diagnosed with diabetes 15 years or more prior to examination however was only 1.3% (3/227) in 1993 compared with 3.4% (8/238) in 1996.

time since diagnosis of diabetes	environment and sex --- non-traditional -- female	male	TOTAL	----- traditional ---- female	male	TOTAL	-------- TOTAL ------- female	male	TOTAL
non-core									
0 - 4	2	1	3	56	23	79	58	24	82
	66.67	33.33	100.00	70.89	29.11	100.00	70.73	29.27	100.00
	100.00	50.00	75.00	69.14	65.71	68.10	69.88	64.86	68.33
5 - 9	.	1	1	22	4	26	22	5	27
	.	100.00	100.00	84.62	15.38	100.00	81.48	18.52	100.00
	.	50.00	25.00	27.16	11.43	22.41	26.51	13.51	22.50
10 - 14	.	.	.	3	6	9	3	6	9
	.	.	.	33.33	66.67	100.00	33.33	66.67	100.00
	.	.	.	3.70	17.14	7.76	3.61	16.22	7.50
10 - 19	.	.	.	.	1	1	.	1	1
	.	.	.	.	100.00	100.00	.	100.00	100.00
	.	.	.	.	2.86	0.86	.	2.70	0.83
25 - 29	.	.	.	.	1	1	.	1	1
	.	.	.	.	100.00	100.00	.	100.00	100.00
	.	.	.	.	2.86	0.86	.	2.70	0.83
TOTAL	2	2	4	81	35	116	83	37	120
	50.00	50.00	100.00	69.83	30.17	100.00	69.17	30.83	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00
core									
0 - 4	6	3	9	41	17	58	47	20	67
	66.67	33.33	100.00	70.69	29.31	100.00	70.15	29.85	100.00
	60.00	50.00	56.25	62.12	68.00	63.74	61.84	64.52	62.62
5 - 9	4	1	5	21	6	27	25	7	32
	80.00	20.00	100.00	77.78	22.22	100.00	78.13	21.88	100.00
	40.00	16.67	31.25	31.82	24.00	29.67	32.89	22.58	29.91
10 - 14	.	2	2	3	2	5	3	4	7
	.	100.00	100.00	60.00	40.00	100.00	42.86	57.14	100.00
	.	33.33	12.50	4.55	8.00	5.49	3.95	12.90	6.54
25 - 29	.	.	.	1	.	1	1	.	1
	.	.	.	100.00	.	100.00	100.00	.	100.00
	.	.	.	1.52	.	1.10	1.32	.	0.93
TOTAL	10	6	16	66	25	91	76	31	107
	62.50	37.50	100.00	72.53	27.47	100.00	71.03	28.97	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 5.2.7 Distribution of time since diagnosis by sex and environment in 1993. Entries are frequencies, row and column percentages.

time since diagnosis of diabetes	environment and sex								
	--- non-traditional --			----- traditional ----			-------- TOTAL -------		
	female	male	TOTAL	female	male	TOTAL	female	male	TOTAL
non-core									
0 - 4	6	7	13	50	33	83	56	40	96
	46.15	53.85	100.00	60.24	39.76	100.00	58.33	41.67	100.00
	54.55	63.64	59.09	74.63	78.57	76.15	71.79	75.47	73.28
5 - 9	3	1	4	12	5	17	15	6	21
	75.00	25.00	100.00	70.59	29.41	100.00	71.43	28.57	100.00
	27.27	9.09	18.18	17.91	11.90	15.60	19.23	11.32	16.03
10 - 14	1	3	4	3	1	4	4	4	8
	25.00	75.00	100.00	75.00	25.00	100.00	50.00	50.00	100.00
	9.09	27.27	18.18	4.48	2.38	3.67	5.13	7.55	6.11
15 - 19				2	1	3	2	1	3
	.	.	.	66.67	33.33	100.00	66.67	33.33	100.00
	.	.	.	2.99	2.38	2.75	2.56	1.89	2.29
20 - 24	1		1		1	1	1	1	2
	100.00	.	100.00	.	100.00	100.00	50.00	50.00	100.00
	9.09	.	4.55	.	2.38	0.92	1.28	1.89	1.53
25 - 29					1	1		1	1
	.	.	.	.	100.00	100.00	.	100.00	100.00
	.	.	.	.	2.38	0.92	.	1.89	0.76
TOTAL	11	11	22	67	42	109	78	53	131
	50.00	50.00	100.00	61.47	38.53	100.00	59.54	40.46	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00
core									
0 - 4	4	2	6	16	7	23	20	9	29
	66.67	33.33	100.00	69.57	30.43	100.00	68.97	31.03	100.00
	40.00	33.33	37.50	24.24	28.00	25.27	26.32	29.03	27.10
5 - 9	4	2	6	39	13	52	43	15	58
	66.67	33.33	100.00	75.00	25.00	100.00	74.14	25.86	100.00
	40.00	33.33	37.50	59.09	52.00	57.14	56.58	48.39	54.21
10 - 14	2	1	3	10	5	15	12	6	18
	66.67	33.33	100.00	66.67	33.33	100.00	66.67	33.33	100.00
	20.00	16.67	18.75	15.15	20.00	16.48	15.79	19.35	16.82
15 - 19		1	1					1	1
	.	100.00	100.00	.	.	.	.	100.00	100.00
	.	16.67	6.25	.	.	.	.	3.23	0.93
25 - 29				1		1	1		1
	.	.	.	100.00	.	100.00	100.00	.	100.00
	.	.	.	1.52	.	1.10	1.32	.	0.93
TOTAL	10	6	16	66	25	91	76	31	107
	62.50	37.50	100.00	72.53	27.47	100.00	71.03	28.97	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 5.2.8 Distribution of time since diagnosis by sex and environment in 1996. Entries are frequencies, row and column percentages.

The diagnosis of diabetes is a major issue in Aboriginal communities and a crucial one for the study of diabetic retinopathy, since the duration of diabetes at the time of ophthalmological examination is a risk factor for the development of diabetic retinopathy.

Welborn in the Australian Diabetes Screening Study have measured the random blood sugar level (BSL) of 50859 subjects, and those who had a BSL above 5.5 mmol/l were referred for a glucose tolerance test (GTT) (Welborn, 1997). Welborn et al describe the distribution of random BSLs in those subjects who had been classified as diabetic, non-diabetic or had impaired glucose tolerance on the basis of a GTT. In this Australian population (drawn from all states, except the Australian Capital Territory and the Northern Territory), of all those subjects who were identified as diabetics, about 45% of subjects appeared with random BSL values in the range of 8.5-11.5 mmol/l and only about 14 % had a random blood sugar of 11.5 mmol/l or higher at the time of diagnosis. The mean of those who had random BSLs higher than 11.5 mmol/l random BSL was not reported.

Qiao et al in a Scandinavian population of 1008 persons (that included 18 known diabetics) studied the sensitivity of random BSL in the diagnosis of diabetes. They also described the distribution of random BSL values in those who were identified as diabetics, non-diabetics or having impaired glucose tolerance (IGT), as defined by the GTT. Among those who proved to be diabetics, the mean random BSL was 7.5 mmol/l in men and 6 mmol/l among women. From the graphs presented by these authors it can be seen that fewer than 20% of those diagnosed with diabetes had a random BSL higher than 11mmol/l in men and only one subject who was diagnosed with diabetes had a higher than 10mmol/l random BSL in women (Qiao, 1995).

In the light of these studies the random BSL values at diagnosis in Aboriginal communities are remarkable.

In 1993 there were data on 85 subjects, 83 of whom were diagnosed on the basis of random BSLs and 2 using the GTT. Of the 83 subjects there were complete data on 82. The subject with incomplete data was a female living in a traditional environment. Of the subjects with missing data there were 3 female and 4 male subjects living in a non-traditional environment and, 101 females and 41 males living in a traditional environment.

In 1996 data on 147 diabetics were recorded, 137 of whom were diagnosed on the basis of random BSLs and the remaining ten subjects were diagnosed with a GTT. There was a female subject living in a traditional environment with incomplete data, so of these 137 subjects there are complete data on 136 subjects. There were 11 males and nine females in the non-traditional environment and 24 male and 52 female subjects in the traditional environment (total 96 subjects) where the method of diagnosis and the test values were not recorded.

All 83 diabetics in 1993 and all but one of the 136 in 1996 had NIDDM.

As table 5.2.9 shows, the random BSL values were high in the Aboriginal population at the time of diagnosis. Hence, the majority of Aboriginal diabetics were diagnosed using a random blood sugar level only and only a small percentage of the diabetics presented with a random blood sugar value in the critical range which required a GTT to establish the diagnosis.
These findings raise the question: was there any bias in recording the method of diagnosis in the study subjects (are these figures representative of those whose

method of diagnosis was unknown)? This question is answered in part by results shown in tables 5.2.9 - 5.2.12.

		year of ophthalmological examination and environment					
		1993			1996		
		traditional environment			traditional environment		
	sex	no	yes	Total	no	yes	Total
female	freq	9	48	57	12	75	87
	mean	14.3	15.6	15.4	15.3	15.3	15.3
	SD	3.5	3.1	3.1	3.4	3.2	3.2
	min	11.2	9.9	9.9	11.2	9.3	9.3
	max	21.1	21.5	21.5	21.1	25.7	25.7
male	freq	4	21	25	6	43	49
	mean	18.5	15.0	15.6	16.4	15.3	15.5
	SD	7.7	3.7	4.6	6.9	3.8	4.2
	min	13.0	11.5	11.5	11.4	11.2	11.2
	max	30.0	25.9	30.0	30.0	25.9	30.0
Total	freq	13	69	82	18	118	136
	mean	15.6	15.4	15.5	15.6	15.3	15.4
	SD	5.2	3.3	3.6	4.7	3.4	3.6
	min	11.2	9.9	9.9	11.2	9.3	9.3
	max	30.0	25.9	30.0	30.0	25.9	30.0

Table 5.2.9 Random BSL values (mmol/l) at the time of diagnosis of diabetes by sex and environment in 1993 and 1996.

Tables 5.2.10 and 5.2.11 show the distribution of subjects by gender and environment in those subjects where the diagnosis was made by random BSL, compared with those where the method of diagnosis was not recorded. These tables show that in both 1993 and in 1996 the proportion of males and females was nearly the same in those subjects where the diagnosis was established by random BSL compared with those where the method of diagnosis was not recorded. The distribution of traditional and non-traditional living environments is different between the diagnostic groups in both 1993 and 1996, but it can be seen in Table 5.2.9 that the random BSL does not differ between the two environments.

method of diagnosis is known	environment and sex --- non-traditional -- female	male	TOTAL	----- traditional ---- female	male	TOTAL	-------- TOTAL female	male	TOTAL
no	3	4	7	101	41	142	104	45	149
	42.86	57.14	100.00	71.13	28.87	100.00	69.80	30.20	100.00
	25.00	50.00	35.00	67.79	66.13	67.30	64.60	64.29	64.50
yes	9	4	13	48	21	69	57	25	82
	69.23	30.77	100.00	69.57	30.43	100.00	69.51	30.49	100.00
	75.00	50.00	65.00	32.21	33.87	32.70	35.40	35.71	35.50
TOTAL	12	8	20	149	62	211	161	70	231
	60.00	40.00	100.00	70.62	29.38	100.00	69.70	30.30	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 5.2.10 Distribution of subjects in 1993 by sex and environment in the group where diabetes was diagnosed with random BSL compared to the group where the method of diagnosis was not recorded. Entries are frequencies, row and column percentages.

method of diagnosis is known	environment and sex --- non-traditional -- female	male	TOTAL	----- traditional ---- female	male	TOTAL	-------- TOTAL female	male	TOTAL
no	9	11	20	52	24	76	61	35	96
	45.00	55.00	100.00	68.42	31.58	100.00	63.54	36.46	100.00
	42.86	64.71	52.63	40.94	35.82	39.18	41.22	41.67	41.38
yes	12	6	18	75	43	118	87	49	136
	66.67	33.33	100.00	63.56	36.44	100.00	63.97	36.03	100.00
	57.14	35.29	47.37	59.06	64.18	60.82	58.78	58.33	58.62
TOTAL	21	17	38	127	67	194	148	84	232
	55.26	44.74	100.00	65.46	34.54	100.00	63.79	36.21	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 5.2.11 Distribution of subjects in 1993 by sex and environment in the group where diabetes was diagnosed with random BSL compared to the group where the method of diagnosis was not recorded. Entries are frequencies, row and column percentages.

Tables 5.2.12 and 5.2.13 show the distribution of age by gender and living environment in those subjects where the diagnosis of diabetes was made by random BSL compared with those where the method of diagnosis was not recorded.

Table 5.2.12 shows, that in 1993 the mean age of those whose diabetes was diagnosed by random BSL was 50.2 years compared with 50.6 years where the method of diagnosis of diabetes was not recorded. In 1993 the males were 3 years older (on average) in the group where the diagnosis was recorded, but the females in the same group were (on average) 1.8 year younger. Since the male/female ratio is nearly the same in both the "diagnosis recorded and diagnosed with random BSL" group and the "diagnosis not recorded" group, the different age distribution by sex in the sample is unlikely to cause bias if the results from this sample are applied to the 1993 study population.

In 1996 however, both females and males are older in the "diagnosis not recorded" group (table 5.2.13). It means that in case the event of detection bias (diagnosis was made well after clinical symptoms were present) the younger age in the "diagnosis recorded and diagnosed with random BSL" may bias the findings towards a lower BSL level if the results from the sample were to be applied to the entire study population and diabetes would have developed at the same age in both populations. Detection bias however is unlikely to be present in the study (see later).

method of diagnosis is known		environment and sex: non-traditional female	non-traditional male	non-traditional Total	traditional female	traditional male	traditional Total	Total female	Total male	Total Total
no	freq	3	4	7	101	41	142	104	45	149
	mean	45.7	50.3	48.3	50.8	50.3	50.7	50.7	50.3	50.6
	SD	6.4	9.7	8.2	15.7	11.7	14.6	15.5	11.5	14.4
	min	41.0	39.0	39.0	13.0	22.0	13.0	13.0	22.0	13.0
	max	53.0	62.0	62.0	83.0	73.0	83.0	83.0	73.0	83.0
yes	freq	9	4	13	48	21	69	57	25	82
	mean	45.1	49.8	46.5	49.6	54.0	50.9	48.9	53.3	50.2
	SD	5.8	8.2	6.6	14.2	10.9	13.4	13.3	10.5	12.6
	min	39.0	42.0	39.0	20.0	39.0	20.0	20.0	39.0	20.0
	max	56.0	61.0	61.0	91.0	78.0	91.0	91.0	78.0	91.0
Total	freq	12	8	20	149	62	211	161	70	231
	mean	45.3	50.0	47.2	50.4	51.6	50.8	50.0	51.4	50.4
	SD	5.6	8.3	7.0	15.2	11.5	14.2	14.7	11.1	13.7
	min	39.0	39.0	39.0	13.0	22.0	13.0	13.0	22.0	13.0
	max	56.0	62.0	62.0	91.0	78.0	91.0	91.0	78.0	91.0

Table 5.2.12 Distribution of age in 1993 by sex and environment in the group where diabetes was diagnosed with a random BSL compared to the group where the method of diagnosis was not recorded.

It is unlikely that these extreme values were found due to the lack of vigilance in diagnosis and these diabetics were found well after their diabetes had been present, since all health care professionals in the Northern Territory were so focused on the high prevalence of diabetes in the Aboriginal population that if any Aboriginal patient presented with any minor ailments, a random blood sugar was routinely measured. Burns reported that the 2100 people in Maningrida and its 24 surrounding outstations (located at the Western boundary of Arnheim Land in the Northern Territory) make 26500 presentations per year to Maningrida Health Center (Burns, 1998). Even though the presentations were not stratified by age, these figures show that there were ample occasions for opportunistic screening in the communities.

method of diagnosis is known		environment and sex --- non-traditional -- female	male	Total	----- traditional ---- female	male	Total	-------- Total ------- female	male	Total
no	freq	9	11	20	52	24	76	61	35	96
	mean	45.1	49.7	47.7	51.9	52.3	52.0	50.9	51.5	51.1
	SD	13.6	11.8	12.5	14.6	12.6	13.9	14.6	12.3	13.7
	min	21.0	32.0	21.0	24.0	25.0	24.0	21.0	25.0	21.0
	max	60.0	67.0	67.0	76.0	76.0	76.0	76.0	76.0	76.0
yes	freq	12	6	18	75	43	118	87	49	136
	mean	50.4	47.2	49.3	48.4	50.8	49.3	48.7	50.4	49.3
	SD	12.1	11.9	11.8	14.5	14.6	14.6	14.2	14.3	14.2
	min	34.0	30.0	30.0	23.0	16.0	16.0	23.0	16.0	16.0
	max	82.0	64.0	82.0	94.0	81.0	94.0	94.0	81.0	94.0
Total	freq	21	17	38	127	67	194	148	84	232
	mean	48.1	48.8	48.4	49.8	51.3	50.3	49.6	50.8	50.0
	SD	12.7	11.5	12.0	14.6	13.9	14.3	14.3	13.4	14.0
	min	21.0	30.0	21.0	23.0	16.0	16.0	21.0	16.0	16.0
	max	82.0	67.0	82.0	94.0	81.0	94.0	94.0	81.0	94.0

Table 5.2.13 Distribution of age in 1996 by sex and environment in the group where diabetes was diagnosed with random BSL compared to the group where the method of diagnosis was not recorded.

Bell suggested that the extremely high random BSL values may have been due to detection bias, since between 1990 and 1994 the opportunistic screening for diabetes was strongly encouraged by the District Medical Officers (DMOs) and therefore subjects who avoided detection previously may have been diagnosed with diabetes at a much later stage of their disease, hence the high random BSL values (Bell, 2002).

To determine whether this may be the case requires examination of the distribution of random BSL values at the time of diagnosis in subjects that were diagnosed prior to 1990, between 1990 – 1994 and after 1994. Table 5.2.14 shows that the start of an opportunistic screening program did not have an effect on the random BSL levels at diagnosis. The high random BSL at the time of diagnosis in the 1993 and 1996 study population raises the question of weather the health centers in the communities are in a position to diagnose diabetes at an early stage due to the mobility of the population; or the onset of type II diabetes in Aboriginal patients is "subacute" with high random blood sugar values already at the time of diagnosis.

diabetes diagnosed	random BSL -- 1993 -- yes	-- 1996 -- yes
before 1990		
freq	31	33
mean	15.7	16.1
SD	4.6	4.5
min	9.9	11.3
max	30.0	30.0
1990 - 1994		
freq	51	73
mean	15.3	14.8
SD	2.9	2.7
min	11.2	11.2
max	21.5	21.5
1995 - 1996		
freq	.	28
mean	.	15.8
SD	.	4.3
min	.	9.3
max	.	25.7

Table 5.2.14 Random blood sugar values in the "diagnosed by random BSL group" in 1993 and in 1996 by year of diagnosis. (Note the opportunistic screening campaign was encouraged after 1990).

It is well documented that time since diagnosis of diabetes is a risk factor for the development of the diabetic retinopathy. Therefore, it is important to examine the proportion of diabetics diagnosed before the age of 40 in the 1993 and in the 1996 study population. These subjects, due to improved medical services will most likely be exposed to the adverse pathological processes of diabetes for longer during their lives and therefore, more likely to develop diabetic retinopathy.

Table 5.2.15 shows that the proportion of subjects diagnosed under the age of 40, after the emphasis on opportunistic screening program, increased in the 1996 study population. This has an impact on future service needs (see discussion).

age at diagnosis	year 1993	1996
< 40	41 31.78	62 37.58
≥ 40	88 68.22	103 62.42
TOTAL	129 100.00	165 100.00

Table 5.2.15 Age at the time of diagnosis in 1993 and 1996 in those diagnosed with diabetes since 1990 (introduction of opportunistic screening program in Katherine region).Entries are frequencies and percentages.

5.3 Description of the "core" population and "non-core" populations of the study

The "core population" denotes a cohort of Aboriginal diabetics who were present in the Katherine region in 1993 and in 1996 and in whom at least one of the retinas could be examined on both occasions.

The "non-core population" of 1993 denotes Aboriginal diabetics who were present in Katherine region in 1993 and at least one of the retinas could be examined but were lost to follow-up by the end of the study.

The "non-core population" of 1996 denotes Aboriginal diabetics who were not seen in Katherine region in 1993 but were seen in 1996 and at least one of the retinas could be examined.

It is important to describe the core population separately, since it represents the subpopulation which gives information about the progression of diabetic eye disease in Katherine region. The description of the non-core population in 1993 allows us to identify the characteristics of the subpopulation that was lost to follow-up, and the description of the non-core population of 1996 shows the characteristics of the newcomers in the study.

Of the total diabetic study population of 234 in 1993 and 243 in 1996, there were 118 subjects who were reviewed in both years. Of these, in nine subjects neither retina could be examined in 1993. (Both retinas of six of

these patients, and one of the retinas of one of the patients could be examined in 1996. There were two patients in whom neither retina could be seen in 1996.) There was one subject who could be examined in 1993 but neither of her retinas could be seen in 1996. Therefore, there were 108 diabetic subjects in whom at least one of the retinas could be examined in 1993 and in 1996.

Table 5.3.1 shows the distribution of the core population by settlement in 1993 and in 1996. The distribution of the core population by settlement is fairly similar to the distribution of the entire study population (see table 5.2.1).

Tables 5.3.2 and 5.3.3 show the distribution of sex and living environment in the core and non-core population in 1993 and in 1996. They show that in 1993 the female/male ratio was 2.2 in the non-core and 2.5 in the core populations, respectively. The respective findings for 1996 are 1.5 and 2.5.

Table 5.3.4 shows the distribution of age in the core and the non-core population in 1993.

Table 5.3.5 shows the distribution of age in the core and the non-core population in 1996. The date of birth of one male non-core subject living in a traditional environment was not recorded in the 1996 study population.

Table 5.3.4 shows that in 1993 the non-core population was older than the core population (difference in means = 2.3 years). Those females who were lost to follow up were older than those who stayed in the study (difference in means = 5.8 years). Among males, those who were lost to follow-up were younger than those who stayed in the study (differences of

means = 5.9 years). This distribution may reflect the mortality among women and an increased mobility among men (see discussion).

Settlement	year of ophthalmological examination 1993	1996
Barunga	5 4.63	4 3.70
Beswick	3 2.78	3 2.78
Binjari	3 2.78	3 2.78
Borroloola	14 12.96	14 12.96
Bulla Camp	3 2.78	3 2.78
Bulman	3 2.78	3 2.78
Duck Creek	1 0.93	1 0.93
Hodgson Downs/River	2 1.85	2 1.85
Kalkaringi	10 9.26	10 9.26
Kildurk	2 1.85	2 1.85
Lajamanu	22 20.37	22 20.37
Mataranka	3 2.78	3 2.78
Ngukurr	7 6.48	7 6.48
Pine Creek	1 0.93	1 0.93
Timber Creek	3 2.78	3 2.78
Wurli-Wurlinjang	25 23.15	26 24.07
Yarralin	1 0.93	1 0.93
Total	108 100.00	108 100.00

Table 5.3.1 Distribution of the core population in 1993 and in 1996. Entries are frequencies and percentages.

sex	core subjects and environment: non-core non-traditional	non-core traditional	non-core TOTAL	core non-traditional	core traditional	core TOTAL	TOTAL non-traditional	TOTAL traditional	TOTAL TOTAL
female	2	85	87	10	67	77	12	152	164
	2.30	97.70	100.00	12.99	87.01	100.00	7.32	92.68	100.00
	50.00	69.67	69.05	62.50	72.83	71.30	60.00	71.03	70.09
male	2	37	39	6	25	31	8	62	70
	5.13	94.87	100.00	19.35	80.65	100.00	11.43	88.57	100.00
	50.00	30.33	30.95	37.50	27.17	28.70	40.00	28.97	29.91
TOTAL	4	122	126	16	92	108	20	214	234
	3.17	96.83	100.00	14.81	85.19	100.00	8.55	91.45	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 5.3.2 Distribution of sex by living environment in the core and non-core population in 1993. Entries are frequencies, row and column percentages.

sex	core subjects and environment: non-core non-traditional	non-core traditional	non-core TOTAL	core non-traditional	core traditional	core TOTAL	TOTAL non-traditional	TOTAL traditional	TOTAL TOTAL
female	11	69	80	10	67	77	21	136	157
	13.75	86.25	100.00	12.99	87.01	100.00	13.38	86.62	100.00
	50.00	61.06	59.26	62.50	72.83	71.30	55.26	66.34	64.61
male	11	44	55	6	25	31	17	69	86
	20.00	80.00	100.00	19.35	80.65	100.00	19.77	80.23	100.00
	50.00	38.94	40.74	37.50	27.17	28.70	44.74	33.66	35.39
TOTAL	22	113	135	16	92	108	38	205	243
	16.30	83.70	100.00	14.81	85.19	100.00	15.64	84.36	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 5.3.3 Distribution of sex by living environment in the core and non-core population in 1996. Entries are frequencies, row and column percentages.

		core subjects and environment								
		------- non-core ------			--------- core --------			-------- TOTAL --------		
	sex	non-traditional	traditional	TOTAL	non-traditional		TOTAL	non-traditional	traditional	TOTAL
female	freq	2	85	87	10	67	77	12	152	164
	mean	42.5	52.7	52.5	45.8	46.9	46.7	45.3	50.1	49.8
	SD	0.7	15.7	15.6	6.1	14.1	13.3	5.6	15.2	14.8
	min	42.0	13.0	13.0	39.0	20.0	20.0	39.0	13.0	13.0
	max	43.0	83.0	83.0	56.0	91.0	91.0	56.0	91.0	91.0
male	freq	2	37	39	6	25	31	8	62	70
	mean	47.5	48.9	48.8	50.8	55.6	54.7	50.0	51.6	51.4
	SD	7.8	11.4	11.2	9.0	10.6	10.3	8.3	11.5	11.1
	min	42.0	22.0	22.0	39.0	37.0	37.0	39.0	22.0	22.0
	max	53.0	73.0	73.0	62.0	78.0	78.0	62.0	78.0	78.0
Total	freq	4	122	126	16	92	108	20	214	234
	mean	45.0	51.5	51.3	47.7	49.3	49.0	47.2	50.6	50.3
	SD	5.4	14.6	14.4	7.4	13.7	13.0	7.0	14.2	13.8
	min	42.0	13.0	13.0	39.0	20.0	20.0	39.0	13.0	13.0
	max	53.0	83.0	83.0	62.0	91.0	91.0	62.0	91.0	91.0

Table 5.3.4 Distribution of age by sex and environment in the core and non-core population in 1993.

		core subjects and environment								
		------- non-core ------			--------- core --------			-------- TOTAL --------		
	sex	non-traditional	traditional	TOTAL	non-traditional		TOTAL	non-traditional	traditional	TOTAL
female	freq	11	69	80	10	67	77	21	136	157
	mean	47.5	48.8	48.7	48.8	49.9	49.7	48.1	49.3	49.2
	SD	17.0	15.3	15.5	6.1	14.1	13.3	12.7	14.7	14.4
	min	21.0	24.0	21.0	42.0	23.0	23.0	21.0	23.0	21.0
	max	82.0	80.0	82.0	59.0	94.0	94.0	82.0	94.0	94.0
male	freq	11	43	54	6	25	31	17	68	85
	mean	46.1	47.5	47.2	53.8	58.6	57.7	48.8	51.6	51.0
	SD	12.2	13.8	13.4	9.0	10.6	10.3	11.5	13.7	13.3
	min	30.0	16.0	16.0	42.0	40.0	40.0	30.0	16.0	16.0
	max	67.0	76.0	76.0	65.0	81.0	81.0	67.0	81.0	81.0
Total	freq	22	112	134	16	92	108	38	204	242
	mean	46.8	48.3	48.1	50.7	52.2	52.0	48.4	50.1	49.8
	SD	14.5	14.7	14.6	7.4	13.7	13.0	12.0	14.4	14.0
	min	21.0	16.0	16.0	42.0	23.0	23.0	21.0	16.0	16.0
	max	82.0	80.0	82.0	65.0	94.0	94.0	82.0	94.0	94.0

Table 5.3.5 Distribution of age by sex and environment in the core and non-core population in 1996.

One might think that the lower mean age of diabetics in the study population of 1996 is simply an artefact of better disease surveillance in

the communities. However, the surveillance system did not change in the communities between 1990, when policies for DM surveillance were first formulated, and 1996. Tables 5.3.4 and 5.3.5 show that the mean age of the "newcomers" (non-core population of 1996) in 1996 was 3.9 years younger than those who stayed in the study until 1996 (core population in 1996) and 2.2 years younger than the total population in 1993. Therefore, the "newcomers" (non-core population of 1996) represent a younger group in the study.

This finding necessitates the comparison of age at diagnosis between groups, since diagnosis at an earlier age may suggest either more intensive surveillance for the disease or a change in the natural history of diabetes in Aboriginal communities.

Table 5.3.6 shows the distribution of age at diagnosis in the core and non-core population in 1993. There were seven subjects living in a traditional environment (two non-core males, four non-core females and one core female) whose age at diagnosis was not recorded.
Table 5.3.7 shows the distribution of age at diagnosis in the core and non-core population in 1996. There were six subjects living in a traditional environment (three non-core males, two non-core females and one core female) whose age at diagnosis was not recorded.

These tables show that the non-core women in 1993 were diagnosed with diabetes at a much later age than core women (difference in means = 5.8 years); by the end of the study this difference was reduced by more than half (difference in means = 2.3 years). In the male population the mean age at diagnosis of diabetes was less in the non-core than in the core population in both years (difference in means = 7.8 years in 1993 and 8

years in 1996). The striking features of the tables are that the mean age at diagnosis of the non-core diabetics in 1996 was 3.5 years less among women and 0.2 years less among men than in the non-core population in 1993. The non-core population in 1996 was diagnosed with diabetes 0.9 years earlier than the core population of the study, 2.7 years earlier than the non-core population in 1993, and 1.9 years earlier than the entire study population of 1993.

These data show that in the newcomers (non-core population of 1996) diabetes was diagnosed at a younger age. Since there has been no change in surveillance for the disease, these data suggest that the earlier appearance of diabetes is due to the changing natural history of diabetes in Aboriginal communities. Due to improved medical services these diabetics may live longer and since time from diagnosis is a known risk factor for diabetic retinopathy an increase in prevalence of diabetic retinopathy in this newly diagnosed population is likely (see discussion).

		core subjects and environment								
		------- non-core ------			--------- core --------			-------- TOTAL --------		
	sex	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL
female	freq	2	81	83	10	66	76	12	147	159
	mean	40.5	48.7	48.5	42.1	42.8	42.7	41.8	46.0	45.7
	SD	0.7	15.5	15.3	5.5	13.8	13.0	5.0	15.0	14.5
	min	40.0	12.0	12.0	36.0	14.0	14.0	36.0	12.0	12.0
	max	41.0	79.0	79.0	52.0	90.0	90.0	52.0	90.0	90.0
male	freq	2	35	37	6	25	31	8	60	68
	mean	43.5	42.9	42.9	45.3	52.0	50.7	44.9	46.7	46.5
	SD	2.1	12.6	12.2	6.2	10.6	10.1	5.4	12.5	11.9
	min	42.0	14.0	14.0	34.0	34.0	34.0	34.0	14.0	14.0
	max	45.0	62.0	62.0	51.0	72.0	72.0	51.0	72.0	72.0
Total	freq	4	116	120	16	91	107	20	207	227
	mean	42.0	46.9	46.8	43.3	45.3	45.0	43.0	46.2	45.9
	SD	2.2	14.8	14.6	5.8	13.6	12.7	5.2	14.3	13.8
	min	40.0	12.0	12.0	34.0	14.0	14.0	34.0	12.0	12.0
	max	45.0	79.0	79.0	52.0	90.0	90.0	52.0	90.0	90.0

Table 5.3.6 Distribution of age at diagnosis by sex and environment in the core and non-core population in 1993.

		core subjects and environment								
		------- non-core ------			--------- core --------			-------- TOTAL --------		
	sex	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL
female	freq	11	67	78	10	66	76	21	133	154
	mean	42.3	45.5	45.0	42.1	42.8	42.7	42.2	44.1	43.9
	SD	17.4	14.8	15.1	5.5	13.8	13.0	12.8	14.3	14.1
	min	21.0	16.0	16.0	36.0	14.0	14.0	21.0	14.0	14.0
	max	81.0	76.0	81.0	52.0	90.0	90.0	81.0	90.0	90.0
male	freq	11	41	52	6	25	31	17	66	83
	mean	41.2	43.1	42.7	45.3	52.0	50.7	42.6	46.5	45.7
	SD	10.8	14.4	13.7	6.2	10.6	10.1	9.5	13.7	13.0
	min	26.0	15.0	15.0	34.0	34.0	34.0	26.0	15.0	15.0
	max	62.0	67.0	67.0	51.0	72.0	72.0	62.0	72.0	72.0
Total	freq	22	108	130	16	91	107	38	199	237
	mean	41.7	44.6	44.1	43.3	45.3	45.0	42.4	44.9	44.5
	SD	14.1	14.6	14.5	5.8	13.6	12.7	11.3	14.1	13.7
	min	21.0	15.0	15.0	34.0	14.0	14.0	21.0	14.0	14.0
	max	81.0	76.0	81.0	52.0	90.0	90.0	81.0	90.0	90.0

Table 5.3.7 Distribution of age at diagnosis by sex and environment in the core and non-core population in 1996.

Table 5.3.8 shows the time since diagnosis of diabetes at the time of ophthalmological examination in 1993 by sex and environment in the core and non – core populations. In two non-core males, four non-core females, one core female, all living in a traditional environment, the time since diagnosis was not recorded.

Table 5.3.9 shows the time since diagnosis of diabetes at the time of ophthalmological examination in 1996 by sex and environment in the core and non – core populations. In two non-core males, two non-core females, one core female, all living in a traditional environment, the time since diagnosis was not recorded.

Tables 5.3.8 and 5.3.9 show that in 1993 the mean time since diagnosis of the core and non-core population is almost identical (differences of means = 0.1 years, shorter in the non-core subjects), by 1996 the difference is considerably increased (difference in means = 3.1 years). However, the mean time since diagnosis in the non-core population is identical in 1993 and 1996. Therefore, the increase in the means in the years since diagnosis from 4 years in 1993 to 5.3 years in 1996 is attributable to the ageing of the core population.

		core subjects and environment								
		non-core			core			TOTAL		
	sex	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL
female	freq	2	81	83	10	66	76	12	147	159
	mean	2.0	3.4	3.4	3.8	4.0	4.0	3.5	3.7	3.7
	SD	1.4	3.0	3.0	3.2	3.9	3.8	3.0	3.5	3.4
	min	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
	max	3.0	14.0	14.0	9.0	26.0	26.0	9.0	26.0	26.0
male	freq	2	35	37	6	25	31	8	60	68
	mean	4.0	5.2	5.1	5.5	3.6	4.0	5.1	4.6	4.6
	SD	5.7	6.0	5.9	5.5	3.4	3.8	5.1	5.1	5.1
	min	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	max	8.0	25.0	25.0	14.0	11.0	14.0	14.0	25.0	25.0
Total	freq	4	116	120	16	91	107	20	207	227
	mean	3.0	4.0	3.9	4.4	3.9	4.0	4.2	3.9	4.0
	SD	3.6	4.2	4.2	4.1	3.8	3.8	3.9	4.0	4.0
	min	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	max	8.0	25.0	25.0	14.0	26.0	26.0	14.0	26.0	26.0

Table 5.3.8 Distribution of time since diagnosis at the time of examination in 1993 by sex and environment in the core and non-core population.

		core subjects and environment								
		non-core			core			TOTAL		
	sex	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL
female	freq	11	67	78	10	66	76	21	133	154
	mean	5.3	3.5	3.7	6.7	7.0	7.0	6.0	5.3	5.4
	SD	6.4	3.8	4.2	3.1	3.9	3.8	5.0	4.2	4.3
	min	0.0	0.0	0.0	4.0	3.0	3.0	0.0	0.0	0.0
	max	21.0	17.0	21.0	12.0	29.0	29.0	21.0	29.0	29.0
male	freq	11	42	53	6	25	31	17	67	84
	mean	4.9	3.9	4.1	8.5	6.6	7.0	6.2	4.9	5.2
	SD	4.5	5.7	5.5	5.5	3.4	3.8	5.0	5.1	5.1
	min	0.0	0.0	0.0	3.0	3.0	3.0	0.0	0.0	0.0
	max	13.0	27.0	27.0	17.0	14.0	17.0	17.0	27.0	27.0
Total	freq	22	109	131	16	91	107	38	200	238
	mean	5.1	3.7	3.9	7.4	6.9	7.0	6.1	5.1	5.3
	SD	5.4	4.6	4.7	4.1	3.8	3.8	4.9	4.5	4.6
	min	0.0	0.0	0.0	3.0	3.0	3.0	0.0	0.0	0.0
	max	21.0	27.0	27.0	17.0	29.0	29.0	21.0	29.0	29.0

Table 5.3.9 Distribution of time since diagnosis at the time of examination in 1996 by sex and environment in the core and non-core population.

Tables 5.3.10 and 5.3.11 show the mean blood sugar levels at the time of diagnosis in 1993 and in 1996. In 1993 in the non-core population one non-traditional male's, one non-traditional female's, 73 traditional females' and 33 traditional males' data (total 108 subjects) were not recorded. In the core population two non-traditional females', three non-traditional males', 28 traditional females' and eight traditional males' data (total 41 subjects) were not recorded. In 1996 in the non-core population data of seven non-traditional females, 24 traditional females, 8 non-traditional males, and 16 traditional males (total 55 subjects) were not recorded. In the core population 3 non-traditional and 8 traditional males', 2 non-traditional and 28 traditional females' data (total 96 subjects' data) were not recorded.

The distribution of random BSL at the time of diagnosis of diabetes in 1996 was very similar to that in 1993.

		core population and environment								
		core population								
		no			yes			Total		
		traditional environment			traditional environment			traditional environment		
	sex	no	yes	Total	no	yes	Total	no	yes	Total
female	freq	1	12	13	8	36	44	9	48	57
	mean	12.2	15.4	15.2	14.6	15.7	15.5	14.3	15.6	15.4
	SD		3.5	3.5	3.6	3.0	3.1	3.5	3.1	3.1
	min	12.2	9.9	9.9	11.2	11.3	11.2	11.2	9.9	9.9
	max	12.2	20.6	20.6	21.1	21.5	21.5	21.1	21.5	21.5
male	freq	1	4	5	3	17	20	4	21	25
	mean	15.3	14.5	14.7	19.6	15.1	15.8	18.5	15.0	15.6
	SD		2.6	2.3	9.1	4.0	5.0	7.7	3.7	4.6
	min	15.3	11.6	11.6	13.0	11.5	11.5	13.0	11.5	11.5
	max	15.3	17.2	17.2	30.0	25.9	30.0	30.0	25.9	30.0
Total	freq	2	16	18	11	53	64	13	69	82
	mean	13.8	15.2	15.0	16.0	15.5	15.6	15.6	15.4	15.5
	SD	2.2	3.3	3.1	5.6	3.3	3.7	5.2	3.3	3.6
	min	12.2	9.9	9.9	11.2	11.3	11.2	11.2	9.9	9.9
	max	15.3	20.6	20.6	30.0	25.9	30.0	30.0	25.9	30.0

Table 5.3.10 Distribution of random blood sugar at the time of diagnosis by gender and environment in the core and non-core populations of 1993.

		core population and environment								
		core population								
		no			yes			Total		
		traditional environment			traditional environment			traditional environment		
	sex	no	yes	Total	no	yes	Total	no	yes	Total
female	freq	4	39	43	8	36	44	12	75	87
	mean	16.5	15.0	15.1	14.6	15.7	15.5	15.3	15.3	15.3
	SD	2.9	3.3	3.3	3.6	3.0	3.1	3.4	3.2	3.2
	min	14.7	9.3	9.3	11.2	11.3	11.2	11.2	9.3	9.3
	max	20.9	25.7	25.7	21.1	21.5	21.5	21.1	25.7	25.7
male	freq	3	26	29	3	17	20	6	43	49
	mean	13.1	15.5	15.3	19.6	15.1	15.8	16.4	15.3	15.5
	SD	2.2	3.8	3.7	9.1	4.0	5.0	6.9	3.8	4.2
	min	11.4	11.2	11.2	13.0	11.5	11.5	11.4	11.2	11.2
	max	15.5	24.3	24.3	30.0	25.9	30.0	30.0	25.9	30.0
Total	freq	7	65	72	11	53	64	18	118	136
	mean	15.1	15.2	15.2	16.0	15.5	15.6	15.6	15.3	15.4
	SD	3.0	3.5	3.4	5.6	3.3	3.7	4.7	3.4	3.6
	min	11.4	9.3	9.3	11.2	11.3	11.2	11.2	9.3	9.3
	max	20.9	25.7	25.7	30.0	25.9	30.0	30.0	25.9	30.0

Table 5.3.11 Distribution of random blood sugar at the time of diagnosis by sex and environment in the core and non-core populations of 1996.

5.4 Detailed analysis of the non-core population of 1996

The description of various subgroups in Chapter 5 sections 2 and 3 allows identification of the characteristics of:

- the study population at the beginning and at the end of the study
- the core population.

The non-core population of 1996 is, however, not a homogeneous group with respect to diabetes related variables such as age at the time of examination, age at the time of diagnosis or time since diagnosis. It was shown in Chapter 5, section 3, that description of these variables is necessary to estimate the future burden of diabetic eye disorders in Aboriginal communities in Katherine region.

In order to describe the characteristics of those subjects diagnosed with diabetes between 1994 and 1996, the non-core group of 1996 was divided into the following groups:

- "newly diagnosed diabetics" which consisted of 75 subjects in the non-core population of 1996 who were diagnosed with diabetes between 1994 and 1996
- "non-newly diagnosed diabetics" consisted of 60 subjects in the non-core population of 1996 who were diagnosed with diabetes prior to 1994 (Fig 5.2).

There were 135 subjects in the non-core population of 1996 of whom 75 were diagnosed with diabetes between 1993 and 1996 (25 subjects were diagnosed in 1994, 23 subjects in 1995, 23 subjects in 1996). In the non-core population of 1996 the time of diagnosis is missing in the data from two traditional males and two traditional females.

Table 5.4.1 shows the distribution of the newly diagnosed diabetics.

Settlement-live	Freq.	Percent	Cum.
Barunga	9	12.00	12.00
Beswick	1	1.33	13.33
Binjari	3	4.00	17.33
Borroloola	14	18.67	36.00
Bulla Camp	2	2.67	38.67
Bulman	4	5.33	44.00
Duck Creek	1	1.33	45.33
Hodgson Downs/River	4	5.33	50.67
Kalkaringi	5	6.67	57.33
Kildurk	1	1.33	58.67
Lajamanu	15	20.00	78.67
Mataranka	1	1.33	80.00
Ngukurr	3	4.00	84.00
Timber Creek	1	1.33	85.33
Wurli-Wurlinjang	10	13.33	98.67
Yarralin	1	1.33	100.00
Total	75	100.00	

Table 5.4.1 Geographical distribution of the newly diagnosed diabetics (1994-1996).

While the distribution of the core population was fairly similar to the distribution of the study subjects in the beginning and at the end of the study (tables 5.2.1 and 5.3.1), there were communities (Barunga and Borroloola) where the proportion of newly diagnosed diabetics was considerably higher (12% and 19%, table 5.4.1) than expected on the basis of the proportion of diabetics in these two communities in 1993 (5.6% and 11.1%) and 1996 (6.6% and 15.6%) (table 5.2.1).
Table 5.4.2 shows the distribution of sex and living environment in the non-core population of 1996.

Table 5.4.2 reveals that the majority of the newly diagnosed diabetics (87%) come from a traditional environment. There is however a change in the distribution of sex in this population. While the total study population of 1993 and 1996 consisted predominantly of females (70% and 65% -

see table 5.2.2) table 5.4.2 shows that the proportion of males substantially increased in the subgroup of newly diagnosed diabetics in the 1996 non-core population (42.7%).

A change of similar magnitude can be seen if the diabetics diagnosed between 1994 and 1996 are compared to all those diabetics in the study who were diagnosed prior to 1994 (from 32% to 43%) (table 5.4.3).

sex	time of diagnosis of diabetes and environment - diabetes diagnosed - before 1994 non-traditional	traditional	TOTAL	- diabetes diagnosed - 1994 - 1996 non-traditional	traditional	TOTAL	-------- TOTAL -------- non-traditional	traditional	TOTAL
female	5	32	37	6	37	43	11	69	80
	13.51	86.49	100.00	13.95	86.05	100.00	13.75	86.25	100.00
	41.67	66.67	61.67	60.00	56.92	57.33	50.00	61.06	59.26
male	7	16	23	4	28	32	11	44	55
	30.43	69.57	100.00	12.50	87.50	100.00	20.00	80.00	100.00
	58.33	33.33	38.33	40.00	43.08	42.67	50.00	38.94	40.74
TOTAL	12	48	60	10	65	75	22	113	135
	20.00	80.00	100.00	13.33	86.67	100.00	16.30	83.70	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 5.4.2 Distribution of gender and living environment in the non-core population of 1996. Entries are frequencies, row and column percentages.

sex	traditional environment no	yes	Total
female	17	184	201
	8.46	91.54	100.00
	53.13	70.23	68.37
male	15	78	93
	16.13	83.87	100.00
	46.88	29.77	31.63
Total	32	262	294
	10.88	89.12	100.00
	100.00	100.00	100.00

Table 5.4.3 Distribution of sex and living environment in the diabetic population of the study if their diabetes was diagnosed before 1994. Entries are frequencies, row and column percentages.

Table 5.4.4 shows the distribution of age in the non-core population of 1996. In the newly diagnosed group the age of one traditional male subject was not recorded.

		time of diagnosis of diabetes and environment								
		- diabetes diagnosed before 1994 -			- diabetes diagnosed 1994 - 1996 -			-------- TOTAL --------		
	sex	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL
female	freq	5	32	37	6	37	43	11	69	80
	mean	49.4	52.8	52.4	46.0	45.4	45.5	47.5	48.8	48.7
	SD	9.8	14.3	13.8	22.3	15.5	16.3	17.0	15.3	15.5
	min	34.0	25.0	25.0	21.0	24.0	21.0	21.0	24.0	21.0
	max	59.0	80.0	80.0	82.0	76.0	82.0	82.0	80.0	82.0
male	freq	7	16	23	4	28	32	11	44	55
	mean	48.7	51.1	50.3	41.5	45.4	44.9	46.1	47.5	47.2
	SD	13.9	13.8	13.6	7.9	13.6	13.0	12.2	13.8	13.4
	min	30.0	25.0	25.0	32.0	16.0	16.0	30.0	16.0	16.0
	max	67.0	76.0	76.0	49.0	67.0	67.0	67.0	76.0	76.0
Total	freq	12	48	60	10	65	75	22	113	135
	mean	49.0	52.2	51.6	44.2	45.4	45.2	46.8	48.3	48.1
	SD	11.9	14.1	13.6	17.4	14.6	14.9	14.5	14.7	14.6
	min	30.0	25.0	25.0	21.0	16.0	16.0	21.0	16.0	16.0
	max	67.0	80.0	80.0	82.0	76.0	82.0	82.0	80.0	82.0

Table 5.4.4 Distribution of age by sex and environment in the non-core population of 1996.

Table 5.4.4 shows that the newly diagnosed group in 1996 were, on average, 6.4 years younger than those diagnosed prior to 1994 (45.2 years and 51.6 years respectively). The mean age of the non-newly diagnosed group was 5.1 years less than that of the study sample in 1993 (see table 5.2.3) with a change more marked in the male population (from 51.4 years to 44.9 years in males and from 49.8 to 45.5 years in females). These results show that those diagnosed between 1994 – 1996 were younger at the time of examination than those diagnosed prior to 1994. This may have however been due to the death of those diagnosed prior to 1994. This necessitates the examination of the age at the time of diagnosis.

Table 5.4.5 shows the age at diagnosis of the newly diagnosed diabetics. Three traditional males' and two traditional females' data in the newly diagnosed group are missing.

		time of diagnosis of diabetes and environment								
		- diabetes diagnosed before 1994 -			- diabetes diagnosed 1994 - 1996 -			-------- TOTAL --------		
	sex	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL
female	freq	5	32	37	6	35	41	11	67	78
	mean	39.0	46.5	45.5	45.0	44.5	44.6	42.3	45.5	45.0
	SD	11.3	14.5	14.2	22.0	15.2	16.0	17.4	14.8	15.1
	min	26.0	16.0	16.0	21.0	23.0	21.0	21.0	16.0	16.0
	max	50.0	73.0	73.0	81.0	76.0	81.0	81.0	76.0	81.0
male	freq	7	16	23	4	25	29	11	41	52
	mean	41.6	42.7	42.3	40.5	43.4	43.0	41.2	43.1	42.7
	SD	12.7	15.6	14.5	8.3	13.9	13.2	10.8	14.4	13.7
	min	26.0	15.0	15.0	30.0	15.0	15.0	26.0	15.0	15.0
	max	62.0	67.0	67.0	49.0	66.0	66.0	62.0	67.0	67.0
Total	freq	12	48	60	10	60	70	22	108	130
	mean	40.5	45.3	44.3	43.2	44.1	43.9	41.7	44.6	44.1
	SD	11.7	14.8	14.3	17.2	14.6	14.8	14.1	14.6	14.5
	min	26.0	15.0	15.0	21.0	15.0	15.0	21.0	15.0	15.0
	max	62.0	73.0	73.0	81.0	76.0	81.0	81.0	76.0	81.0

Table 5.4.5 Distribution of age at the time of diagnosis by sex and environment in the non-core population of 1996.

As was shown in tables 5.2.4, 5.3.6 and 5.3.7 the mean age at diagnosis decreased in the study population (from 45.9 years in 1993 to 44. 5 years) and in the non-core population (from 46.8 years to 44.1 years) by 1996. However, in the newly diagnosed diabetics the mean age at diagnosis was only 43.9 years (table 5.4.5). The distribution of age at diagnosis in the non-core population of 1996 is shown in table 5.4.6.

age at diagnosis of diabetes	time of diagnosis of diabetes: diabetes diagnosed before 1994	diabetes diagnosed 1994 -1996	TOTAL
15 - 19	3	1	4
	75.00	25.00	100.00
	5.00	1.43	3.08
20 - 24	2	4	6
	33.33	66.67	100.00
	3.33	5.71	4.62
25 - 29	5	6	11
	45.45	54.55	100.00
	8.33	8.57	8.46
30 - 34	5	11	16
	31.25	68.75	100.00
	8.33	15.71	12.31
35 - 39	5	11	16
	31.25	68.75	100.00
	8.33	15.71	12.31
40 - 44	12	4	16
	75.00	25.00	100.00
	20.00	5.71	12.31
45 - 49	9	10	19
	47.37	52.63	100.00
	15.00	14.29	14.62
50 - 54	5	4	9
	55.56	44.44	100.00
	8.33	5.71	6.92
55 - 59	3	5	8
	37.50	62.50	100.00
	5.00	7.14	6.15
60 - 64	5	7	12
	41.67	58.33	100.00
	8.33	10.00	9.23
65 - 69	3	4	7
	42.86	57.14	100.00
	5.00	5.71	5.38
70 - 74	3	1	4
	75.00	25.00	100.00
	5.00	1.43	3.08
75+	.	2	2
	.	100.00	100.00
	.	2.86	1.54
TOTAL	60	70	130
	46.15	53.85	100.00
	100.00	100.00	100.00

Table 5.4.6 Distribution of age at diagnosis in the non-core population of 1996.

Table 5.4.6 shows that in the non-core population of the study 33% of those diagnosed before 1994 were younger than 40, compared with 47% in those diagnosed between 1993 – 1996. These results show that the younger age in those diagnosed between 1994 – 1996 is unlikely to be the result of death in those diagnosed prior to 1994. The younger age at examination was due to the earlier presentation of diabetes in those diagnosed between 1993 – 1996. This has major implications on the health of the Aboriginal communities and therefore has to be considered when planning health programs in Aboriginal communities.

As shown in tables 5.3.8 and 5.3.9 the time since diagnosis is fairly short in this diabetic population and it has somewhat increased by the end of the study (from 4 years to 5.3 years). This change is due to the ageing of the core population (Chapter 5, section 3). The mean of the time since diagnosis in the subgroups of the non-core population in 1996 is remarkable.

Table 5.4.7 shows the time since diagnosis in those subjects in the non-core population of 1996. Data from two males and two females, all living in a traditional environment are missing.

Table 5.4.8 shows the time since diagnosis of diabetes in the subgroups of the non-core population of 1996 in five year intervals.

Table 5.4.7 shows that the diabetic population of the non-core group in 1996, that was diagnosed before 1994, is comparable to the core-group of the 1996 study sample as far as time since diagnosis concerned, since it is almost identical in the two groups (7.3 and seven years). This raises further questions regarding the composition of the diabetic population in Katherine

region and the dynamics of diabetes itself, however, it is beyond the scope of this thesis to address these questions in detail.

		time of diagnosis of diabetes and environment								
		- diabetes diagnosed before 1994 -			- diabetes diagnosed 1994 - 1996 -			TOTAL		
	sex	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL
female	freq	5	32	37	6	35	41	11	67	78
	mean	10.4	6.3	6.8	1.0	0.9	1.0	5.3	3.5	3.7
	SD	6.4	3.8	4.3	0.9	0.8	0.8	6.4	3.8	4.2
	min	5.0	3.0	3.0	0.0	0.0	0.0	0.0	0.0	0.0
	max	21.0	17.0	21.0	2.0	2.0	2.0	21.0	17.0	21.0
male	freq	7	16	23	4	26	30	11	42	53
	mean	7.1	8.4	8.0	1.0	1.2	1.1	4.9	3.9	4.1
	SD	4.1	7.3	6.4	1.2	0.8	0.8	4.5	5.7	5.5
	min	3.0	3.0	3.0	0.0	0.0	0.0	0.0	0.0	0.0
	max	13.0	27.0	27.0	2.0	2.0	2.0	13.0	27.0	27.0
Total	freq	12	48	60	10	61	71	22	109	131
	mean	8.5	7.0	7.3	1.0	1.0	1.0	5.1	3.7	3.9
	SD	5.2	5.3	5.2	0.9	0.8	0.8	5.4	4.6	4.7
	min	3.0	3.0	3.0	0.0	0.0	0.0	0.0	0.0	0.0
	max	21.0	27.0	27.0	2.0	2.0	2.0	21.0	27.0	27.0

Table 5.4.7 Distribution of time since diagnosis by gender environment in the non-core population of 1996.

diagnosis of diabetes and time since diagnosis of diabetes	environment and sex --- non-traditional -- female	male	TOTAL	----- traditional ---- female	male	TOTAL	-------- TOTAL female	male	TOTAL
diagnosed prior to 1994									
0 - 4	.	3 42.86	3 25.00	15 46.88	7 43.75	22 45.83	15 40.54	10 43.48	25 41.67
5 - 9	3 60.00	1 14.29	4 33.33	12 37.50	5 31.25	17 35.42	15 40.54	6 26.09	21 35.00
10 - 14	1 20.00	3 42.86	4 33.33	3 9.38	1 6.25	4 8.33	4 10.81	4 17.39	8 13.33
10 - 19	.	.	.	2 6.25	1 6.25	3 6.25	2 5.41	1 4.35	3 5.00
20 - 24	1 20.00	.	1 8.33	.	1 6.25	1 2.08	1 2.70	1 4.35	2 3.33
25 - 29	.	.	.	.	1 6.25	1 2.08	.	1 4.35	1 1.67
TOTAL	5 100.00	7 100.00	12 100.00	32 100.00	16 100.00	48 100.00	37 100.00	23 100.00	60 100.00
diagnosed between 1994 - 1996									
0 - 4	6 100.00	4 100.00	10 100.00	35 100.00	26 100.00	61 100.00	41 100.00	30 100.00	71 100.00
TOTAL	6 100.00	4 100.00	10 100.00	35 100.00	26 100.00	61 100.00	41 100.00	30 100.00	71 100.00

Table 5.4.8 Distribution of time since diagnosis by sex and environment in the non-core population of 1996.

Table 5.4.8 shows the distribution of time since diagnosis by sex and environment in the non-core population in 1996. Comparing these figures to the core and non-core populations of 1993 will allow conclusions to be drawn regarding the dynamics of diabetes in the study populations (see discussion).

Table 5.4.9 shows the distribution of the random blood sugar values of diabetics in the non-core population of 1996. There were 50 subjects in the non-core population diagnosed prior to 1994, two of them with a GTT (glucose tolerance test). There are 29 missing data; three females and six males in the non-traditional population, twelve females and eight males in the traditional population. In the 75 subjects in the non-core population of 1996 diagnosed between 1994 and 1996 there were six subjects were diagnosed with a GTT. Of the remaining 69 subjects there are missing data for 26 subjects; four females and two males in the non-traditional population and 12 females and eight males in the traditional population.

As shown in tables 5.2.8, 5.3.11 and 5.3.12 the study population was diagnosed with the same high random BSL in 1993 (15.5 mmol/l) and 1996 (15.4 mmol/l). The newly diagnosed group in the non-core population of 1996 was diagnosed with an equally high random BSL (15.2 mmol/l) (table 5.4.8). As demonstrated before (Chapter 5 section 2) the surveillance technique for diabetes had not changed since 1990 in Katherine region. This observation has a major significance in understanding the natural history of the disease and in the screening for diabetes (see discussion).

		time of diagnosis of diabetes and environment								
		- diabetes diagnosed - before 1994			- diabetes diagnosed - 1994 - 1996			-------- TOTAL --------		
	sex	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL	non-traditional	traditional	TOTAL
female	freq	2	18	20	2	21	23	4	39	43
	mean	14.9	15.3	15.3	18.1	14.6	14.9	16.5	15.0	15.1
	SD	0.4	3.2	3.0	3.9	3.4	3.5	2.9	3.3	3.3
	min	14.7	11.2	11.2	15.4	9.3	9.3	14.7	9.3	9.3
	max	15.2	20.6	20.6	20.9	25.7	25.7	20.9	25.7	25.7
male	freq	1	8	9	2	17	19	3	25	28
	mean	15.5	14.6	14.7	11.9	16.2	15.8	13.1	15.7	15.4
	SD		3.1	3.0	0.6	4.0	4.0	2.2	3.8	3.7
	min	15.5	11.4	11.4	11.4	11.7	11.4	11.4	11.4	11.4
	max	15.5	18.7	18.7	12.3	24.3	24.3	15.5	24.3	24.3
Total	freq	3	26	29	4	38	42	7	64	71
	mean	15.1	15.1	15.1	15.0	15.3	15.3	15.1	15.2	15.2
	SD	0.4	3.2	3.0	4.3	3.7	3.7	3.0	3.5	3.4
	min	14.7	11.2	11.2	11.4	9.3	9.3	11.4	9.3	9.3
	max	15.5	20.6	20.6	20.9	25.7	25.7	20.9	25.7	25.7

Table 5.4.9 Distribution of random blood sugar by sex and environment in the non-core population of 1996.

5.5 Pivotal findings in the general characteristics of the study population

There were 234 diabetics in the Katherine region in 1993 and 239 in 1996. The majority of diabetics were located in the four major settlements at the beginning and at the end of the study: Borroloola, Kalkaringi, Lajamanu and Wurli-Wurlinjang. In 1996 the proportion of newly diagnosed diabetics was considerably higher in Barunga and Borroloola than expected on the basis of the proportion of diabetics in these two communities.

The female/male ratio was 2.3 in 1993 and 1.8 in 1996. The increase in the proportion of males is most marked in the newly diagnosed group (female/male ratio = 1.3).

In 1993 91.5% and in 1996 84.4% of the study subjects came from a traditional environment.

In the 1996 survey the mean age of the diabetic population was 49.8 years compared with 50.3 years in 1996. In the non-core population of 1996 the mean age of the newly diagnosed diabetics at examination was 6.4 years less than in the non-newly diagnosed group and 5.1 years less than in the study population of 1993, with a change more marked in the male population. Examination of the age at diagnosis shows, that this is unlikely to be due to the death of the diabetics diagnosed earlier; the mean age at diagnosis decreased from 45.9 years in 1993 to 44.5 years in 1996. In the newly diagnosed group, however, the mean age at diagnosis was only 43.9 years.

These results have a major significance for the health of the Aboriginal population.

It was shown that the methods of surveillance have not changed since 1990, and the presentation of diabetes at an earlier age is due to the changing nature of the disease. It has major implications for planning health services in the region.

The mean time since diagnosis in this study population was 4 years in 1993 and 5.3 years in 1996. It was shown that the difference in the time since diagnosis in the two study populations was due to the aging of the core population (see chapter 5.3). Detailed description of the non-core population of 1996 shows that the non-core population of 1996 (the "newcomers") consisted of two subpopulations: the newly diagnosed group with a mean time since diagnosis of one year and the non-newly diagnosed group with a mean time since diagnoses of 7.3 years – a value fairly similar to the core population in 1996. In 1993 the prevalence of those diagnosed with diabetes for longer than 10 years prior to examination was 7% (16/227) compared with 12% (28/238) in 1996.

The most common method of diagnosing diabetes was the measurement of random BSL. Despite the alertness of Territory health professionals, diabetics are diagnosed with unusually high random BSL values (mean above 15 mmol/l). The greater emphasis on opportunistic screening for diabetes between 1990 – 1994 had no effect on the random blood sugar level diabetes was diagnosed with.

Chapter 6

6 Description of diabetic retinopathy in 1993 and in 1996

6.1 Overview of presentation of results

In this chapter the descriptive epidemiology of diabetic retinopathy will be presented in terms of:

- the stages of diabetic retinopathy
- the prevalence of diabetic maculopathy
- the prevalence of CSME (clinically significant macula edema)
- the prevalence of VTR (vision threatening retinopathy).

These characteristics will be tabulated separately for the right and left eyes, in all eyes where the retina was examinable and in the worst affected eye in each subject. The latter is hereafter termed "subject retinopathy".

Tabulation of diabetic retinopathy in the right and left eyes will show if retinopathy appears bilaterally in the same stage. A deviation from the symmetry of the lesions would warrant further investigation.

Presentation of diabetic retinopathy in all eyes seen gives an estimate of the burden diabetic retinopathy places on ophthalmic services (each eye of a subject affected by VTR is lasered).

Considering diabetic retinopathy as "subject retinopathy" is more relevant to everyday clinical practice. The prevalence of subject retinopathy will determine the number of subjects who need to travel for treatment (subjects travel and not individual eyes) and subject retinopathy will determine the follow-up of the patient (CSME represents a special case - see description later).

CSME and maculopathy are tabulated by stages of diabetic retinopathy.

Since laser treatment interferes with the presence of diabetic retinopathy in the study population, the prevalence of diabetic retinopathy, maculopathy, CSME and VTR in the study population is described after adjusting for the effect of previous laser treatments as well. Subjects without current evidence of the respective condition but who had had confirmed laser therapy for the condition were pooled with those who had the condition. The prevalence of diabetic retinopathy as found in the community is referred to by the term " field observations", the term "observation corrected for the effect of laser treatment" refers to the population with the above correction. The term "lasered subjects" refers to those who underwent laser therapy. This detailed analysis is necessary to avoid bias in interpreting the results of the study. Although proportions of subjects are given on many occasions in this chapter, in some instances they are based on very small numbers.

In 1993 there were five subjects (seven eyes) with laser scarring relating to previous CSME, there were no laser scars relating to panretinal photocoagulation (PRP). In 1996 there were 17 subjects with laser scars. Of these, nine subjects had laser scarring relating to CSME and PRP and eight subjects had laser scarring relating to CSME only. Of the 32 eyes with laser

scars 16 eyes had laser scars relating to previous CSME only; 15 eyes had laser scars due to PRP and previous CSME. In one eye there were laser scars relating to PRP only (this person had laser scars due to CSME in one eye and proliferative diabetic retinopathy in the other eye). There was one other subject who had laser therapy in the 1996 study population, but the retinas were not visible in 1996, therefore this subject was omitted from the analysis.

The distribution of diabetics in the region and the relatively better access to services in some warrants the introduction of the term "major centers". This term refers to those communities where there were more than 20 known diabetics and where access to services was better than in the rest of the communities. The term "major centers" includes Borroloola, Kalkaringi, Lajamanu and Wurli-Wurlinjang Health Center. Since equal access to services is always an issue in the ophthalmic care of Katherine Region, a description of diabetic retinopathy in terms of major centers may provide data to suggest better resource allocation. Therefore section 3 provides a description of the distribution of diabetic retinopathy by communities and with a comparison of its distribution in "major centers" and in smaller communities.

6.2 Description of diabetic retinopathy in 1993 and in 1996.

While the study population was described in detail in Chapter 5, Table 6.2.1 gives a brief overview of the most important characteristics of those subjects where at least one of the retinas could be seen in 1993 and in 1996.

Demographic characteristics		1993	1996
Number of subjects		213	239
Number of eyes where the retina was visualised		415	472
Gender	Males	65	84
	Females	148	155
Environment			
	Traditional	194	201
	Non – traditional	19	38
Habitation			
	major centers	152	167
	other communities	61	72
Age at the time of examination (years)			
	freq	213	238
	mean	48.9	49.5
	SD	13.1	13.8
	min	13	16
	max	91	94
Time since diagnosis (years)			
	freq	210	234
	mean	3.9	5.2
	SD	4.0	4.6
	min	0.0	0.0
	max	26.0	29.0
Age at the time of diagnosis of diabetes (years)			
	freq	210	233
	mean	44.9	44.2[1]
	SD	13.2	13.6
	min	12.0	14.0
	max	90.0	90.0

Table 6.2.1 Demographic characteristics of the KRDRS study population in 1993 and in 1996. Entries are frequencies, except where explicitly noted for age and time.

[1] The earlier age at diagnosis of diabetes in 1996 compared with 1993 reflects the younger age at diagnosis among newcomers to the study in 1996.

Table 6.2.2 presents the prevalence of diabetic retinopathy in 1993 and in 1996. Mitchell (1981) found the prevalence of diabetic retinopathy to be 49% in a diabetic clinic in Newcastle and 39% in the Singleton census area. On the basis of these prevalence figures from the KRDRS, it appears that diabetic retinopathy affects the Aboriginal population of Katherine region to a lesser extent than the non-Aboriginal population of Australia. We can only speculate if this may be due to death of those with severe disease or their move to Darwin for further treatment. However one must also consider the severity of the condition expressed by the proportion of subjects with clinically significant macula edema (CSME) and vision threatening retinopathy (VTR).

	1993 n (%)	1996 n (%)	Risk Ratio	95% CI	P value
Total number of subjects	213	239			
Total number of eyes assessed	415	472			
Subjects with diabetic retinopathy					
Mild BDR	29 (13.6)	25 (10.5)	0.8	0.5, 1.3	0.29
Moderately sever BDR	5 (2.4)	18 (7.5)	3.2	1.2, 8.7	0.02
Severe BDR	2 (0.9)	4 (1.7)	1.9	0.4, 10.6	0.45
Proliferative diabetic retinopathy	2 (0.9)	3 (1.3)	1.5	0.3, 8.6	0.68
Subtotal	38 (17.8)	50 (20.9)	1.3	0.9, 1.8	0.16
Eyes with diabetic retinopathy					
Mild BDR	51 (12.3)	25 (10.5)	0.7	0.5, 1.17	0.20
Moderately sever BDR	7 (1.7)	18 (7.5)	3.48	1.3, 9.9	0.01
Severe BDR	4 (1.0)	4 (1.7)	1.3	0.2, 7.6	0.74
Proliferative diabetic retinopathy	3 (0.7)	3 (1.3)	1.2	0.2, 7.9	0.85
Subtotal	65 (15.7)	50 (20.9)	1.4	0.8, 1.6	0.45
Concordance between stages of diabetic retinopathy in the right and left eyes (%)	95	90			

Table 6.2.2 Diabetic retinopathy in the KRDRS in 1993 and in 1996. Entries are frequencies, percentages, risk ratios, 95% CIs and P values.

Table 6.2.3 shows the prevalence of diabetic maculopathy in the study population in 1993 and in 1996. The prevalence values for maculopathy are high in this population. In 1993, 13% of subjects (10% of eyes) in the study had maculopathy. The respective figures for 1996 were 10% and 8%. If the frequencies of subjects (and eyes) with maculopathy are expressed as a proportion of subjects (and eyes) having diabetic retinopathy (see Table 6.2.2) we find that in 1993, 27 of the 38 subjects (71%) and 43 of the 65 eyes (66%) having retinopathy had maculopathy. The respective figures in 1996 were 24/50 (48%) for subjects and 39/84 (46%) for eyes.

In 1993, 25 subjects with diabetic maculopathy the condition was bilateral in 16 (64%). In 1996, in 15 of 23 subjects (65%) with maculopathy the condition was bilateral.

Table 6.2.4 shows the prevalence of CSME in the KRDRS in 1993 and in 1996. Beyond the overall high prevalence of CSME in this population, the high prevalence of CSME in eyes with mild BDR is particularly important. Comparison of Table 6.2.2 and Table 6.2.4 shows that in 1993, 16 of the 38 subjects (42%) and 25 of the 65 eyes (38%) with retinopathy had CSME. The respective figures in 1996 were 14/50 (28%) for subjects and 21/84 (25%) for eyes.

In 1993, of 14 subjects with CSME the condition was bilateral in 9 (64%). In 1996, in 7 of 13 subjects (54%) with CSME it was bilateral.

	1993 n (%)	1996 n (%)	Risk Ratio	95% CI	P value
Total number of subjects	213	239			
Total number of eyes assessed	413[1]	471			
Subjects with maculopathy	27 (13)	24 (10)	0.8	0.5,1.2	0.29
Eyes with maculopathy	43 (10)	39 (8)	0.8	0.5,1.2	0.32
Concordance between stages of diabetic retinopathy in the right and left eyes (%)	96	97			

1 In 1993 there were two eyes with proliferative diabetic retinopathy where the macula could not be seen.

Table 6.2.3 Diabetic maculopathy among diabetics in the KRDRS in 1993 and in 1996. Entries are frequencies, percentages, risk ratios, 95% CIs and P values.

	1993 n (%)	1996 n (%)	Risk Ratio	95% CI	P value
Total number of subjects	212	239			
Total number of eyes assessed	413[1]	471			
Subjects with CSME and diabetic retinopathy					
Mild BDR	10 (34)	4 (16)	0.5	0.2, 1.3	0.14
Moderately sever BDR	4 (80)	6 (33)	0.4	0.2, 0.9	0.03
Severe BDR	2 (100)	3 (75)	0.7	0.4, 1.3	0.29
Proliferative diabetic retinopathy	0	1 (33)			
Subtotal	16 (7.5)	14 (5.9)	0.8	0.4, 1.4	0.40
Eyes with CSME and diabetic retinopathy					
Mild BDR	16 (31)	6 (14)	0.5	0.16, 1.3	0.15
Moderately sever BDR	5 (71)	11 (34)	0.5	0.22, 1.0	0.06
Severe BDR	4 (100)	4 (67)	0.6	0.3, 1.5	0.30
Proliferative diabetic retinopathy	0	0 (1.3)			
Subtotal	25 (6.1)	21 (4.5)	0.5	0.4, 1.4	0.3
Concordance between stages of diabetic retinopathy in the right and left eyes (%)	98	97			

1 In 1993 there were two eyes with proliferative diabetic retinopathy where the macula could not be seen.

Table 6.2.4 CSME among diabetics in the KRDRS in 1993 and in 1996. Entries are frequencies, percentages, risk ratios, 95% CIs and P values.

Table 6.2.5 shows the prevalence of VTR in the KRDRS in 1993 and in 1996. The important feature of this table is that despite the low overall prevalence of diabetic retinopathy among diabetics (Table 6.2.2) the prevalence of VTR is comparable to Mitchell's study (1985), where the prevalence of diabetic retinopathy was approximately double that of the KRDRS. A comparison of Tables 6.2.2 and 6.2.5 shows that in 1993, 18 of 38 subjects (47%) and 28 of 65 eyes (43%) with retinopathy had VTR. The respective figures in 1996 were 16/50 (32%) for subjects and 25/84 (30%) for eyes. Mitchell found that 27% of subjects with retinopathy had VTR in the Newcastle diabetic clinic compared with 22% of subjects in the Singleton census area (Mitchell, 1985). A comparison of results from the KRDRS with Mitchell's figures (Mitchell, 1985) suggests that in Aboriginal communities of the Katherine region, whenever diabetic retinopathy develops, a higher proportion of subjects will be suffering from VTR than in the Caucasian population of Australia.

In 1993, of 18 subjects with VTR, the condition was bilateral in 10 (56%). In 1996 in 9 of 16 subjects (54%) with VTR it was bilateral. None of the subjects with bilateral VTR in 1996 had the condition in 1993.

	1993 n (%)	1996 n (%)	Risk Ratio	95% CI	P value
Total number of subjects	213	239			
Total number of eyes assessed	415	471			
Subjects with VTR	18 (8.5)	16 (6.7)	0.9	0.5,1.5	0.59
Eyes with VTR	28 (6.7)	25 (5.3)	0.4	0.4,1.4	0.43
Concordance between stages of diabetic retinopathy in the right and left eyes (%)	96	97			

Table 6.2.5 VTR among diabetics in the KRDRS in 1993 and in 1996. Entries are frequencies, percentages, risk ratios, 95% CIs and P values.

Table 6.2.6 shows the prevalence of diabetic retinopathy, maculopathy, CSME and VTR in 1993 and in 1996 after correction for the effect of laser treatment. (Subjects without current evidence of the respective condition but who had had confirmed laser therapy for the condition were pooled with those who had the condition.)

A comparison of this table with Table 6.2.2 shows that correction for the effect of laser treatment moderately increased the prevalence of diabetic retinopathy and diabetic maculopathy in 1993 and in 1996. Correction for the effect of laser treatment increased the prevalence of CSME and VTR to approximately twice of the field observations in 1996 (see Tables 6.2.4 and 6.2.5). In 1993, after correction for the effect of laser treatment of those with retinopathy the proportion of subjects with maculopathy was 73% (30/41), with CSME 46% (19/41) with VTR 51% (21/41). The respective figures for eyes were 70% (46/65), 45% (29/65) and 49% (32/65). In 1996 the respective figures were 60% (31/52), 50% (26/52) and 54% (28/52) for subjects and 60% (51/85), 52% (44/85) and 58% (49/85) for eyes.

	1993 n (%)	1996 n (%)	Risk Ratio	95% CI	P value
Subjects with diabetic retinopathy	41(19)	52(22)	1.2	0.9,1.7	0.25
Eyes with diabetic retinopathy	65 (16)	85(18)	1.1	0.8, 1.6	0.45
Subjects with PDR	2 (1)	12 (5)	5.3	1.2, 23.9	0.03
Eyes with PDR	3 (1)	20 (4)	5.9	1.2, 27.1	0.03
Subjects with maculopathy	30 (14)	31 (13)	0.9	0.6,1.3	0.56
Eyes with maculopathy	46 (11)	51 (11)	1.0	0.6,1.4	0.83
Subjects with CSME	19 (9)	26 (11)	1.2	0.7, 1.9	0.49
Eyes with CSME	29 (7)	44 (9)	1.3	0.8, 2.1	0.27
Subjects with VTR	21 (10)	28 (12)	1.2	0.8, 2.0	0.35
Eyes with VTR	32 (8)	49 (10)	1.3	0.8,2.1	0.24

Table 6.2.6 Diabetic retinopathy, proliferative diabetic retinopathy, maculopathy, CSME and VTR after correction for the effect of laser treatment in the KRDRS in 1993 and in 1996. Entries are frequencies, percentages, risk ratios, 95% CIs and P values.

In the Katherine region there were more females than males in the identified diabetic population. Despite identical screening methods, frequency of attendance at the clinic, a selection bias for entry into the study (reflecting incomplete community lists) may be operating.

Analysis of the tables in Stanton's study shows that in the Aboriginal population of his study the overall prevalence of retinopathy, the prevalence of retinopathy in males and the prevalence of retinopathy in females were 31%, 20.2% and 35.9%, respectively (Stanton 1985). Therefore, examination of diabetic retinopathy by sex in the study population is warranted.

Table 6.2.7 shows the prevalence of diabetic retinopathy, maculopathy, CSME and VTR by sex. The important feature of this table is that in 1993 the prevalence of diabetic retinopathy was approximately the same in both genders, and in those with retinopathy in males 45% (5/11) had VTR, compared with 48% (13/27) in females. In 1996 the proportion of subjects with retinopathy among diabetics was higher in males than in females. Of those with retinopathy, in males 23% (15/22) of subjects had VTR compared with 39% (11/28) in females. This explains the similar VTR prevalences among diabetics in the male and female population in 1996, despite the different overall retinopathy prevalences.

Table 6.2.8 shows the distribution of diabetic retinopathy, maculopathy, CSME and VTR by sex after correction for the effect of laser treatment. It shows that in 1993, of those with retinopathy, 46% (5/11) of male subjects had VTR, compared with 53% (16/30) of females. The respective figures for 1996 were 41% (9/22) in males and 63% (19/30) in females. As Table 6.2.8 shows, correction for the effect of laser treatment had only changed the

overall prevalence of diabetic retinopathy to a small extent, but almost doubled the estimated prevalence of VTR in 1996.

	Female n (%)	Male n (%)	Risk Ratio	95% CI	P value
1993					
Total number of subjects assessed	148	65			
Subjects with diabetic retinopathy					
Mild BDR	20 (14)	9 (14)	1.02	0.5, 2.1	0.95
Moderately sever BDR	3 (2)	2 (3)	1.5	0.3, 8.9	0.64
Severe BDR	2 (1)	0			
Proliferative diabetic retinopathy	2 (1)	0			
Subtotal	27 (18)	11 (17)	0.9	0.5, 1.75	0.82
Subjects with maculopathy	17 (12)	10 (15)	1.3	0.2, 2.7	0.44
Subjects with CSME	11 (7)	5 (8)	1.02	0.4, 2.8	0.96
Subjects with VTR	13 (9)	5 (8)	0.88	0.3, 2.4	0.79

Table 6.2.7 Distribution of diabetic retinopathy by sex in 1993 and in 1996 (continued next page).

	Female n (%)	Male n (%)	Risk Ratio	95% CI	P value
1996					
Total number of subjects assessed	155	84			
Subjects with diabetic retinopathy					
Mild BDR	12 (8)	13 (15)	2.0	1.0, 4.2	0.07
Moderately sever BDR	12 (8)	6 (7)	0.9	0.4, 2.4	0.87
Severe BDR	2 (1)	2 (1)	0.9	0.4, 2.4	0.87
Proliferative diabetic retinopathy	2 (1)	1 (1)	0.9	0.1, 10.0	0.95
Subtotal	28 (18)	22 (26)	1.4	0.9, 2.4	0.14
Subjects with maculopathy	16 (10)	8 (10)	0.9	0.4, 2.1	0.85
Subjects with CSME	10 (6)	4 (5)	0.7	0.2, 2.3	0.60
Subjects with VTR	11 (7)	5 (6)	0.84	0.3, 2.3	0.74

Table 6.2.7 Distribution of diabetic retinopathy by sex in 1993 and in 1996 (continued).

	Female n (%)	Male n (%)	Risk Ratio	95% CI	P value
1993					
Total number of subjects assessed	148	65			
Subjects with diabetic retinopathy	30 (20)	11 (17)	0.8	0.4,1.6	0.57
Subjects with maculopathy	20 (14)	10 (15)	1.1	0.6, 2.3	0.73
Subjects with CSME	14 (9)	5 (8)	1.5	0.3, 8.9	0.64
Subjects with VTR	16 (11)	5 (8)	0.8	0.3, 2.2	0.68
1996					
Total number of subjects assessed	155	84			
Subjects with diabetic retinopathy	30 (19)	22 (26)	1.4	0.8, 2.2	0.22
Subjects with maculopathy	21 (14)	10 (12)	0.9	0.4, 1.7	0.72
Subjects with CSME	18 (12)	8 (10)	0.8	0.4, 1.8	0.62
Subjects with VTR	19 (12)	9 (11)	0.9	0.4, 1.8	0.72

Table 6.2.8 Distribution of diabetic retinopathy by sex after correction for the effect of laser treatment in 1993 and in 1996.

The eye clinic of the Royal Darwin Hospital often encountered Aboriginal patients from non-traditional living environments presenting with VTR. However, advanced stages of diabetic retinopathy were only rarely seen in these subjects. Therefore, a description of diabetic retinopathy by living environment will show if diabetic retinopathy presents in a more severe form in subjects living in a non-traditional environment, or whether the clinical observation was due to selection bias?

The important feature Table 6.2.9 is that in 1993 the prevalence of diabetic retinopathy was approximately the same in subjects from both living environments, however in the traditional living environment the prevalence of maculopathy, CSME and VTR was approximately twice that of the subjects from the non-traditional living environment. In 1996 the proportion of subjects with retinopathy was higher in diabetics living in the non-traditional environment. On the basis of the uncorrected figures it may seem that of those with retinopathy a higher proportion of subjects from the traditional communities presented with VTR. After corrections for the effect of laser treatment, in 1993, of those with retinopathy nearly twice as many subjects in traditional communities than in non-traditional communities presented with VTR. The respective figure for 1996 was around 50% in both non-traditional and traditional living environments. Some frequencies in the table are small, so the proportions must be treated with caution.

	Non - traditional n (%)	Traditional n (%)	Risk Ratio	95% CI	P value
1993					
Total number of subjects assessed	19	194			
Subjects with diabetic retinopathy					
Mild BDR	3 (16)	8 (13)	0.8	0.3, 2.5	0.77
Moderately sever BDR	0	5 (3)			
Severe BDR	1 (5)	1 (1)	0.1	0.01, 1.5	0.10
Proliferative diabetic retinopathy	0	2 (1)			
Subtotal	4 (21)	34 (18)	0.8	0.3, 2.1	0.70
Subjects with maculopathy	1 (5)	26 (13)	2.6	0.4, 17.9	0.34
Subjects with CSME	1 (5)	15 (8)	1.5	0.2, 10.6	0.70
Subjects with VTR	1 (5)	17 (9)	1.7	0.2, 11.8	0.61

Table 6.2.9 Distribution of diabetic retinopathy by environment in 1993 and in 1996 (continued next page).

	Non - traditional n (%)	Traditional n (%)	Risk Ratio	95% CI	P value
1996					
Total number of subjects assessed	38	201			
Subjects with diabetic retinopathy					
Mild BDR	5 (13)	20 (10)	0.8	0.3, 1.9	0.55
Moderately sever BDR	6 (16)	12 (6)	0.4	0.2, 0.9	0.04
Severe BDR	0	4 (2)			
Proliferative diabetic retinopathy	0	3 (1)			
Subtotal	11 (29)	39 (19)	0.7	0.4, 1.2	0.17
Subjects with maculopathy	1 (3)	23 (11)	4.3	0.6, 31.2	0.14
Subjects with CSME	1 (3)	13 (6)	2.5	0.3, 18.2	0.38
Subjects with VTR	1 (3)	15 (7)	2.8	0.4, 20.8	0.31

Table 6.2.9 Distribution of diabetic retinopathy by environment in 1993 and in 1996 (continued).

	Non - traditional n (%)	Traditional n (%)	Risk Ratio	95% CI	P value
1993					
Total number of subjects assessed	19	194			
Subjects with diabetic retinopathy	4 (21)	37 (19)	0.9	0.4, 2.3	0.83
Subjects with maculopathy	1 (5)	29 (15)	2.9	0.4,19.8	0.29
Subjects with CSME	1 (5)	18 (9)	1.8	0.2, 12.5	0.57
Subjects with VTR	1 (5)	20 (10)	2.0	0.3, 13.8	0.50
1996					
Total number of subjects assessed					
Subjects with diabetic retinopathy	12 (32)	40 (20)	0.6	0.4, 1.1	0.10
Subjects with maculopathy	6 (16)	25 (12)	0.8	0.3, 1.8	0.57
Subjects with CSME	6 (16)	20 (10)	0.6	0.3, 1.5	0.28
Subjects with VTR	6 (16)	22 (11)	0.7	0.3, 1.6	0.39

Table 6.2.10 Distribution of diabetic retinopathy by environment in 1993 and in 1996 after correction for the effect of laser treatment.

Tables 6.2.11 and 6.2.12 show the prevalence of diabetic retinopathy, maculopathy CSME and VTR in 1993 and in 1996 without (Table 6.2.11) and after (Table 6.2.12) adjustment for the effect of laser treatment was made.

An examination of diabetic retinopathy, maculopathy, CSME and VTR by age at diagnosis (<30 or ≥30) and time since diagnosis (0-4, 5-9, 10-14, 15-19, 20-24, 25-29 years) shows that their prevalence increases with increasing time since diagnosis. In some 'age at diagnosis' and 'time since diagnosis brackets', the prevalence figures are based on very small numbers; therefore they must be treated with caution. It is important to note, that in those diagnosed with diabetes aged more than 30 years and less than 4 years prior to examination, 10% (12.121) had retinopathy in 1993 and 11% (12/105) had retinopathy in 1996. 3% (4.121) of subjects had VTR in 1993 compared with 4% (4/105) in 1996. These figures are even higher (4% and 7% respectively) if adjusted for the effect of previous laser treatment. If diabetes was diagnosed prior to age 30, in the similar time since diagnosis bracket, in 1993 there was only one subject of seventeen (6%) who had retinopathy and VTR; in 1996, 2 of the 19 subjects (11%) had retinopathy and none had VTR.

There have been no data on the prevalence of diabetic retinopathy at the time of diagnosis in and Aboriginal Australian diabetic population. The presence of VTR in the 0-4 years time since diagnosis brackets, in particular, necessitates examination of the prevalence of diabetic retinopathy, maculopathy CSME and VTR at the time of diagnosis of diabetes. If diabetic retinopathy and VTR in particular already exist at the time of diagnosis of diabetes, this has implications for a screening program. Since in the KRDRS it was not possible to examine the subjects at the time of diagnosis, those subjects who were diagnosed one year prior to examination were regarded as if they were seen at the time of examination. The KRDRS found that in

1993, 8 of the 70 subjects (11.4%) had diabetic retinopathy at the time of diagnosis and 2 of the 70 subjects (2.9%) had VTR. In 1996, 4 of the 46 subjects (8.7%) had retinopathy at the time of diagnosis and only one of the 46 subjects (2.2%) had VTR. Due to the small frequencies it is not possible to stratify these frequencies by age at the time of diagnosis of diabetes.

year and age at the time of diagnosis of diabetes	time since diagnosis of diabetes (year)					
	0 - 4	5 - 9	10 - 14	15 - 19	20 -24	25 - 29
1993						
<30						
subject retinopathy	1/17 (6%)	1/4 (25%)	1/2 (50%)	1/1 (100%)	.	1/1 (100%)
proliferative DR	0/17	0/4	0/2	0/1	.	0/1
subject maculopathy	1/17 (6%)	0/4	1/2 (50%)	1/1 (100%)	.	1/1 (100%)
subject CSME	1/17 (6%)	0/4	0/2	1/1 (100%)	.	1/1 (100%)
subject VTR	1/17 (6%)	0/4	0/2	1/1 (100%)	.	1/1 (100%)
≥30						
subject retinopathy	12/121 (10%)	15/52 (29%)	5/11 (45%)	.	.	1/1 (100%)
proliferative DR	0/121	2/52 (4%)	0/11	.	.	0/1
subject maculopathy	9/121 (7%)	11/51 (22%)	3/11 (27%)	.	.	0/1
subject CSME	4/121 (3%)	8/51 (16%)	1/11 (9%)	.	.	0/1
subject VTR	4/121 (3%)	10/52 (19%)	1/11 (9%)	.	.	0/1
1996						
<30						
subject retinopathy	2/19 (11%)	1/8 (13%)	1/2 (50%)	0/1	0/2	1/1 (100%)
proliferative DR	0/19	0/8	0/2	0/1	0/2	0/1
subject maculopathy	0/19	1/8 (13%)	1/2 (50%)	0/1	0/2	1/1 (100%)
subject CSME	0/19	1/8 (13%)	1/2 (50%)	0/1	0/2	1/1 (100%)
subject VTR	0/19	1/8 (13%)	1/2 (50%)	0/1	0/2	1/1 (100%)
≥30						
subject retinopathy	12/105 (11%)	23/69 (33%)	9/23 (39%)	1/2	.	0/1
proliferative DR	1/105 (1%)	1/69 (1%)	1/23 (4%)	0/2	.	0/1
subject maculopathy	6/105 (6%)	11/69 (16%)	4/23 (17%)	0/2	.	0/1
subject CSME	3/105 (3%)	5/69 (7%)	3/23 (13%)	0/2	.	0/1
subject VTR	4/105 (4%)	5/69 (7%)	4/23 (17%)	0/2	.	0/1

Table 6.2.11 Subject retinopathy, maculopathy, CSME and VTR in 1993 and in 1996. Entries are frequencies and percentages.

year and age at the time of diagnosis of diabetes	time since diagnosis of diabetes (year)					
	0 - 4	5 - 9	10 - 14	15 - 19	20 -24	25 - 29
1993						
<30						
subject retinopathy	1/17 (6%)	1/4 (25%)	1/2 (50%)	1/1 (100%)	.	1/1 (100%)
proliferative DR	0/17	0/4	0/2	0/1	.	0/1
subject maculopathy	1/17 (6%)	0/4	1/2 (50%)	1/1 (100%)	.	1/1 (100%)
subject CSME	1 /17 (6%)	0/4	0/2	1/1 (100%)	.	1/1 (100%)
subject VTR	1 (6%)	0	0	1 (100%)	.	1 (100%)
≥30						
subject retinopathy	13/121 (11%)	17/52 (33%)	5/11 (45%)	.	.	1/1 (100%)
proliferative DR	0/121	2/52 (4%)	0/11	.	.	0/1
subject maculopathy	10/121 (8%)	13/51 (25%)	3/11 (27%)	.	.	0/1
subject CSME	5/121 (4%)	10/52 (19%)	1/11 (9%)	.	.	0/1
subject VTR	5/121 (4%)	12/52 (23%)	1/11 (9%)	.	.	0/1
1996						
<30						
subject retinopathy	2/19 (11%)	1/8 (13%)	1/2 (50%)	0/1	0/2	1/1 (100%)
proliferative DR	0/19	0/8	0/2	0/1	0/2	1/1 (100%)
subject maculopathy	0/19	1/8 (13%)	1/2 (50%)	0/1	0/2	1/1 (100%)
subject CSME	0/19	1/8 (13%)	1/2 (50%)	0/1	0/2	0/1 (100%)
subject VTR	0/19	1/8 (13%)	1/2 (50%)	0/1	0/2	1/1 (100%)
≥30						
subject retinopathy	12/105 (11%)	23/69 (33%)	11/23 (48%)	1/2 (50%)	.	0/1
proliferative DR	3/105 (3%)	4/69 (6%)	4/23 (17%)	0/2	.	0/1
subject maculopathy	7/105 (7%)	12/69 (17%)	9/23 (39%)	0/2	.	0/1
subject CSME	6/105 (6%)	8/69 (12%)	9/23 (39%)	0/2	.	0/1
subject VTR	7/105 (7%)	8/69 (12%)	10/23 (43%)	0/2	.	0/1

Table 6.2.12 Subject retinopathy, maculopathy, CSME and VTR in 1993 and in 1996 if corrected for the effect of laser treatment. Entries are frequencies and percentages.

6.3 Description of diabetic retinopathy, maculopathy, CSME and VTR by communities

This section describes diabetic retinopathy, maculopathy, CSME and VTR by communities. This section will address the following issues (see 3 below):

1. What is the prevalence of diabetic retinopathy, maculopathy, CSME and VTR in diabetics of each community? (Numerator: subjects with the condition in the particular community; denominator: subjects with diabetes in the community.)
2. What is the burden of diabetes, diabetic retinopathy, maculopathy, CSME and VTR in the particular community relative to the total number of subjects with the condition in the region? (Numerator: subjects with the relevant condition in the community; denominator: subjects with the relevant condition in the region.)

Comparison of the proportions showing the relative burden of diabetes and diabetic retinopathy in the particular community as defined in (2) will identify the communities which carry a disproportionate burden of diabetic retinopathy compared to the proportion of diabetes in the community.

This section presents the observed prevalences of diabetic retinopathy, maculopathy, CSME and VTR by communities and also after corrections for the effect of previous laser treatments were made. The former will be referred to in the tables as "field observations", the latter will be referred to as "observations after corrections for the effect of laser treatment". The

prevalence of diabetic retinopathy after correction for the effect of laser treatment will give a better estimate of the burden of diabetic retinopathy in the communities.

Table 6.3.1 describes diabetic retinopathy, maculopathy, CSME and VTR in each community. It also shows the burden of disease in the community relative to the region by describing diabetes, diabetic retinopathy, maculopathy, CSME and VTR as a proportion of subjects with the respective condition in the particular community compared to those with the condition across all communities. The discrepancies of the denominators in 1993 in Kalkaringi are due to the fact that in one subject, who had proliferative diabetic retinopathy, the macula could not be seen.

The pivotal information in this table is:

- There is a large variation among communities in the proportion of diabetics with diabetic retinopathy. In 1993 the field observations show that in some small communities there were no subjects with diabetic retinopathy (Bulla Camp, Bulman, Hodgson Downs/River), in some other small communities a high proportion of diabetics presented with diabetic retinopathy (Pine Creek 67%(2/3), Binjari (67% (2/3)). These figures however are based on very small numbers. In 1993, in communities with the highest number of diabetics, the proportion of diabetics with diabetic retinopathy was 20% (5/20) in Borroloola, 10% (2/10) in Kalkaringi, 4/45 (9%) in Lajamanu and 30% (14/36) in Wurli – Wurlinjang Health Center). Correcting for the effect of laser treatment doubled the proportion of subjects with diabetic retinopathy in Kalkaringi, however, these figures are based on small numbers.

- In 1993 the proportion of subjects with VTR in the communities varied considerably. With respect to diabetic maculopathy, CSME and VTR the proportion of the subjects with the condition varies in smaller communities. In the major centers the prevalence of VTR is the highest in Borroloola (16% (4/25). The respective figure for Kalkaringi was 10% (2/20), for Lajamanu 4% (2/45) and for Wurli - Wurlinjang 9% (4/46). The proportion of subjects with VTR changed in Kalkaringi to 20% (4/20) after correction for the effect of laser treatment.

- In 1996 the same variation was seen in smaller communities in the proportion of subjects with diabetic retinopathy, maculopathy, CSME and VTR as in 1993. In some communities (for example in Bulla Camp) the proportions were doubled after correcting for the effect of laser treatment in the communities. These figures are based on very small numbers. In 1996, among major centers, the proportion of subjects with diabetic retinopathy among diabetics was the highest in Wurli - Wurlinjang Health Center (32% (16/50)) followed by Borroloola (21% (8/38)) and Lajamanu (21% (10/47). The proportion of subjects with VTR varied in these centers from 2% (1/50) in Kalkaringi to 11% (4/38) in Borroloola. Correction for the effect of laser treatment tripled the proportion of VTR in Wurli - Wurlinjang Health Center from 6% (3/50) to 18% (9/50).

A number of communities had a disproportionate burden of diabetic retinopathy relative to the burden of diabetes in the community. The proportions used for this comparison were derived by comparing the number of subjects with the condition in the particular community, relative to the total number of subjects with the condition in the region. If the

proportion of subjects with diabetic retinopathy (relative to the regional total) was higher in the respective community than the proportion of diabetics (relative to the regional total), it would warrant greater emphasis on clinical and health promotion programs in that community, specifically addressing diabetes and its complications.

In both cross sections of the study, due to the small frequencies of diabetes and diabetic retinopathy, comparisons could only be made in the major centers only. In 1993, the proportion of subjects with VTR in Borroloola was nearly twice that expected on the basis of the proportion of subjects with diabetes. In Lajamanu, the proportion of subjects with VTR was less than expected and in Kalkaringi and the Wurli - Wurlinjang Health Center was around the expected number. After correcting for the effect of laser treatment, in Kalkaringi the proportion of subjects with VTR was nearly twice the expected on the basis of the proportion of subjects with diabetes in the community. On the basis of these figures it may seem that beyond the high concentration of diabetics in the four major centers diabetic retinopathy was a particularly important health problem in Borroloola and in Kalkaringi. However, these figures are based on small numbers.

In 1996, only the Wurli - Wurlinjang Health Center stood out regarding the proportion of diabetics and the proportion of VTR relative to the region. 21% of the subjects with diabetes of the study sample were living in the area serviced by Wurli - Wurlinjang Health Center[1], but 32% of subjects with diabetic retinopathy and 19% of subjects with VTR in the region were living in the area serviced by this health center. If corrections for the effect

[1] The number of diabetics seen at Wurli-Wurlinjang Health Center represents the Aboriginal diabetics from Kalano community, Katherine and various "town-camps" in Katherine. "Town camp" is the term used to denote the place of the habitat of various Aboriginal groups who were staying in Katherine for an uncertain period of time.

of laser treatment are made, 32% of subjects with VTR in the region were living in the area serviced by the Wurli – Wurlinjang Health Center.

	1993		1996	
communities	field observations	observations after corrections for the effect of prior laser treatment	field observations	observations after corrections for the effect of prior laser treatment
Barunga				
No of diabetics	12	12	16	16
Subjects with				
retinopathy	3/12 (25%)	3/12 (25%)	2/16 (13%)	2/16 (13%)
maculopathy	2/12 (17%)	2/12 (17%)	1/16 (6%)	1/16 (6%)
CSME	2/12 (17%)	2/12 (17%)	1/16 (6%)	1/16 (6%)
VTR	2/12 (17%)	2/12 (17%)	1/16 (6%)	1/16 (6%)
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	12/213 (6%)	2/213 (6%)	16/239 (7%)	16/239 (7%)
retinopathy	3/38 (8%)	3/41 (7%)	2/50 (4%)	2/52 (4%)
maculopathy	2/27 (7%)	2/30 (7%)	1/24 (4%)	1/31 (13%)
CSME	2/16 (13%)	2/19 (11%)	1/14 (7%)	1/26 (4%)
VTR	2/18 (11%)	2/21 (10%)	1/16 (6%)	1/28 (4%)
Beswick				
No of diabetics	7	7	6	6
Subjects with				
retinopathy	1/7 (14%)	1/7 (14%)	0/6	0/6
maculopathy	1/7 (14%)	1/7 (14%)	0/6	0/6
CSME	1/7 (14%)	1/7 (14%)	0/6	0/6
VTR	1/7 (14%)	1/7 (14%)	0/6	0/6
Subjects with condition as proportion of those with condition across all communities*				
diabetes	7/213 (3%)	7/213 (3%)	6/239 (3%)	6/239 (3%)
retinopathy	1/38 (3%)	1/41 (2%)	0/50	0/52
maculopathy	1/27 (4%)	1/30 (3%)	0/24	0/31
CSME	1/16 (6%)	1/19 (5%)	0/14	0/26
VTR	1/18 (6%)	1/21 (5%)	0/16	0/28

Table 6.3.1 Description of diabetic retinopathy, maculopathy, CSME and VTR by communities. (* In these proportions the numerator represents the frequencies of the respective conditions in the particular community, the denominator the frequencies across all communities.)

	1993		1996	
communities	field observations	observations after corrections for the effect of prior laser treatment	field observations	observations after corrections for the effect of prior laser treatment
Binjari				
No of diabetics	3	3	6	6
Subjects with				
retinopathy	2/3 (67%)	2/3 (67%)	0/6	0/6
maculopathy	1/3 (33%)	1/3 (33%)	0/6	0/6
CSME	0/3	0/3	0/6	0/6
VTR	0/3	0/3	0/6	0/6
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	3/213 (1%)	3/213 (1%)	6/239 (3%)	6/239 (3%)
retinopathy	2/38 (5%)	2/41 (5%)	0/50	0/52
maculopathy	1/27 (4%)	1/30 (3%)	0/24	0/31
CSME	2/16 (13%)	0/19	0/14	0/26
VTR	0/18	0/21	0/16	0/28
Borroloola				
No of diabetics	25	25	38	38
Subjects with				
retinopathy	5/25 (20%)	5/25 (20%)	8/38 (21%)	9/38 (24%)
maculopathy	5/25 (20%)	5/25 (20%)	4/38 (11%)	5/38 (13%)
CSME	4/25 (16%)	4/25 (16%)	4/38 (11%)	5/38 (13%)
VTR	4/25 (16%)	4/25 (16%)	4/38 (11%)	5/38 (13%)
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	25/213 (12%)	25/213 (12%)	38/239 (16%)	38/239 (16%)
retinopathy	5/38 (13%)	5/41 (12%)	8/50 (16%)	9/52 (17%)
maculopathy	5/27 (19%)	5/30 (17%)	4/24 (17%)	5/31 (16%)
CSME	1/16 (6%)	4/19 (21%)	4/14 (29%)	5/26 (19%)
VTR	4/18 (22%)	4/21 (19%)	4/16 (25%)	5/28 (18%)

Table 6.3.1 (continued) Description of diabetic retinopathy, maculopathy, CSME and VTR by communities. (* In these proportions the numerator represents the frequencies of the respective conditions in the particular community, the denominator the frequencies across all communities.)

	1993		1996	
communities	field observations	observations after corrections for the effect of prior laser treatment	field observations	observations after corrections for the effect of prior laser treatment
Bulla Camp				
No of diabetics	3	3	7	7
Subjects with				
retinopathy	0/3	1/3 (33%)	1/7 (14%)	2/7 (29%)
maculopathy	0/3	1/3 (33%)	1/7 (14%)	2/7 (29%)
CSME	0/3	1/3 (33%)	1/7 (14%)	2/7 (29%)
VTR	0/3	1/3 (33%)	1/7 (14%)	2/7 (29%)
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	3/213 (1%)	3/213 (1%)	7/239 (3%)	7/239 (3%)
retinopathy	0/38	1/41 (2%)	1/50 (2%)	2/52 (4%)
maculopathy	0/27	1/30 (3%)	1/24 (4%)	2/31 (6%)
CSME	0/16	1/19 (5%)	1/14 (7%)	2/26 (8%)
VTR	0/18	1/21 (5%)	1/16 (6%)	2/28 (7%)
Bulman				
No of diabetics	6	6	9	9
Subjects with				
retinopathy	0/6	0/6	3/9 (33%)	3/9 (33%)
maculopathy	0/6	0/6	3/9 (33%)	3/9 (33%)
CSME	0/6	0/6	3/9 (33%)	3/9 (33%)
VTR	0/6	0/6	3/9 (33%)	3/9 (33%)
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	6/213 (3%)	6/213 (3%)	9/239 (4%)	9/239 (4%)
retinopathy	0/38	0/41	3/50 (6%)	3/52 (6%)
maculopathy	0/27	0/30	3/24 (13%)	3/31 (10%)
CSME	0/16	0/19	3/14 (21%)	3/26 (12%)
VTR	0/18	0/21	3/16 (19%)	3/28 (11%)

Table 6.3.1 (continued) Description of diabetic retinopathy, maculopathy, CSME and VTR by communities. (* In these proportions the numerator represents the frequencies of the respective conditions in the particular community, the denominator the frequencies across all communities.)

	1993		1996	
communities	field observations	observations after corrections for the effect of prior laser treatment	field observations	observations after corrections for the effect of prior laser treatment
Duck Creek				
No of diabetics	2	2	5	5
Subjects with				
retinopathy	1/2 (50%)	1/2 (50%)	1/5 (20%)	1/5 (20%)
maculopathy	1/2 (50%)	1/2 (50%)	1/5 (20%)	1/5 (20%)
CSME	0/2	0/2	1/5 (20%)	1/5 (20%)
VTR	0/2	0/2	1/5 (20%)	1/5 (20%)
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	2/213 (1%)	2/213 (1%)	5/239 (2%)	5/239 (2%)
retinopathy	1/38 (3%)	1/41 (2%)	1/50 (2%)	1/52 (2%)
maculopathy	1/27 (3.7%)	1/30 (3%)	1/24 (4%)	1/31 (3%)
CSME	0/16	0/19	1/14 (7%)	1/26 (4%)
VTR	0/18	0/21	1/16 (6%)	1/28 (4%)
Hodgson Downs/River				
No of diabetics	8	8	6	6
Subjects with				
retinopathy	0/8	0/8	1/6 (17%)	1/6 (17%)
maculopathy	0/8	0/8	1/6 (17%)	1/6 (17%)
CSME	0/8	0/8	0/6	0/6
VTR	0/8	0/8	0/6	0/6
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	8/213 (4%)	8/213 (4%)	6/239 (3%)	6/239 (3%)
retinopathy	0/38	0/41	1/50 (2%)	1/52 (2%)
maculopathy	0/27	0/30	1/24 (4%)	1/31 (3%)
CSME	0/16	0/19	0/14	0/26
VTR	0/18	0/21	0/16	0/28

Table 6.3.1 (continued) Description of diabetic retinopathy, maculopathy, CSME and VTR by communities. (* In these proportions the numerator represents the frequencies of the respective conditions in the particular community, the denominator the frequencies across all communities.)

	1993		1996	
communities	field observations	observations after corrections for the effect of prior laser treatment	field observations	observations after corrections for the effect of prior laser treatment
Kalkaringi				
No of diabetics	20	20	20	20
Subjects with				
retinopathy	2/20 (10%)	4/20 (20%)	3/20 (15%)	3/20 (15%)
maculopathy	1/19 (5%)	3/19 (16%)	1/20 (5%)	1/20 (5%)
CSME	1/19 (5%)	3/19 (16%)	1/20 (5%)	1/20 (5%)
VTR	2/20 (10%)	4/20 (20%)	1/20 (5%)	1/20 (5%)
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	20/213 (9%)	20/213 (9%)	20/239 (8%)	20/239 (8%)
retinopathy	2/38 (5%)	4/41 (10%)	3/50 (6%)	3/52 (6%)
maculopathy	1/27 (4%)	3/30 (10%)	1/24 (4%)	1/31 (3%)
CSME	1/16 (6%)	3/19 (16%)	1/14 (7%)	1/26 (4%)
VTR	2/18 (11%)	4/21 (19%)	1/16 (6%)	1/28 (4%)
Kildurk				
No of diabetics	3	3	20	20
Subjects with				
retinopathy	0/3	0/3	3/20 (15%)	3/20 (15%)
maculopathy	0/3	0/3	0/3	0/3
CSME	0/3	0/3	0/3	0/3
VTR	0/3	0/3	0/3	0/3
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	3/213 (213%)	3/213 (1%)	20/239 (8%)	20/239 (8%)
retinopathy	0/38	0/41	3/50 (6%)	3/52 (6%)
maculopathy	0/27	0/30	0/24	0/31
CSME	0/16	0/19	0/14	0/26
VTR	0/18	0/21	0/16	0/28

Table 6.3.1 (continued) Description of diabetic retinopathy, maculopathy, CSME and VTR by communities. (* In these proportions the numerator represents the frequencies of the respective conditions in the particular community, the denominator the frequencies across all communities.)

	1993		1996	
communities	field observations	observations after corrections for the effect of prior laser treatment	field observations	observations after corrections for the effect of prior laser treatment
Lajamanu				
No of diabetics	45	45	47	47
Subjects with				
retinopathy	4/45 (9%)	4/45 (9%)	10/47 (21%)	10/47 (21%)
maculopathy	3/45 (7%)	3/45 (7%)	6/47 (13%)	6/47 (13%)
CSME	1/45 (2%)	1/45 (2%)	0/47	3/47 (12%)
VTR	2/45 (4%)	2/45 (4%)	1/47 (2%)	4/47 (2%)
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	45/213 (21%)	45/213 (21%)	47/239 (20%)	47/239 (20%)
retinopathy	4/38 (11%)	4/41 (9%)	10/50 (20%)	10/50 (20%)
maculopathy	3/27 (11%)	3/30 (10%)	6/24 (25%)	6/31 (13%)
CSME	1/16 (6%)	1/19 (5%)	0/14	3/26 (12%)
VTR	2/18 (11%)	2/21 (5%)	1/16 (6%)	4/28 (14%)
Mataranka				
No of diabetics	4	4	5	5
Subjects with				
retinopathy	0/4	0/4	0/5	0/5
maculopathy	0/4	0/4	0/5	0/5
CSME	0/4	0/4	0/5	0/5
VTR	0/4	0/4	0/5	0/5
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	4/213 (2%)	4/213 (2%)	5/239 (2%)	5/239 (2%)
retinopathy	0/38	0/41	0/50	0/52
maculopathy	0/27	0/30	0/24	0/31
CSME	0/16	0/19	0/14	0/26
VTR	0/18	0/21	0/16	0/28

Table 6.3.1 (continued) Description of diabetic retinopathy, maculopathy, CSME and VTR by communities. (* In these proportions the numerator represents the frequencies of the respective conditions in the particular community, the denominator the frequencies across all communities.)

	1993		1996	
communities	field observations	observations after corrections for the effect of prior laser treatment	field observations	observations after corrections for the effect of prior laser treatment
Ngukurr				
No of diabetics	16	16	12	12
Subjects with				
retinopathy	3/16 (19%)	3/16 (19%)	4/12 (33%)	4/12 (33%)
maculopathy	2/16 (13%)	2/16 (13%)	0/12	1/12 (8%)
CSME	2/16 (13%)	2/16 (13%)	0/12	1/12 (8%)
VTR	2/16 (13%)	2/16 (13%)	1/12 (8%)	2/12 (17%)
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	16/213 (8%)	16/213 (8%)	12/239 (5%)	12/239 (5%)
retinopathy	3/38 (8%)	3/41 (7%)	4/50 (8%)	4/52 (8%)
maculopathy	2/27 (7%)	2/30 (7%)	0/24	1/31 (3%)
CSME	2/16 (13%)	2/19 (11%)	0/14	1/26 (4%)
VTR	2/18 (11%)	2/21 (10%)	1/16 (6%)	2/28 (7%)
Pine Creek				
No of diabetics	3	3	1	1
Subjects with				
retinopathy	2/3 (67%)	2/3 (67%)	0/1	0/1
maculopathy	1/3 (33%)	1/3 (33%)	0/1	0/1
CSME	1/3 (33%)	1/3 (33%)	0/1	0/1
VTR	1/3 (33%)	1/3 (33%)	0/1	0/1
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	3/213 (1%)	3/213 (1%)	1/239 (0.4%)	1/239 (0.4%)
retinopathy	2/38 (5%)	2/41 (5%)	0/50	0/52
maculopathy	1/27 (4%)	1/30 (3%)	0/24	0/31
CSME	1/16 (6%)	1/19 (11%)	0/14	0/26
VTR	1/18 (6%)	1/21 (5%)	0/16	0/28

Table 6.3.1 (continued) Description of diabetic retinopathy, maculopathy, CSME and VTR by communities. (* In these proportions the numerator represents the frequencies of the respective conditions in the particular community, the denominator the frequencies across all communities.)

	1993		1996	
communities	field observations	observations after corrections for the effect of prior laser treatment	field observations	observations after corrections for the effect of prior laser treatment
Timber Creek				
No of diabetics	7	7	5	5
Subjects with				
retinopathy	1/7 (14%)	1/7 (14%)	0/5	0/5
maculopathy	1/7 (14%)	1/7 (14%)	0/5	0/5
CSME	0/7	0/7	0/5	0/5
VTR	0/7	0/7	0/5	0/5
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	7/213 (3%)	7/213 (3%)	5/239 (2%)	5/239 (2%)
retinopathy	1/38 (3%)	1/41 (2%)	0/50	0/52
maculopathy	1/27 (4%)	1/30 (3%)	0/24	0/31
CSME	0/16	0/19	0/14	0/26
VTR	0/18	0/21	0/16	0/28
Wurli - Wurlinjang				
No of diabetics	46	46	50	50
Subjects with				
retinopathy	14/46 (30%)	14/46 (30%)	16/50 (32%)	16/50 (32%)
maculopathy	9/46 (20%)	9/46 (20%)	5/50 (10%)	9/50 (18%)
CSME	4/46 (9%)	4/46 (9%)	3/50 (6%)	9/50 (18%)
VTR	4/46 (9%)	4/46 (9%)	3/50 (6%)	9/50 (18%)
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	46/213 (22%)	46/213 (22%)	50/239 (21%)	50/239 (21%)
retinopathy	14/38 (37%)	14/41 (34%)	16/50 (32%)	16/52 (31%)
maculopathy	9/27 (33%)	9/30 (3%)	5/24 (21%)	9/31 (29%)
CSME	4/16 (25%)	4/19 (21%)	3/14 (21%)	9/26 (35%)
VTR	4/18 (22%)	4/21 (19%)	3/16 (19%)	9/28 (32%)

Table 6.3.1 (continued) Description of diabetic retinopathy, maculopathy, CSME and VTR by communities. (* In these proportions the numerator represents the frequencies of the respective conditions in the particular community, the denominator the frequencies across all communities.)

	1993		1996	
communities	field observations	observations after corrections for the effect of prior laser treatment	field observations	observations after corrections for the effect of prior laser treatment
Yarralin				
No of diabetics	3	3	3	3
Subjects with				
retinopathy	0/3	0/3	1/3 (33%)	1/3 (33%)
maculopathy	0/3	0/3	1/3 (33%)	1/3 (33%)
CSME	0/3	0/3	0/3	0/3
VTR	0/3	0/3	0/3	0/3
*Subjects with condition as proportion of those with condition across all communities**				
diabetes	3/213 (1%)	3/213 (1%)	3/239 (1%)	3/239 (1%)
retinopathy	0/38	0/41	1/50 (2%)	1/52 (2%)
maculopathy	0/27	0/30	1/24 (4%)	1/31 (4%)
CSME	0/16	0/19	0/14	0/26
VTR	0/18	0/21	0/16	0/28

Table 6.3.1 (continued) Description of diabetic retinopathy, maculopathy, CSME and VTR by communities. (* In these proportions the numerator represents the frequencies of the respective conditions in the particular community, the denominator the frequencies across all communities.)

In Table 6.3.1 many proportions are based on very small numbers. Therefore, a better estimate can be obtained if diabetic retinopathy is described in terms of "major centers" and in "other communities".

	1993				1996			
	Field observations		Observations after corrections for the effect of prior laser treatment		Field observations		Observations after corrections for the effect of prior laser treatment	
	Major centers	Other communities	Major centers	Other communities	Major centers	Other communities	Major centers	Other communities
Subjects with								
Retinopathy	28/152 (18%)	10/61 (16%)	30/152 (20%)	11/61 (18%)	40/167 (25%)	9/72 (13%)	42/167 (25%)	10/72 (14%)
Proliferative DR	2/152 (1%)	0	2/152 (1%)	0	3/167 (2%)	0	11/167 (7%)	1/72 (1%)
Maculopathy	20/151 (13%)	7/61 (11%)	22/152 (14%)	8/61 (13%)	16/167 (10%)	8/72 (11%)	22/167 (13%)	9/72 (13%)
CSME	12/151 (8%)	4/61 (7%)	14/152 (9%)	5/61 (8%)	8/167 (5%)	6/72 (8%)	19/167 (11%)	7/72 (10%)
VTR	14/152 (9%)	4/61 (7%)	16/152 (11%)	5/61 (8%)	10/167 (6%)	6/72 (8%)	19/167 (11%)	7/72 (10%)

Table 6.3.2 Description of diabetic retinopathy, maculopathy, CSME and VTR by major centers and other communities.

Table 6.3.2 shows that in 1993 diabetic retinopathy, maculopathy, CSME and VTR were equally important complications of diabetes in both major centers and in smaller communities. In 1996 it may appear (based on the higher prevalence of diabetic retinopathy) that diabetic retinopathy was a more important complication of diabetes in subjects living in major communities. These differences were not due to the effect of previous laser treatment. In 1996 however, the prevalence of VTR was nearly the same in major centers (6%) and in other communities (8%). After correcting for the effect of laser treatment the prevalence of VTR was 11% in major centers and 10% in other communities. Therefore, in 1996, despite the differences in the overall prevalence of diabetic retinopathy, diabetic retinopathy (as expressed as proportion of subjects with VTR) was and equally important complication of diabetes in major centers and in smaller communities.

The pivotal findings of this chapter are that in Aboriginal communities of Katherine region

- on the basis of the overall prevalence of diabetic retinopathy it may not seem to be an important complication of diabetes, but the high prevalences of CSME and VTR suggest otherwise. This is more evident if corrections for the effect of laser treatment are made
- the high prevalence of CSME in subjects with mild BDR is of particular concern
- there were high VTR prevalences in both major centers and smaller communities
- at the time of diagnosis of diabetes approximately 10% of subjects had diabetic retinopathy and one fifth of these had VTR.

Chapter 7

7 Description of diabetic retinopathy in the core population

7.1 Overview of presentation of results

In Chapter 4, section 2, the term "core population" which denotes those subjects in the study who were seen both in 1993 and in 1996 was introduced. The core population was described in detail in Chapter 5, section 2. The analysis of the core population yields some information on the short - term natural history of diabetic retinopathy in Aboriginal communities.

Sections 2, 3 and 4 of the chapter provide a description of the progression of diabetic retinopathy at the subject level and at the level of the individual eye by sex, living environment and place of residence. This information may contribute to a better understanding of diabetic retinopathy in Aboriginal communities and also to the design and implementation of diabetic eye programs in future.

Since laser treatment interferes with the natural history of diabetic retinopathy, those 12 subjects in the core population lasered prior to their examination in 1996 were excluded from the description of the progression of diabetic retinopathy.

Therefore, the progression of diabetic retinopathy was examined in 192 eyes of 96 subjects. The incidence of CSME in those with and without

maculopathy were not stratified by living environment and communities due to the small number of observations in the sample.

Though the proportion of subjects with certain characteristics may show a marked change from the beginning to the end of the study, on some occasions those proportions are based on very small numbers.

Section 5 of this chapter describes the progression of diabetic retinopathy, maculopathy, CSME and VTR by time since diagnosis in 1993.

7.2 Progression of diabetic retinopathy by sex in the non–lasered core population.

Tables 7.2.1 – 7.2.4 show that:

- The rate of progression of subject retinopathy (nil → any retinopathy) in males is approximately twice that of females. With respect to diabetic retinopathy in eyes, the rate of progression in males is even higher (in the nil → any retinopathy, nil → NPDR strata around three times that in females). Of those who had no retinopathy the annual progression rate to VTR (nil → VTR category) was the same in both males and females (Tables 7.2.1 – 7.2.2). Some of the proportions in Tables 7.2.1 and 7.2.2 are based on very small numbers.

- The rate of progression of subject maculopathy in males was approximately twice that of females. With respect to the progression of maculopathy in eyes, the figures are comparable to the progression of subject maculopathy (Tables 7.2.3 – 7.2.4).

- The rate of progression of subject CSME was approximately the same in both genders. If examined in eyes however, the progression rate was 50% higher in males than in females. These proportions however are based on very small numbers (Tables 7.2.3 – 7.2.4).

- The rate of progression of VTR (no VTR → VTR category) is comparable in males and females whether it is examined at subject or at eye level (Tables 7.2.3 – 7.2.4).

- 50% (3/6) of those subjects who progressed to maculopathy during the study also developed CSME. The respective figure for eyes was 59% (5/9) (Tables 7.2.3 – 7.2.4).

- Of those who do not have maculopathy, 1.1% of subjects (0.9% of eyes) progressed to CSME annually. Of those subjects who had maculopathy 4.8% (6.1% of eyes) progressed to CSME annually (Tables 7.2.3 – 7.2.4).

- The different rate of progression of diabetic retinopathy in males and females cannot be explained by the time since diagnosis, since the distribution of time since diagnosis in 1993 is very similar in males and females (Tables 7.2.5).

Progression (1993 → 1996)	Female	Male	Total
nil → any retinopathy			
proportion	7/57	7/27	14/84
3 year rate	12.3%	25.9%	16.7%
95% CI	5%, 25.3%	10.4%, 53.4%	9.1%, 30.0%
annual rate	4.1%	8.6%	5.6%
95% CI	1.7%, 5.1%	3.5%, 17.8%	3.0%, 10.0%
nil → NPDR			
proportion	6/57	7/27	13/84
3 year rate	10.5%	25.9%	15.5%
95% CI	3.9%, 22.9%	10.4%, 53.4%	8.2%, 26.5%
annual rate	3.5%	8.6%	5.2%
95% CI	3.3%, 7.6%	3.5%, 17.8%	2.7%, 8.8%
nil → PDR			
proportion	1/57	0/27	1/84
3 year rate	1.8%		1.2%
95% CI	0.04%, 9.8%	0, 0.14[1]	0.03%, 6.6%
annual rate	0.6%		0.4%
95% CI	0.01%, 3.3%	0, 0.05[1]	0.01%, 2.2%
nil → VTR			
proportion	2/57	1/27	3/84
3 year rate	3.5%	3.7%	3.6%
95% CI	0.4%, 12.7%	0.09%, 20.6%	0.7%, 10.4%
annual rate	1.2%	1.2%	1.2%
95% CI	0.1%, 4.2%	0.03%, 6.8%	0.2%, 3.8%
mild → moderately severe BDR			
proportion	3/9[2]	1/2	4/11[3]
3 year rate	33.3%	50.0%	36.4%
95% CI	6.9%, 97.4%	0.1%, 2.8%	9.9%, 93.1%
annual rate	11.1%	17.0%	12.1%
95% CI	2.3%, 32.8%	0.03%, 0.9%	3.3%, 31.0%
mild BDR → VTR			
proportion	1/6[2]	0/2	1/8[3]
3 year rate	16.7%		12.5%
95% CI	0.4%, 92.9%	0, 1.84[1]	0.3%, 69.6%
annual rate	5.6%		4.2%
95% CI	0.1%, 31.0%	0, 0.61	0.1%, 23.2%

[1] One-sided, 97.5% confidence interval.
[2,3] The different denominator figures suggest that in this cohort 3 subjects with mild BDR had VTR in 1993.

Table 7.2.1 Progression of subject retinopathy by sex in the non-lasered core population between 1993 and 1996.

Progression (1993 → 1996)	Female	Male	Total
nil → any retinopathy			
proportion	9/114[1]	12/53	21/167[2]
3 year rate	7.9%	22.6%	12.6%
95% CI	3.6%, 14.5%	11.5%, 44.5%	7.5%, 21.0%
annual rate	2.6%	7.7%	4.2%
95% CI	1.2%, 4.5%	3.5%. 14.8%	2.5%, 7.0%
nil → NPDR			
proportion	7/112[3]	12/53	19/165[4]
3 year rate	6.3%	22.6%	11.5%
95% CI	2.5%, 12.9%	11.5%, 44.5%	6.7%, 19.7%
annual rate	2.1%	7.7%	3.8%
95% CI	0.8%, 4.3%	3.5%. 14.8%	2.2%, 6.6%
nil → PDR			
proportion	2/114[1]	0/53[5]	2/167[2]
3 year rate	1.8%		1.2%
95% CI	0.02%, 6.3%	0, 0.07	0.1%, 4.3%
annual rate	0.6%		0.4%
95% CI	0.007%,2.1%	0, 0.02	0.03%, 1.4%
nil → VTR			
proportion	4/114[1]	2/53	6/167[2]
3 year rate	3.5%	3.8%	3.8%
95% CI	1.0%, 9.0%	0.5%, 13.6%	0.5%, 13,6%
annual rate	1.2%	1.3%	1.2%
95% CI	0.3%, 3.0%	0.2%, 4.5%	0.2%, 4.5%
mild → moderately severe BDR			
proportion	4/16	1/3	5/19
3 year rate	25.0%	33.3%	26.3%
95% CI	6.8%, 64.0%	0.8%, 1.85	8.5%, 61.4%
annual rate	8.3%	11.1%	8.8%
95% CI	2.3%, 21.3%	0.3%, 61%	2.8%, 20.8%
mild BDR → VTR			
proportion	1/16	0/3[5]	3/19
3 year rate	6.3%		15.8%
95% CI	0.2%, 34.8%	0,1.2[5]	3.3%, 46.1%
annual rate	2.1%		5.3%
95% CI	0.07%,11.9%	0, 0.4[5]	1.1%, 15.4%

[1] The denominator is 114 instead of 115 eyes since in one eye, that had no DR in 1993, the retina could not be judged in 1996.
[2] The denominator is 167 instead of 168 eyes since in one eye, that had no DR in 1993, the retina could not be judged in 1996
[3] The denominator is 112 instead of 115 eyes since in one eye, that had no DR in 1993, the retina could not be judged in 1996 and two eyes had proliferative stage diabetic retinopathy.
[4] The denominator is 165 instead of 168 eyes since in one eye, that had no DR in 1993, the retina could not be judged in 1996 and also, 2 of those eyes not having retinopathy in 1993 had proliferative stage diabetic retinopathy in 1996.
[5] One –sided, 97.5% confidence interval.

Table 7.2.2 Progression of diabetic retinopathy by sex in the eyes of the non-lasered core population between 1993 and 1996.

Progression (1993 → 1996)	Female	Male	Total
subject maculopathy (no → yes)			
proportion	3/61	3/28	6/89
3 year rate	4.9%	10.7%	6.7%
95% CI	1.0%, 14.4%	2.2%, 31.3%	2.0%, 14.7%
annual rate	1.6%	3.6%	2.2%
95% CI	0.3%, 2.8%	0.7%, 10.4%	0.7%, 4.6%
subject CSME (no → yes)			
proportion	2/64	1/29	3/93
3 year rate	3.1%	3.4%	3.2%
95% CI	0.4%, 11.3%	0.09%,19.2%	0.7%, 9.4%
annual rate	1.0%	1.1%	1.1%
95% CI	0.1%, 3.8%	0.03%, 6.7%	0.2%, 3.1%
subject VTR (no → yes)			
proportion	3/64	1/29	4/93
3 year rate	4.7%	3.4%	4.3%
95% CI	1.0%, 13.7%	0.09%,19.2%	1.2%, 11.0%
annual rate	1.6%	1.1%	1.4%
95% CI	0.3%, 4.6%	0.03%, 6.7%	0.4%, 3.7%
subject maculopathy (no) → subject CSME (yes)			
proportion	2/61	1/28	3/89
3 year rate	3.3%	3.6%	3.4%
95% CI	0.4%, 11.8%	0.09%,19.9%	0.7%, 9.8%
annual rate	1.1%	1.2%	1.1%
95% CI	0.1%, 3.9%	0.03%, 6.6%	0.2%, 3.3%
subject maculopathy (yes) → subject CSME (yes)			
proportion	1/6	0/1	1/7
3 year rate	16.7%		14.3%
95% CI	0.4%, 92.8%	0, 3.7[1]	0.4%, 79.6%
annual rate	5.6%		4.8%
95% CI	0.1%, 30.9%	0, 1.2[1]	0.1%, 26.5%

[1] One-sided, 97.5% confidence interval.

Table 7.2.3 Progression of subject maculopathy, CSME and VTR by sex in the non-lasered core population between 1993 and 1996.

Progression (1993 → 1996)	Female	Male	Total
maculopathy (no → **yes**)			
proportion	5/122[1]	4/54	9/176[3]
3 year rate	4.1%	7.4%	5.1%
95% CI	1.3%, 9.6%	2.0%, 19.0%	2.3%, 9.7%
annual rate	1.4%	2.5%	1.7%
95% CI	0.4%, 3.2%	0.7%, 6.3%	0.7%, 3.2%
CSME (no → **yes**)			
proportion	3/126[3]	2/56[5]	5/182[4]
3 year rate	2.4%	3.6%	2.7%
95% CI	0.5%, 7.0%	0.4%, 12.9%	0.9%, 6.4%
annual rate	0.8%	1.2%	0.9%
95% CI	0.2%, 2.7%	0.1%, 4.3%	0.3%, 2.1%
VTR (no → **yes**)			
proportion	5/126[3]	2/56[5]	7/182[4]
3 year rate	4.0%	3.6%	3.8%
95% CI	1.3%, 9.3%	0.4%, 12.9%	1.5%, 7.9%
annual rate	1.3%	1.2%	1.3%
95% CI	0.4%, 3.1%	0.1%, 4.3%	0.5%, 2.6%
maculopathy (no) → CSME (yes)			
proportion	3/122[1]	2/54	5/176[2]
3 year rate	2.5%	3.7%	2.8%
95% CI	0.5%, 7.2%	0.4%, 13.4%	0.9%, 6.6%
annual rate	0.8%	1.2%	0.9%
95% CI	0.2%, 2.4%	0.1%, 4.5%	0.3%, 2.2%
maculopathy (yes) → CSME (yes)			
proportion	2/9	0/2	2/11
3 year rate	22.2%		18.2%
95% CI	2.7%, 80.2%	0, 1.8[5]	2.2%, 65.7%
annual rate	7.4%		6.1%
95% CI	0.9%, 26.7%	0, 0.6[5]	0.7%, 21.9%

[1] In one female subject the macula could not be judged in 1996, therefore the denominator is 122 instead of 123.
[2] For similar reasons the denominator is 176 instead of 177.
[3] For similar reasons the denominator is 126 instead of 127.
[4] For similar reasons the denominator is 182 instead of 183.
[5] In one eye of a male subject the macula could not be seen in 1993 and another eye could not be seen in 1996, therefore the denominator is 56 instead of 58.
[5] One-sided, 97.5% confidence interval.

Table 7.2.4 Progression of diabetic maculopathy, CSME and VTR by sex in eyes of the non-lasered core population between 1993 and 1996.

gender	time since diagnosis of diabetes (years) 0 - 4	5 - 9	10 - 14	25 - 29	missing	TOTAL
female	45	19	1	1	1	67
	67.16	28.36	1.49	1.49	1.49	100.00
male	18	7	4			29
	62.07	24.14	13.79	.	.	100.00
TOTAL	63	26	5	1	1	96
	65.63	27.08	5.21	1.04	1.04	100.00

Table 7.2.5 Distribution of time since diagnosis in 1993 by gender in the non-lasered core population.

7.3 Progression of diabetic retinopathy by living environment in the non–lasered core population.

Tables 7.3.1 - 7.3.5 show that:

- In comparable strata the rate of progression for subject retinopathy is nearly the same in traditional and non–traditional living environments. At eye level however, the rate of progression in traditional living environment is somewhat higher than in non–traditional living environment. Some of these figures are based on very small numbers (Tables 7.3.1 – 7.3.2) and precision of estimation suffers accordingly.

- While there was some evidence of progression of diabetic maculopathy, CSME and VTR among subjects of the traditional living environment, no subjects from the non-traditional living environment experienced progression (Tables 7.3.3– 7.3.4).

- Evidence from Mitchell suggests, that progression most often occurs in subjects whose diagnosis since diabetes exceeds 10 years (Mitchell, 1985). The difference in progression rate between traditional and non-traditional subjects in this study cannot be explained by the different distribution of time since diagnosis (Table 7.3.5).

Progression (1993 → 1996)	Non-traditional	Traditional	Total
nil → any retinopathy			
proportion	2/12	12/72	14/84
3 year rate	16.7%	16.7%	16.7%
95% CI	20.2%,60.2%	8.6%, 29.1%	9.1%, 30.0%
annual rate	5.6%	5.6%	5.6%
95% CI	6.7%, 20.1%	2.9%, 9.7%	3.0%, 10.0%
nil → NPDR			
proportion	2/12	11/72	13/84
3 year rate	16.7%	15.3%	15.5%
95% CI	20.2%,60.2%	7.6%, 27.3	8.2%, 26.5%
annual rate	5.6%	5.1%	5.2%
95% CI	6.7%, 20.1%	2.9%, 9.1%	2.7%, 8.8%
nil → PDR			
proportion	0/12	1/72	1/84
3 year rate		1.4%	1.2%
95% CI	0, 0.31[1]	0.04%, 7.8%	0.03%, 6.6%
annual rate		4.6%	0.4%
95% CI	0, 0.10[1]	0.01%, 2.6%	0.01%, 2.2%
nil → VTR			
proportion	0/12	3/72	3/84
3 year rate		4.2%	3.6%
95% CI	0, 0.31[1]	0.9%, 12,2%	0.7%, 10.4%
annual rate		1.3%	1.2%
95% CI	0, 0.10[1]	0.3%, 4.1%	0.2%, 3.5%
mild → moderately severe BDR			
proportion	1/2	3/9[2]	4/11[3]
3 year rate	50.0%	33.3%	36.4%
95% CI	1.3%, 2.78	6.9%, 97.4%	9.9%, 93.1%
annual rate	16.7%	11.1%	12.1%
95% CI	0.4%, 92.7%	2.3%, 32.5%	3.3%, 31.0%
mild BDR → VTR			
proportion	0/2	1/6[2]	1/8[3]
3 year rate		16.7%	12.5%
95% CI	0, 1.8[1]	4.2%, 92.9%	0.3%, 69.6%
annual rate		5.6%	4.2%
95% CI	0, 0.6[1]	1.4%, 31.0%	0.1%, 23.2%

[1] One-sided, 97.5% confidence interval.
[2,3] The different denominator figures suggest that 3 subjects with mild BDR had VTR in 1993.

Table 7.3.1 Progression of subject retinopathy by living environment in the non-lasered core population between 1993 and 1996.

Progression (1993 → 1996)	Non- traditional	Traditional	Total
nil → any retinopathy			
proportion	2/25	19/142[1]	21/167[2]
3 year rate	8.0%	13.4%	12.6%
95% CI	1.0%,28.9%	7.7%, 23.2%	7.5%, 21.0%
annual rate	2.7%	4.5%	4.2%
95% CI	0.3%, 9.6%	2.6%, 7.7%	2.5%, 7.0%
nil → NPDR			
proportion	2/25	17/140[4]	19/165[3]
3 year rate	8.0%	12.1%	11.5%
95% CI	1.0%,28.9%	6.8%, 21.7%	6.7%, 19.7%
annual rate	2.7%	4.0%	3.8%
95% CI	0.3%, 9.6%	2.3%, 7.3%	2.3%, 6.6%
nil → PDR			
proportion	0/25	2/142[1]	2/167[2]
3 year rate		1.4%	1.2%
95% CI	0, 0.15[5]	0.2%, 5.1%	0.1%, 4.3%
annual rate		0.5%	0.4%
95% CI	0, 0.05[5]	0.7%, 1.7%	0.03%, 1.4%
nil → VTR			
proportion	0/25	6/142[1]	6/167[2]
3 year rate		4.2%	3.6%
95% CI	0, 0.15[5]	1.6%, 9.2%	1.3%, 7.8%
annual rate		1.4%	1.2%
95% CI	0, 0.05[5]	0.5%, 3.1%	0.4%, 2.6%
mild → moderately severe BDR			
proportion	1/2	4/17	5/19
3 year rate	50.0%	23.5%	26.3%
95% CI	1.3%, 2.817.0%	6.4%, 60.2%	8.5%, 61.4%
annual rate	0.4%, 93.3%	7.8%	8.8%
95% CI		2.1%, 20.0%	2.8%, 20.5%
Mild BDR → VTR			
proportion	0/2	3/17	3/19
3 year rate		17.7%	15.8%
95% CI	0, 1.8[5]	3.6%, 51.6%	3.3%, 46.1%
annual rate		5.9%	5.3%
95% CI	0, 0.6[5]	1.2%, 27.2%	1.1%, 15.4%

[1] The denominator is 142 instead of 143 eyes since in one eye, that had no DR in 1993, the retina could not be judged in 1996.
[2] For similar reasons the denominator is 167 instead of 168.
[3] The denominator is 165 instead of 168 eyes since in one eye, that had no DR in 1993, the retina could not be judged in 1996 and also, 2 of those eyes not having retinopathy in 1993 had proliferative stage diabetic retinopathy in 1996.
[4] The denominator is 140 instead of 142 eyes since 2 eyes in the traditional group with no retinopathy in 1993 developed proliferative stage diabetic retinopathy in 1996.
[5] One-sided, 97.5% confidence interval.

Table 7.3.2 Progression of diabetic retinopathy by living environment in eyes of the non-lasered core population between 1993 and 1996.

Progression (1993 → 1996)	Non - traditional	Traditional	Total
subject maculopathy (no → yes)			
proportion	0/14	6/75	6/89
3 yearly rate		8.0%	6.7%
95% CI	0, 0.26[1]	2.9%, 17.4%	2.5%, 14.7%
annual rate		2.7%	3.2%
95% CI	0, 0.09[1]	1.0%, 5.8%	0.8%, 4.9%
subject CSME (no → yes)			
proportion	0/14	3/79	3/93
3 yearly rate		3.8%	3.2%
95% CI	0, 0.26[1]	0.8%, 11.1%	0.7%, 9.4%
annual rate		1.3%	1.1%
95% CI	0, 0.09[1]	0.3%, 3.7%	0.2%, 3.1%
subject VTR (no → yes)			
proportion	0/14	4/79	4/93
3 yearly rate		5.1%	4.3%
95% CI	0, 0.26[1]	1.3%, 13.0%	1.2%, 11.0%
annual rate		2.7%	1.4%
95% CI	0, 0.09[1]	0.4%, 4.3%	0.4%, 3.6%

[1] One-sided, 97.5% confidence interval.

Table 7.3.3 Progression of subject maculopathy, CSME and VTR by living environment in the non-lasered core population between 1993 and 1996.

Progression (1993 → 1996)	Non-traditional	Traditional	Total
maculopathy (no → yes)			
proportion	0/29	9/149[1]	9/176[2]
3 yearly rate		6.0%	5.1%
95% CI	0, 0.14[5]	2.8%, 11.5%	2.3%, 9.7%
annual rate		2.0%	1.7%
95% CI	0, 0.05[5]	0.9%, 3.8%	0.8%, 3.2%
CSME (no → yes)			
proportion	0/29	5/155[4]	5/182[3]
3 yearly rate		3.2%	2.7%
95% CI	0, 0.14[5]	1.0%, 7.5%	0.9%, 6.4%
annual rate		1.1%	0.9%
95% CI	0, 0.05[5]	0.3%, 2.5%	0.3%, 2.1%
VTR (no → yes)			
proportion	0/29	7/155[4]	7/182[3]
3 yearly rate		4.5%	3.8%
95% CI	0, 0.14[5]	1.8%, 9.3%	1.5%, 7.9%
annual rate		1.5%	1.3%
95% CI	0, 0.05[5]	0.6%, 3.1%	0.5%, 2.6%

[1] In one eye that had no maculopathy in 1993 the macula could not be judged in 1996, therefore the denominator is 149 instead of 150.
[2] For similar reasons the denominator is 176 instead of 177.
[3] For similar reasons the denominator is 182 instead of 183.
[4] For similar reasons the denominator is 155 instead of 156.
[5] One-sided, 97.5% confidence interval.

Table 7.3.4 Progression of diabetic maculopathy, CSME and VTR by living environment in the eyes of the non-lasered core population between 1993 and 1996.

environment	time since diagnosis of diabetes 0 - 4	5 - 9	10 - 14	25 - 29	missing	TOTAL
non-traditional	8	4	2			14
	57.14	28.57	14.29	.	.	100.00
traditional	55	22	3	1	1	82
	67.07	26.83	3.66	1.22	1.22	100.00
TOTAL	63	26	5	1	1	96
	65.63	27.08	5.21	1.04	1.04	100.00

Table 7.3.5 Distribution of time since diagnosis in 1993 by living environment in the non – lasered core population.

7.4 Progression of diabetic retinopathy in major centers and in other communities in the non–lasered core population.

Tables 7.4.1 –7.4.5 show that:

- The progression of diabetic retinopathy was fairly similar in major centers and in "other communities" (Tables 7.4.1 – 7.4.2).

- The progression of diabetic maculopathy, CSME and VTR in "other communities" was several times that in major centers (Tables 7.4.3 – 7.4.4).

- Table 7.4.5 shows that there is a somewhat unequal distribution of time since diagnosis in major centers compared with "other communities". Due to the small numbers of subjects (and eyes) it is not possible to determine if the unequal distribution of time since diagnosis is responsible for the differences seen in progression of maculopathy, CSME and VTR.

Progression (1993 → 1996)	Major centers	Other communities	Total
nil → any retinopathy			
proportion	11/61	3/23	14/84
3 year rate	18.0%	13.0%	16.7%
95% CI	9%, 32.3%	2.7%, 38.1%	9.1%, 30.0%
annual rate	6.0%	4.3%	5.6%
95% CI	3%, 10.8%	0.9%, 12.7%	3.0%, 10.0%
nil → NPDR			
proportion	10/61	3/23	13/84
3 year rate	16.4%	13.0%	15.5%
95% CI	7.9%, 30.1%	2.7%, 38.1%	8.2%, 26.5%
annual rate	5.5%	4.3%	5.2%
95% CI	2.6%, 10.0%	0.9%, 12.7%	2.7%. 8.8%
nil → PDR			
proportion	1/61	0/23	1/84
3 year rate	1.6%		1.2%
95% CI	0.04%, 9.1%	0, 0.16[1]	0.03%, 6.6%
annual rate	0.5%		0.4%
95% CI	0.01%, 3.0%	0, 0.05[1]	0.01, 2.2%
nil → VTR			
proportion	1/61	2/23	3/84
3 year rate	1.6%	8.7%	3.6%
95% CI	0.04%, 9.1%	1.1%, 31.4%	0.7%, 0.4%
annual rate	0.5%	2.9%	1.2%
95% CI	0.01%, 3.0%	0.4%, 10.5%	0.2%, 0.1%
mild → moderately severe BDR			
proportion	3/7	1/4	4/11[2]
3 year rate	42.9%	25.0%	36.4%
95% CI	8.8%, 1.25	0.6%, 1.4	9.9%, 3.1%
annual rate	14.3%	8.3%	12.1%
95% CI	2.9%, 41.7%	0.2%, 46.7%	3.3%, 1.0%
mild → VTR			
proportion	0/5	1/3	1/8[3]
3 year rate		33.3%	12.5%
95% CI	0, 0.74[1]	0.8%, 1.85	0.3%, 69.6%
annual rate		11.1%	4.2%
95% CI	0, 0.25[1]	0.3%, 61.7%	0.1%, 23.2%

[1] One-sided, 97.5% confidence interval.

Table 7.4.1 Progression of subject retinopathy in the non-lasered core population by place of residence between 1993 and 1996.

Progression (1993 → 1996)	Major centers	Other communities	Total
nil → any retinopathy			
proportion	15/120[1]	6/47	21/167[2]
3 year rate	12.5%	12.8%	12.6%
95% CI	7.0%, 22.3%	4.7%, 27.8%	7.5%, 21.0%
annual rate	4.2%	4.3%	4.2%
95% CI	2.7%, 7.4%	1.6%, 9.3%	2.5%, 7.0%
nil → NPDR			
proportion	13/118[3]	6/47	19/165[3]
3 year rate	11.0%	12.8%	11.5%
95% CI	6.0%, 20.4%	4.7%, 27.8%	6.7%, 19.7%
annual rate	3.7%	4.3%	3.8%
95% CI	2.0%, 6.8%	1.6%, 9.3%	3.2%, 6.6%
nil → PDR			
proportion	2/120[1]	0/47	2/167[2]
3 year rate	1.7%		1.2%
95% CI	0.2%, 6.0%	0, 0.08[5]	0.1%, 4.3%
annual rate	0.6%		0.4%
95% CI	0.07%, 2.0%	0, 0.03[5]	0.03, 1.4%
nil → VTR			
proportion	2/120[1]	4/47	6/167[2]
3 year rate	1.7%	8.5%	3.6%
95% CI	0.2%, 6.0%	2.3%, 21.8%	1.3%, 7.8%
annual rate	0.6%	2.8%	1.2%
95% CI	0.07%, 2.0%	0.8%, 7.3%	0.4%, 2.6%
mild → moderately severe BDR			
proportion	4/12	1/7	5/19
3 year rate	33.3%	14.3%	26.3%
95% CI	9.1%, 85.3%	0.4%, 79.6%	8.5%, 61.4%
annual rate	11.1%	4.8%	8.8%
95% CI	3.0%, 28.4%	0.1%, 26.5%	2.8%, 20.5%
mild BDR → VTR			
proportion	2/12	1/7	3/19
3 year rate	16.7%	14.3%	15.8%
95% CI	2.0%, 60.2%	0.4%, 79.6%	3.3%, 46.1%
annual rate	5.6%	4.8%	5.3%
95% CI	0.7%, 20.1%	0.1%, 26.5%	1.1%, 15.4%

[1] The denominator is 120 instead of 121 eyes since in one eye, that had no DR in 1993, the retina could not be judged in 1996.
[2] For similar reasons the denominator is 167 instead of 168.
[3] The denominator is 118 instead of 120 since two eyes that had no retinopathy in 1993 had proliferative stage diabetic retinopathy in 1996.
[4] The denominator is 165 instead of 168 eyes since in one eye, that had no DR in 1993, the retina could not be judged in 1996 and also, 2 of those eyes not having retinopathy in 1993 had proliferative stage diabetic retinopathy in 1996.
[5] One-sided, 97.5% confidence interval.

Table 7.4.2 Progression of retinopathy in eyes of the non-lasered core population by place of residence between 1993 and 1996.

Progression (1993 → 1996)	Major centers	Other communities	Total
maculopathy (no → yes)			
proportion	2/63	4/26	6/89
3 year rate	3.2%	15.4%	6.7%
95% CI	0.4%, 11.5%	4.2%, 39.4%	2.5%, 14.7%
annual rate	1.1%	5.1%	3.2%
95% CI	0.1%, 3.8%	1.4%, 13.1%	0.8%, 4.9%
CSME (no → yes)			
proportion	0/66	3/27	3/93
3 year rate		11.1%	3.2%
95% CI	0, 0.06[1]	2.3%, 32.5%	0.7%, 9.4%
annual rate		3.7%	1.1%
95% CI	0, 0.02[1]	0.8%, 10.8%	0.2%, 3.1%
VTR (no → yes)			
proportion	1/66	3/27	4/93
3 year rate	1.5%	11.1%	4.3%
95% CI	0.04%, 8.4%	2.3%, 32.5%	1.2%, 11.0%
annual rate	0.5%	3.7%	1.4%
95% CI	0.01%, 2.8%	0.8%, 10.8%	0.4%, 3.7%

[1] One-sided, 97.5% confidence interval.

Table 7.4.3 Progression of subject maculopathy, CSME and VTR by place of residence in the non-lasered core population between 1993 and 1996.

Progression (1993 → 1996)	Major centers	Other communities	Total
maculopathy (no → yes)			
proportion	3/124[1]	6/52	9/176[2]
3 year rate	2.4%	11.5%	5.1%
95% CI	0.5%, 7.1%	4.2%, 25.1%	2.3%, 9.7%
annual rate	0.8%	3.8%	1.7%
95% CI	0.2%, 2.4%	1.4%, 8.4%	0.8%, 3.2%
CSME (no → yes)			
proportion	0/129	5/53	5/182[3]
3 year rate		9.4%	2.7%
95% CI	0, 0.03[4]	3.1%, 22.0%	0.9%, 6.4%
annual rate		3.1%	0.9%
95% CI	0, 0.01[4]	1.0%, 7.3%	0.3%, 2.1%
VTR (no → yes)			
proportion	2/129	5/53	7/182[3]
3 year rate	1.6%	9.4%	3.8%
95% CI	0.2%, 5.6%	3.1%, 22.0%	1.5%, 7.9%
annual rate	0.5%	3.1%	1.3%
95% CI	0.07%, 1.9%	1.0%, 7.3%	0.5%, 2.6%

[1] The denominator is 124 instead of 125 eyes since in one eye, that had no maculopathy in 1993, the macula could not be judged in 1996.
[2] For similar reasons the denominator is 176 instead of 177.
[3] For similar reasons the denominator is 182 instead of 183.
[4] One-sided, 97.5% confidence interval.

Table 7.4.4 Progression of diabetic maculopathy, CSME and VTR in eyes of the non-lasered core population by place of residence between 1993 and 1996.

communities	time since diagnosis of diabetes 0 - 4	5 - 9	10 - 14	25 - 29	missing	TOTAL
major centers	48 70.59	16 23.53	4 5.88	.	.	68 100.00
other communities	15 53.57	10 35.71	1 3.57	1 3.57	1 3.57	28 100.00
TOTAL	63 65.63	26 27.08	5 5.21	1 1.04	1 1.04	96 100.00

Table 7.4.5 Distribution of time since diagnosis in 1993 by living environment in the non–lasered core population.

7.5 Progression of diabetic retinopathy, maculopathy, CSME and VTR by time since diagnosis in the non – lasered core population.

Examination of the tables presenting progression of diabetic retinopathy shows that they contain very small numbers in rows representing other than the "nil" categories in 1993. Therefore, progression of diabetic retinopathy, maculopathy, CSME and VTR are only reported when the subjects (and the eyes) did not show any of these conditions in 1993.

Table 7.5.1 shows the progression of subject retinopathy by time since diagnosis in 1993 in the non – lasered core population of the study.

Table 7.5.2 shows the progression of diabetic retinopathy by time since diagnosis in 1993 in eyes of the non – lasered core population of the study.

Table 7.5.3 shows the progression of subject maculopathy, CSME and VTR by time since diagnosis in 1993 in the non – lasered core population of the study.

Table 7.5.4 shows the progression of diabetic maculopathy, CSME and VTR by time since diagnosis in 1993 in eyes of the non – lasered core population of the study.

These tables show that the rates of progression for diabetic retinopathy, maculopathy, CSME and VTR were the highest in the "4 to 9 years" time

since diagnosis interval. These tables also show the small number of observations in the higher time since diagnosis intervals.

Progression of subject retinopathy		Time since diagnosis (years) 0 – 4	5 – 9	10 – 14	24 – 29
nil → PDR					
	proportion	0/59	1/21	0/3	.
	3 year rate		4.8%		
	95% CI	0, 0.06[1]	0.1%, 26.5%	0, 1.2[1]	
	annual rate		1.6%		
	95% CI	0, 0.02[1]	0.03%, 8.8%	0, 0.4[1]	
nil → NPDR					
	proportion	7/59	6/21	0/3	.
	3 year rate	11.9%	28.6%		
	95% CI	4.7%, 24.4%	10.5%, 62.2%	0, 1.2[1]	
	annual rate	4.0%	9.5%		
	95% CI	1.6%, 8.1%	3.5%, 20.7%	0, 0.4[1]	
nil → any retinopathy					
	proportion	7/59	7/21	0/3	.
	3 year rate	11.9%	33.3%		
	95% CI	4.7%, 24.4%	13.4%, 68.7%	0, 1.2[1]	
	annual rate	4.0%	11.1%		
	95% CI	1.6%, 8.1%	4.5%, 22.9%	0, 0.4[1]	
nil → mild BDR					
	proportion	5/59	4/21	0/3	.
	3 year rate	8.5%	19.0%		
	95% CI	2.7%, 19.8%	5.2%, 48.8%	0, 1.2[1]	
	annual rate	2.8%	6.3%		
	95% CI	0.9%, 6.6%	1.7%, 16.3%	0, 0.4[1]	
nil → moderately severe BDR					
	proportion	2/59	1/21	0/3	.
	3 year rate	3.4%	4.8%		
	95% CI	0.4%, 12.2%	0.1%, 26.5%	0, 1.2[1]	
	annual rate	1.1%	1.6%		
	95% CI	0.1%, 4.1%	0.03%, 8.8%	0, 0.4[1]	
nil → severe BDR					
	proportion	0/59	1/21	0/3	.
	3 year rate		4.8%		
	95% CI	0, 0.06[1]	0.1%, 26.5%	0, 1.2[1]	
	annual rate		1.6%		
	95% CI	0, 0.02[1]	0.03%, 8.8%	0, 0.4[1]	
nil → VTR					
	proportion	1/59	2/19	0/3	.
	3 year rate	1.7%	10.5%		
	95% CI	0.04%, 9.4%	1.3%, 38.0%	0, 1.2[1]	
	annual rate	0.6%	3.5%		
	95% CI	0.01%, 3.1%	0.4%, 12.7%	0, 0.4[1]	

[1] One-sided 97.5% confidence interval.

Table 7.5.1 Progression of subject retinopathy by time since diagnosis of diabetes in 1993 in the non – lasered core population of the study.

Progression of subject retinopathy	Time since diagnosis (years)			
	0 – 4	5 – 9	10 – 14	24 – 29
nil → PDR				
proportion	0/118	2/40	0/6	0/1
3 year rate		5.0%		
95% CI	0, 0.31[1]	0.6%, 18.1%	0, 0.61[1]	0, 3.7[1]
annual rate		1.7%		
95% CI	0, 0.10[1]	0.2%, 6.0%	0, 0.20[1]	0, 1.2[1]
nil → NPDR				
proportion	10/118	9/40	0/6	0/1
3 year rate	8.5%	22.5%		
95% CI	4.1%, 15.6%	10.3%, 42.7%	0, 0.61[1]	0, 3.7[1]
annual rate	2.8%	7.5%		
95% CI	1.4%, 5.2%	3.8%, 14.2%	0, 0.20[1]	0, 1.2[1]
nil → any retinopathy				
proportion	10/118	11/40	0/6	0/1
3 year rate	8.5%	27.5%		
95% CI	4.1%, 15.6%	13.7%, 49.2%	0, 0.61[1]	0, 3.7[1]
annual rate	2.8%	9.2%		
95% CI	1.4%, 5.2%	4.6%, 16.4%	0, 0.20[1]	0, 1.2[1]
nil → mild BDR				
proportion	7/118	6/40	0/6	0/1
3 year rate	5.9%	15.0%		
95% CI	23.9%, 12.2%	5.5%, 32.6%	0, 0.61[1]	0, 3.7[1]
annual rate	2.0%	5.0%		
95% CI	8.0%, 4.1%	1.8%, 10.9%	0, 0.20[1]	0, 1.2[1]
nil → moderately severe BDR				
proportion	3/118	1/40	0/6	0/1
3 year rate	2.5%	2.5%		
95% CI	0.5%, 7.4%	0.06%, 13.9%	0, 0.61[1]	0, 3.7[1]
annual rate	0.8%	0.8%		
95% CI	0.2%, 2.5%	0.02%, 4.6%	0, 0.20[1]	0, 1.2[1]
nil → severe BDR				
proportion	0/118	2/40	0/6	0/1
3 year rate		5.0%		
95% CI	0, 0.31[1]	0.6%, 18.1%	0, 0.61[1]	0, 3.7[1]
annual rate		1.7%		
95% CI	0, 0.10[1]	0.2%, 6.0%	0, 0.20[1]	0, 1.2[1]
nil → VTR				
proportion	2/118	4/40	0/6	0/1
3 year rate	1.7%	10.0%		
95% CI	0.2%, 6.1%	2.7%, 25.6%	0, 0.61[1]	0, 3.7[1]
annual rate	0.6%	3.3%		
95% CI	0.07%, 2.0%	0.9%, 8.5%	0, 0.20[1]	0, 1.2[1]

[1] One-sided 97.5% confidence interval.

Table 7.5.2 Progression of diabetic retinopathy by time since diagnosis of diabetes in 1993 in eyes of the non – lasered core population of the study.

	Time since diagnosis (years)			
Progression of	0 – 4	5 – 9	10 – 14	24 – 29
subject maculopathy (no → yes)				
proportion	4/60	2/23	0/4	0/1
3 year rate	6.7%	8.7%		
95% CI	1.8%, 17.1%	1.1%, 31.4%	0, 0.92[1]	0, 3.7[1]
annual rate	2.2%	2.9%		
95% CI	0.6%, 5.7%	0.4%, 10.5%	0, 0.31[1]	0, 1.2[1]
subject CSME (no → yes)				
proportion	1/61	2/25	0/5	0/1
3 year rate	1.6%	8.0%		
95% CI	0.04%, 9.1%	1.0%, 28.9%	0, 0.74[1]	0, 3.7[1]
annual rate	0.5%	2.7%		
95% CI	0.01%, 3.0%	0.3%, 9.6%	0, 0.25[1]	0, 1.2[1]
subject VTR (no → yes)				
proportion	1/61	3/25	0/5	0/1
3 year rate	1.6%	12.0%		
95% CI	0.04%, 9.1%	24.8%, 35.1%	0, 0.74[1]	0, 3.7[1]
annual rate	0.5%	4.0%		
95% CI	0.01%, 3.0%	8.3%, 11.7%	0, 0.25[1]	0, 1.2[1]

[1] One-sided 97.5% confidence interval.

Table 7.5.3 Progression of subject maculopathy, CSME and VTR by time since diagnosis of diabetes in 1993 in the non – lasered core population of the study.

	Time since diagnosis (years)			
Progression of	0 – 4	5 – 9	10 – 14	24 – 29
diabetic maculopathy (no → yes)				
proportion	5/119	4/46	0/7	0/2
3 year rate	4.2%	8.7%		
95% CI	1.4%, 9.8%	2.4%, 22.3%	0, 0.52[1]	0, 1.8[1]
annual rate	1.4%	2.9%		
95% CI	0.5%, 3.3%	0.8%, 7.4%	0, 0.17[1]	0, 0.6[1]
CSME (no → yes)				
proportion	2/121	3/48	0/9	0/2
3 year rate	1.7%	6.3%		
95% CI	0.2%, 6.0%	12.9%, 18.3%	0, 0.41[1]	0, 1.8[1]
annual rate	0.6%	2.1%		
95% CI	0.07%, 2.0%	4.3%, 6.1%	0, 0.14[1]	0, 0.6[1]
VTR (no → yes)				
proportion	2/121	5/48	0/9	0/2
3 year rate	1.7%	10.4%		
95% CI	0.2%, 6.0%	3.4%, 24.3%	0, 0.41[1]	0, 1.8[1]
annual rate	0.6%	3.5%		
95% CI	0.07%, 2.0%	1.1%, 8.1%	0, 0.14[1]	0, 0.6[1]

[1] One-sided 97.5% confidence interval.

Table 7.5.4 Progression of diabetic maculopathy, CSME and VTR by time since diagnosis of diabetes in 1993 in eyes of the non – lasered core population of the study.

The pivotal findings of this chapter are that in the non-lasered core population of the study

- The annual progression rate for diabetic retinopathy from not presenting with the condition at the beginning of the study to having the condition at the end of the study was in subjects and (eyes) 5.6% (4.2%) for retinopathy, 1.1% (0.9%) for CSME and 1.4% (1.3%) for VTR. The annual progression rate from no retinopathy to VTR was 1.2% (1.2%).

- The progression of diabetic retinopathy and maculopathy from not presenting with the condition at the beginning of the study to having the condition at the end of the study was approximately twice (if examined in subjects; RR = 2.3, 95%CI: 0.9, 6.0) to three times (if examined in eyes; RR = 3.1, 95%CI: 1.1, 8.50) higher in males than in females.

- The progression of CSME and VTR was approximately the same in both males and females. (If CSME was examined in subjects RR = 1.2, 95%CI: 0.11, 12.4; if examined in eyes: RR = 1.5, 95% CI: 0.14, 17.3. The respective figures for VTR were RR = 0.8, 95%CI: 0.08, 7.2 in subjects and RR = 0.9, 95%CI: 0.097, 8.8 in eyes).

- The progression of subject retinopathy from not presenting with the condition at the beginning of the study to having the condition at the end of the study was 5.6% in both living environments. The progression of maculopathy, CSME and VTR could not be compared due to the lack of observations in the non-traditional environment.

- While the progression of subject retinopathy from not presenting with the condition at the beginning of the study to having the condition at the end of the study was approximately the same in smaller communities

and in major centers (if examined in subjects RR= 1.5, 95% CI:0.46, 5.0; if examined in eyes: RR = 1.02, 95%CI: 0.3,3.48), the progression of subject maculopathy, CSME and VTR was much higher in smaller communities than in major centers. The respective risk ratios and 95% CIs were in subjects and eyes for maculopathy RR= 4.8, 95% CI:0.93, 25.2 and RR = 4.9, 95%CI: 1.1, 21.3; for VTR RR= 7.3, 95% CI:0.78, 68 and RR = 6.1, 95%CI: 0.6, 58). All newly developed CSME were living in smaller communities, so RR could not be calculated.

Chapter 8

8. Description of visual acuity in the Aboriginal diabetic population of the Katherine Region

8.1 Overview of presentation of results

Clinical practice in Aboriginal communities has shown that the ocular complications of diabetes coexist with other ocular conditions. If corrections for the effect on visual acuity of ocular pathologies other than diabetic retinopathy are made, we can better estimate the effect of diabetic retinopathy itself. It is important to establish the major causes of impaired vision in this diabetic population, since it will have an impact on planning health services in the region.

It is well established that a visual acuity of 6/6 is not inconsistent with clinically significant macula edema (CSME)(Early Treatment Diabetic Retinopathy Study Group, 1985). Clinical practice also shows that macula edema is sometimes the only feature of CSME, which is undetectable with a single non - stereoscopic photograph. If visual acuity is still good in subjects with CSME, measuring visual acuity in subjects with CSME may add little (or no) information to the retinal photographs of this Aboriginal population. Therefore, examination of visual acuity in subjects with CSME will influence recommendations for a screening program.

The following characteristics will be described:

- best corrected subject vision

- impaired vision, monocular and binocular blindness
- ocular pathology of eyes with impaired vision and blindness
- the effect of diabetic retinopathy, maculopathy, CSME and VTR on vision.

In 1993, of the 234 diabetic subjects in the communities, there were complete records of visual acuity in 230 subjects, in one subject the record was incomplete. Of those with missing records there were 3 females, one in the 40-49, 50-59 and 60-69 age brackets each. The only male with a missing record was in the 50-59 year age bracket.

In 1996, of the 243 diabetic subjects in the communities, there were complete records of visual acuity in 237 subjects, in one subject the record incomplete. Of those with missing records there were 4 females, one in the 40-49, and 60-69 age brackets each and two in the 70+ age bracket. The only male with missing records was in the 50-59 year age bracket.

8.2 Description of visual acuity in 1993 and in 1996

This section describes visual impairment in the KRDRS by sex and age at the time of examination. The analysis of visual acuity by living environment has also been carried out, but the data are not presented in this thesis. This is due to the low frequencies in the non-traditional living environment and the low frequency of visual impairment. (There was one subject with visual impairment in both 1993 and in 1996, and one subject with monocular blindness but with adequate subject vision in 1996.)

In this section all tables present data for vision after correction.

Tables 8.2.1 – 8.2.8 show that:

- In 1993, after correction, 52% of subjects had 6/6 vision compared with 61% in 1996. This is due to the increase in the proportion of males with perfect vision (from 50% in 1993 to 73% in 1996) (Table 8.2.1).

- Table 8.2.5 summarizes the pivotal results regarding visual impairment in the KRDRS. It shows that visual impairment in the study population was much higher than in the non-Aboriginal Australian population and as high (or higher in 1993) as that in previous studies from the Aboriginal Australian population (see discussion).

- In 1993, of those with visual impairment, every seventh subject was monocularly blind and every third subject was binocularly blind. In 1996,

of those with visual impairment, every ninth subject was monocularly blind and every third person was binocularly blind (Tables 8.2.2 – 8.2.5).

- Of those with monocular blindness, 76% (13/17) had adequate vision in their other eye in 1993. The corresponding figure for 1996 was 86% (12/14) (Table 8.2.2).

Stocks found that 10.6% (17/163) of the Aboriginal Australian population in the 60+ age bracket was blind (Stocks, 1997). 85% of blind Aboriginal persons in their study were in the 60+ age bracket. Therefore, examination of visual impairment in this age group is warranted.

- The proportion of subjects with impaired vision, monocular blindness and binocular blindness increased with age in both 1993 and in 1996 (Tables 8.2.6 – 8.2.8).

- The summary of visual impairment in the 60+ age group is presented in Table 8.2.9. It shows that the prevalence of impaired vision, monocular and binocular blindness was much higher in females than in males in 1993. In 1996 these prevalence figures were lower in the female population. Research suggests that gender roles in society also have implications for knowledge about access to health resources (Brilliant, 1985), with women having less access than men to preventive and curative services. One possible explanation of the change in the prevalences by sex (especially in the 60+ age group) may be that the active search for the visually impaired in the KRDRS has overcome the barriers that prevented women to access ophthalmic services.

- In the 1996 study population, the overall prevalences of impaired vision, monocular and binocular blindness was less than that in 1993.

best corrected vision of the person	year and gender					
	1993			1996		
	female	male	TOTAL	female	male	TOTAL
<6/60	10	1	11	2	1	3
	90.91	9.09	100.00	66.67	33.33	100.00
	6.10	1.43	4.70	1.27	1.16	1.23
6/60	6	1	7			
	85.71	14.29	100.00	.	.	.
	3.66	1.43	2.99	.	.	.
6/36	3	2	5			
	60.00	40.00	100.00	.	.	.
	1.83	2.86	2.14	.	.	.
6/24	2	2	4	4	2	6
	50.00	50.00	100.00	66.67	33.33	100.00
	1.22	2.86	1.71	2.55	2.33	2.47
6/18	12	4	16	8	2	10
	75.00	25.00	100.00	80.00	20.00	100.00
	7.32	5.71	6.84	5.10	2.33	4.12
6/15				1		1
	.	.	.	100.00	.	100.00
	.	.	.	0.64	.	0.41
6/12	10	4	14	10	4	14
	71.43	28.57	100.00	71.43	28.57	100.00
	6.10	5.71	5.98	6.37	4.65	5.76
6/9	22	15	37	41	11	52
	59.46	40.54	100.00	78.85	21.15	100.00
	13.41	21.43	15.81	26.11	12.79	21.40
6/7.5	9	5	14	2	2	4
	64.29	35.71	100.00	50.00	50.00	100.00
	5.49	7.14	5.98	1.27	2.33	1.65
6/6	87	35	122	85	63	148
	71.31	28.69	100.00	57.43	42.57	100.00
	53.05	50.00	52.14	54.14	73.26	60.91
missing	3	1	4	4	1	5
	75.00	25.00	100.00	80.00	20.00	100.00
	1.83	1.43	1.71	2.55	1.16	2.06
TOTAL	164	70	234	157	86	243
	70.09	29.91	100.00	64.61	35.39	100.00
	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.2.1 Distribution of best corrected subject vision in 1993 and in 1996. Entries are frequencies, row and column percentages.

year and subject vision with correction	monocular blindness: no — binocular blindness: no	no — binocular blindness: yes	no — Total	yes — binocular blindness: no	yes — binocular blindness: yes	yes — Total	Total: no	Total: yes	Total: Total
1993									
impaired	12	11	23	4	.	4	16	11	27
adequate	190	.	190	13	.	13	203	.	203
Total	202	11	213	17	.	17	219	11	230
1996									
impaired	4	3	7	2	.	2	6	3	9
adequate	217	.	217	12	.	12	229	.	229
Total	221	3	224	14	.	14	235	3	238

Table 8.2.2 Distribution of adequate and impaired subject vision, monocular and binocular blindness in 1993 and in1996.
Entries are frequencies, row and column percentages.

gender and subject vision with correction	monocular blindness: no — binocular blindness: no	no — binocular blindness: yes	no — Total	yes — binocular blindness: no	yes — binocular blindness: yes	yes — Total	Total: no	Total: yes	Total: Total
female									
impaired	9	10	19	2	.	2	11	10	21
adequate	131	.	131	9	.	9	140	.	140
Total	140	10	150	11	.	11	151	10	161
male									
impaired	3	1	4	2	.	2	5	1	6
adequate	59	.	59	4	.	4	63	.	63
Total	62	1	63	6	.	6	68	1	69

Table 8.2.3 Distribution of adequate and impaired subject vision, monocular and binocular blindness by sex in 1993. Entries are frequencies.

gender and subject vision with correction	monocular blindness: no — binocular blindness: no	no — binocular blindness: yes	no — Total	yes — binocular blindness: no	yes — binocular blindness: yes	yes — Total	Total: no	Total: yes	Total: Total
female									
impaired	3	2	5	1	.	1	4	2	6
adequate	141	.	141	6	.	6	147	.	147
Total	144	2	146	7	.	7	151	2	153
male									
impaired	1	1	2	1	.	1	2	1	3
adequate	76	.	76	6	.	6	82	.	82
Total	77	1	78	7	.	7	84	1	85

Table 8.2.4 Distribution of adequate and impaired subject vision, monocular and binocular blindness by sex in 1996. Entries are frequencies.

	1993	1996	Risk ratio	95% CI	P value
Impaired vision					
male	6/69 8.7%	3/85 3.5%	0.4	0.12, 1.4	0.15
female	21/161 13.0%	6/153 3.9%	0.3	0.13, 0.7	0.007
Total	27/130 11.7%	9/238 3.8%	0.3	0.2, 0.7	0.002
Monocular blindness					
male	7/69 10.1%	6/85 7.1%	0.9	0.4, 2.4	0.92
female	11/161 6.8%	6/153 3.9%	0.67	0.31, 1.4	0.30
Total	18/230 7.8%	12/238 5.0%	0.8	-0.4, 1.4	0.45
Binocular blindness					
male	1/69 1.4%	1/85 1.2%	0.8	0.6, 1.03	0.09
female	10/161 6.2%	2/153 1.3%	0.2	0.05, 0.96	0.045
Total	11/230 4.8%	3/238 1.3%	0.3	0.08, 0.86	0.03

Table 8.2.5 Summary of visual impairment in the KRDRS. Entries are proportions, percentages risk ratios 95% confidence intervals (CI) and P values.

age at the time of examination	year and subject vision with correction					
	1993			1996		
	impaired	adequate	TOTAL	impaired	adequate	TOTAL
10 - 19		2	2		1	1
	.	100.00	100.00	.	100.00	100.00
	.	0.99	0.87	.	0.44	0.42
20 - 29	1	11	12		17	17
	8.33	91.67	100.00	.	100.00	100.00
	3.70	5.42	5.22	.	7.46	7.17
30 - 39	1	37	38	1	43	44
	2.63	97.37	100.00	2.27	97.73	100.00
	3.70	18.23	16.52	11.11	18.86	18.57
40 - 49	2	60	62	1	58	59
	3.23	96.77	100.00	1.69	98.31	100.00
	7.41	29.56	26.96	11.11	25.44	24.89
50 - 59	5	48	53		51	51
	9.43	90.57	100.00	.	100.00	100.00
	18.52	23.65	23.04	.	22.37	21.52
60 - 69	6	36	42	5	45	50
	14.29	85.71	100.00	10.00	90.00	100.00
	22.22	17.73	18.26	55.56	19.74	21.10
≥70	12	9	21	2	13	15
	57.14	42.86	100.00	13.33	86.67	100.00
	44.44	4.43	9.13	22.22	5.70	6.33
TOTAL	27	203	230	9	228	237
	11.74	88.26	100.00	3.80	96.20	100.00
	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.2.6 Distribution of adequate and impaired subject vision (after correction) by age in 1993 and in 1996. Entries are frequencies, row and column percentages.

age at the time of examination	year and monocular blindness					
	1993			1996		
	no	yes	TOTAL	no	yes	TOTAL
10 - 19	2		2	1		1
	100.00		100.00	100.00		100.00
	0.92		0.85	0.44		0.41
20 - 29	12		12	17		17
	100.00		100.00	100.00		100.00
	5.53		5.13	7.46		7.02
30 - 39	37	1	38	44		44
	97.37	2.63	100.00	100.00		100.00
	17.05	5.88	16.24	19.30		18.18
40 - 49	61	2	63	57	3	60
	96.83	3.17	100.00	95.00	5.00	100.00
	28.11	11.76	26.92	25.00	21.43	24.79
50 - 59	50	5	55	49	3	52
	90.91	9.09	100.00	94.23	5.77	100.00
	23.04	29.41	23.50	21.49	21.43	21.49
60 - 69	38	5	43	46	5	51
	88.37	11.63	100.00	90.20	9.80	100.00
	17.51	29.41	18.38	20.18	35.71	21.07
≥70	17	4	21	14	3	17
	80.95	19.05	100.00	82.35	17.65	100.00
	7.83	23.53	8.97	6.14	21.43	7.02
TOTAL	217	17	234	228	14	242
	92.74	7.26	100.00	94.21	5.79	100.00
	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.2.7 Distribution of monocular blindness by age in 1993 and in 1996. Entries are frequencies, row and column percentages.

age at the time of examination	year and binocular blindness with correction					
	1993			1996		
	no	yes	TOTAL	no	yes	TOTAL
10 - 19	2		2	1		1
	100.00	.	100.00	100.00	.	100.00
	0.91	.	0.87	0.43	.	0.42
20 - 29	12		12	17		17
	100.00	.	100.00	100.00	.	100.00
	5.48	.	5.22	7.26	.	7.17
30 - 39	38		38	44		44
	100.00	.	100.00	100.00	.	100.00
	17.35	.	16.52	18.80	.	18.57
40 - 49	62		62	58	1	59
	100.00	.	100.00	98.31	1.69	100.00
	28.31	.	26.96	24.79	33.33	24.89
50 - 59	52	1	53	51		51
	98.11	1.89	100.00	100.00	.	100.00
	23.74	9.09	23.04	21.79	.	21.52
60 - 69	39	3	42	49	1	50
	92.86	7.14	100.00	98.00	2.00	100.00
	17.81	27.27	18.26	20.94	33.33	21.10
≥70	14	7	21	14	1	15
	66.67	33.33	100.00	93.33	6.67	100.00
	6.39	63.64	9.13	5.98	33.33	6.33
TOTAL	219	11	230	234	3	237
	95.22	4.78	100.00	98.73	1.27	100.00
	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.2.8 Distribution of binocular blindness (after correction) by age in 1993 and in 1996. Entries are frequencies, row and column percentages.

	1993	1996	Risk ratio	95% CI	P value
Impaired vision					
male	2/18 11.1%	3/27 11.1%	0.97	-.3, 3.6	0.96
female	16/45 35.6%	4/38 10.5%	0.3	0.1, 0.8	0.02
Total	18/63 28.6%	7/65 10.8%	0.4	0.2, 0.8	0.01
Monocular blindness					
male	2/18 11.1%	3/27 14.8%	1.3	0.4, 4.9	0.67
female	7/46 15.2%	4/41 9.7%	0.6	0.2, 1.9	0.42
Total	9/63 14.3%	6/65 9.2%	0.8	0.4, 1.8	0.63
Binocular blindness					
male	1/18 5.6%	1/27 3.7%	0.6	0.4, 1.0	0.37
female	9/45 20.0%	1/38 2.6%	0.1	0.02, 1.05	0.06
Total	10/63 15.9%	2/65 3.1%	0.2	0.05, 0.76	0.02

Table 8.2.9. Impaired vision, monocular and binocular blindness in the KRDRS in 1993 and in 1996 in the 60+ age group. Entries are proportions, percentages risk differences (coef), 95% confidence intervals (CI) and P values.

8.3 Description of ocular pathology in eyes with impaired vision

Tables 8.3.1 – 8.3.11 show that:

- In 1993 the main ocular pathology causing impaired vision was cataract (67% (54/81)), with diabetic retinopathy responsible for only 6% (5/81) of impaired vision. The respective figures for 1996 were 58% (26/45) and 13% (6/45) (Table 8.3.1). (In this table "phacolytic glaucoma" was also counted under "cataract" due to the obvious underlying pathology.)

- In 1993, in eyes of females, 70% (40/57) of impaired vision was due to cataract and diabetic retinopathy was responsible for only 7% (4/57). The respective figures in males were 58% (14/24) and 4% (1/24) (Table 8.3.1).

- In 1996, in eyes of females, 66% (19/29) of impaired vision was due to cataract with diabetic retinopathy responsible for only 21% (6/29). In males, 44% (7/16) of eyes with impaired vision was due to cataract. There was no eye with impaired vision due to diabetic retinopathy among males in 1996 (Table 8.3.1).

- In both 1993 and in 1996, over 90% of visual impairment could have been prevented (Table 8.3.1).

- In both 1993 and 1996, in blind eyes, the main cause of blindness was cataract (58% (23/40) and 39% (7/18) of cases, respectively). In blind

eyes, blindness was due to diabetic retinopathy in 5% (2/40) of eyes in 1993 compared with 11% (2/18) of eyes in 1996 (Table 8.3.2).

- In 1993 and in 1996, in blind eyes, cataract was the single main cause of blindness in both genders. In 1993, in females, blindness was due to cataract in 65% (20/31) of cases (blind eyes) compared with 33% (3/9) in males. In 1996, in females, blindness was due to cataract in 60%(6/10) of cases (blind eyes), compared with 12.5% (1/8) in males (Table 8.3.2).

- Blindness in females was due to diabetic retinopathy in 6% (6/10) of cases (blind eyes) in 1993 compared with 20% (2/10) in 1996. There were no eyes blind due to diabetic retinopathy in males in either 1993 or in 1996 (Table 8.3.2).

- Almost 100% of blindness in this diabetic population is preventable (Table 8.3.2).

- Tables 8.3.3 and 8.3.4 show the ocular pathology behind binocular blindness in 1993 and in 1996. It can be clearly seen that all cases of binocular blindness could have been prevented.

- Table 8.3.5 shows that in 1993 47% (8/17) of cases of monocular blindness was due to cataract. The respective figure for 1996 was 43% (6/14). In both years nearly all cases of monocular blindness could have been prevented.

- Tables 8.3.6 – 8.3.7 show the effect of glasses on vision in eyes in 1993 and in 1996. The relevant findings for impaired vision and

blindness in eyes are further summarized in Tables 8.3.8 and 8.3.9. It is remarkable that in 1993 in this diabetic population only 6% (5/86) of those eyes with impaired vision could be improved to adequate vision. The respective figures for 1996 were 45% (37/82). Of the blind eyes, only 2.4% (1/41) could be improved to "no blindness" in 1993. The respective figure for 1996 was 18% (4/22).

- Tables 8.3.6 and 8.3.7 show that in 1993, in 3.7% (17/458) of eyes the vision could be improved one line, in 2.6% (12/458) of eyes with three lines after correction. The respective figures for 1996 were 32.6% (154/473) and 13.5% (63/473).

Examining the eyes of subjects provides information on the burden of disease in the community and will determine the need for intervention. It does not however indicate the benefits of refraction at the subject level. Therefore, Tables 8.3.10 – 8.3.11 show the effect of glasses on vision in subjects in 1993 and in 1996. The relevant findings for impaired vision and blindness in subjects are further summarized in Tables 8.3.12 and 8.3.13.

- Tables 8.3.10 – 8.3.13 show that in 1993, 7% (2/29) of those with impaired vision could be improved to adequate vision. The respective figures in 1996 were 55% (11/20). In 1993, 8.3% (1/12) of blind subjects could be improved to the level of no blindness. The equivalent figure for 1996 was 25% (1/4).

- Tables 8.3.10 – 8.3.11 show that in 1993 in 3.5% (8/229) of subjects the vision could be improved one line, in 1.3% (3/229) of subjects with

three lines after correction. The corresponding figures for 1996 were 30.0% (71/237) and 7.6% (18/237).

Tables 8.3.6 – 8.3.13 show the importance to report visual acuity in subjects and eyes. Reporting the effect of refraction on visual acuity in the no blindness → blindness and impaired → adequate vision categories in eyes alone would have underestimated its significance in the population. On the other hand, reporting the effect of refraction on vision in eyes, where vision could be improved with three or more lines, would have overestimated its significance in the population.

- Table 8.3.14 shows the ocular pathologies of eyes where vision could be improved from impaired vision to adequate vision with glasses in 1993 and in 1996.

- Table 8.3.15 shows the ocular pathologies of eyes that remained blind after correction was provided in 1993 and in 1996.

main diagnosis (ICD9)	year and gender 1993 female	1993 male	1993 TOTAL	1996 female	1996 male	1996 TOTAL
adherent leucoma	.	1 100.00 4.17	1 100.00 1.23	.	1 100.00 6.25	1 100.00 2.22
after-cataract obscuring vision	2 66.67 3.51	1 33.33 4.17	3 100.00 3.70	.	1 100.00 6.25	1 100.00 2.22
blind hypotensive eye	2 100.00 3.51	.	2 100.00 2.47	.	2 100.00 12.50	2 100.00 4.44
central opacity, cornea	.	.	.	1 50.00 3.45	1 50.00 6.25	2 100.00 4.44
chorioretinal scar nos	1 100.00 1.75	.	1 100.00 1.23	.	.	.
chorioretinitis nos nec	1 100.00 1.75	.	1 100.00 1.23	.	.	.
coloboma of optic disc	1 100.00 1.75	.	1 100.00 1.23	1 100.00 3.45	.	1 100.00 2.22
contusion of eyeball	1 100.00 1.75	.	1 100.00 1.23	.	.	.
corneal opacity nos	3 100.00 5.26	.	3 100.00 3.70	.	.	.
diabetic cataract	.	2 100.00 8.33	2 100.00 2.47	.	1 100.00 6.25	1 100.00 2.22
diabetic retinopathy nos	2 66.67 3.51	1 33.33 4.17	3 100.00 3.70	5 100.00 17.24	.	5 100.00 11.11
glaucoma nos	.	.	.	2 100.00 6.90	.	2 100.00 4.44
glaucoma with ocular trauma	.	1 100.00 4.17	1 100.00 1.23	.	.	.

Table 8.3.1. The main ocular pathology in eyes where the vision remained impaired after correction in 1993 and in 1996 (continued next page). Entries are frequencies, row and column and percentages (nos = not otherwise specified, nec = not elsewhere classified).

main diagnosis (ICD9)	year and gender: 1993 female	1993 male	1993 TOTAL	1996 female	1996 male	1996 TOTAL
macular degeneration nos	1		1			
	100.00	.	100.00	.	.	.
	1.75	.	1.23	.	.	.
mature cataract	12		12	5		5
	100.00	.	100.00	100.00	.	100.00
	21.05	.	14.81	17.24	.	11.11
mydriasis not due to mydriatic	.	1	1			
	.	100.00	100.00	.	.	.
	.	4.17	1.23	.	.	.
old detachment, partial		1	1		1	1
	.	100.00	100.00	.	100.00	100.00
	.	4.17	1.23	.	6.25	2.22
optic atrophy nos		1	1		1	1
	.	100.00	100.00	.	100.00	100.00
	.	4.17	1.23	.	6.25	2.22
periph progess pterygium	1	2	3			
	33.33	66.67	100.00	.	.	.
	1.75	8.33	3.70	.	.	.
phacolytic glaucoma		1	1			
	.	100.00	100.00	.	.	.
	.	4.17	1.23	.	.	.
posterior subcapsular senile cataract	8	6	14	5	1	6
	57.14	42.86	100.00	83.33	16.67	100.00
	14.04	25.00	17.28	17.24	6.25	13.33
proliferative diabetic retinopathy	2		2	1		1
	100.00	.	100.00	100.00	.	100.00
	3.51	.	2.47	3.45	.	2.22
retinal detachment nos		1	1		1	1
	.	100.00	100.00	.	100.00	100.00
	.	4.17	1.23	.	6.25	2.22
senile cataract nec	2		2	5	1	6
	100.00	.	100.00	83.33	16.67	100.00
	3.51	.	2.47	17.24	6.25	13.33
senile nuclear cataract	18	5	23	4	4	8
	78.26	21.74	100.00	50.00	50.00	100.00
	31.58	20.83	28.40	13.79	25.00	17.78
subluxation of lens					1	1
	.	.	.	.	100.00	100.00
	.	.	.	.	6.25	2.22
TOTAL	57	24	81	29	16	45
	70.37	29.63	100.00	64.44	35.56	100.00
	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.3.1 (cont) The main ocular pathology in eyes where the vision remained impaired after correction in 1993 and in 1996. Entries are frequencies, row and column percentages (nos = not otherwise specified, nec = not elsewhere classified).

main diagnosis (ICD9)	year and gender: 1993 female	1993 male	1993 TOTAL	1996 female	1996 male	1996 TOTAL
adherent leucoma	.	1 100.00 11.11	1 100.00 2.50	.	1 100.00 12.50	1 100.00 5.56
after-cataract obscuring vision	2 100.00 6.45	.	2 100.00 5.00	.	1 100.00 12.50	1 100.00 5.56
blind hypotensive eye	2 100.00 6.45	.	2 100.00 5.00	.	2 100.00 25.00	2 100.00 11.11
central opacity, cornea	.	.	.	1 100.00 10.00	.	1 100.00 5.56
coloboma of optic disc	1 100.00 3.23	.	1 100.00 2.50	1 100.00 10.00	.	1 100.00 5.56
contusion of eyeball	1 100.00 3.23	.	1 100.00 2.50	.	.	.
corneal opacity nos	3 100.00 9.68	.	3 100.00 7.50	.	.	.
diabetic retinopathy nos	.	.	.	1 100.00 10.00	.	1 100.00 5.56
glaucoma with ocular trauma	.	1 100.00 11.11	1 100.00 2.50	.	.	.
mature cataract	11 100.00 35.48	.	11 100.00 27.50	2 100.00 20.00	.	2 100.00 11.11

Table 8.3.2 The main ocular pathology in eyes that remained blind after correction in 1993 and in 1996 (continued next page). Entries are frequencies, row and column percentages (nos = not otherwise specified, nec = not elsewhere classified).

main diagnosis (ICD9)	year and gender: 1993 female	1993 male	1993 TOTAL	1996 female	1996 male	1996 TOTAL
mydriasis not due to mydriatic	.	1 100.00 11.11	1 100.00 2.50	.	.	.
old detachment, partial	.	1 100.00 11.11	1 100.00 2.50	.	1 100.00 12.50	1 100.00 5.56
optic atrophy nos	.	1 100.00 11.11	1 100.00 2.50	.	.	.
phacolytic glaucoma	.	1 100.00 11.11	1 100.00 2.50	.	.	.
posterior subcapsular senile cataract	2 100.00 6.45	.	2 100.00 5.00	1 100.00 10.00	.	1 100.00 5.56
proliferative diabetic retinopathy	2 100.00 6.45	.	2 100.00 5.00	1 100.00 10.00	.	1 100.00 5.56
retinal detachment nos	.	1 100.00 11.11	1 100.00 2.50	.	1 100.00 12.50	1 100.00 5.56
senile cataract nec	2 100.00 6.45	.	2 100.00 5.00	2 100.00 20.00	.	2 100.00 11.11
senile nuclear cataract	5 71.43 16.13	2 28.57 22.22	7 100.00 17.50	1 50.00 10.00	1 50.00 12.50	2 100.00 11.11
subluxation of lens	.	.	.	.	1 100.00 12.50	1 100.00 5.56
TOTAL	31 77.50 100.00	9 22.50 100.00	40 100.00 100.00	10 55.56 100.00	8 44.44 100.00	18 100.00 100.00

Table 8.3.2 (cont) The main ocular pathology in eyes that remained blind after correction in 1993 and in 1996. Entries are frequencies, row and column percentages (nos = not otherwise specified, nec = not elsewhere classified).

Main Diagnosis Right Eye (ICD9)	Main Diagnosis Left Eye (ICD9) blind hypotensive eye	mature cataract	prolif diabetic retinopathy	senile cataract nec	senile nuclear cataract	Total
adherent leucoma	.	.	.	.	1	1
aftr-catar obscur vision	.	1	.	.	.	1
corneal opacity nos	1	.	.	.	1	2
mature cataract	.	3	.	.	.	3
prolif diab retinopathy	.	.	1	.	.	1
senile cataract nec	.	.	.	1	.	1
senile nuclear cataract	.	.	.	.	2	2
Total	1	4	1	1	4	11

Table 8.3.3 Distribution of diagnoses between the two eyes in subjects that remained binocularly blind after correction in 1993. Entries are frequencies (aftr-catar obscur = after cataract obscuring, prolif diab = proliferative diabetic, nos = not otherwise specified, nec = not elsewhere classified).

Main Diagnosis Right Eye (ICD9)	Main Diagnosis Left Eye prolif diabetic retinopathy	senile cataract nec	senile nuclear cataract	Total
adherent leucoma	.	.	1	1
diabetic retinopathy nos	1	.	.	1
senile cataract nec	.	1	.	1
Total	1	1	1	3

Table 8.3.4 Distribution of diagnoses between the two eyes in subjects that remained binocularly blind after correction in 1996. Entries are frequencies (nos = not otherwise specified, nec = not elsewhere classified).

main diagnosis (ICD9)	year and gender: 1993 female	1993 male	1993 TOTAL	1996 female	1996 male	1996 TOTAL
aftr-catar obscur vision	1 9.09	.	1 5.88	.	1 14.29	1 7.14
blind hypotensive eye	1 9.09	.	1 5.88	.	2 28.57	2 14.29
central opacity, cornea	.	.	.	1 14.29	.	1 7.14
coloboma of optic disc	1 9.09	.	1 5.88	1 14.29	.	1 7.14
contusion of eyeball	1 9.09	.	1 5.88	.	.	.
corneal opacity nos	1 9.09	.	1 5.88	.	.	.
glaucoma w ocular trauma	.	1 16.67	1 5.88	.	.	.
mature cataract	4 36.36	.	4 23.53	3 42.86	.	3 21.43
mydriasis not d/t mydrtc	.	1 16.67	1 5.88	.	.	.
old detachment, partial	.	1 16.67	1 5.88	.	1 14.29	1 7.14
optic atrophy nos	.	1 16.67	1 5.88	.	.	.
phacolytic glaucoma	.	1 16.67	1 5.88	.	.	.
post subcap senile catar	2 18.18	.	2 11.76	1 14.29	.	1 7.14
retinal detachment nos	.	.	.	.	1 14.29	1 7.14
senile cataract nec	.	.	.	.	1 14.29	1 7.14
senile nuclear cataract	.	1 16.67	1 5.88	1 14.29	.	1 7.14
subluxation of lens	.	.	.	.	1 14.29	1 7.14
TOTAL	11 100.00	6 100.00	17 100.00	7 100.00	7 100.00	14 100.00

Table 8.3.5 The main ocular pathology in blind eyes in cases of monocular blindness. Entries are frequencies and column percentages..

uncorrected vision of the eye	best corrected vision of the eye									
	<6/60	6/60	6/36	6/24	6/18	6/12	6/9	6/7.5	6/6	Total
<6/60	40	.	.	.	.	.	1	.	.	41
6/60	.	16	1	.	.	.	.	.	.	17
6/36	.	.	14	.	.	2	2	.	.	18
6/24	.	.	.	10	.	.	.	.	.	10
6/18	.	.	.	.	29	.	3	.	2	34
6/12	.	.	.	.	.	32	4	.	1	37
6/9	.	.	.	.	.	.	58	.	1	59
6/7.5	.	.	.	.	.	.	.	32	.	32
6/6	.	.	.	.	.	.	.	.	210	210
Total	40	16	15	10	29	34	68	32	214	458

Table 8.3.6 The effect of glasses on vision in eyes in 1993. Entries are frequencies.

uncorrected vision of the eye	best corrected vision of the eye										
	<6/60	6/60	6/36	6/24	6/18	6/15	6/12	6/9	6/7.5	6/6	Total
<6/60	18	2	1	1	.	.	.	.	.	.	22
6/60	.	6	2	3	1	.	2	2	.	.	16
6/36	.	.	3	4	3	.	5	3	1	.	19
6/24	.	.	.	5	5	.	1	9	.	5	25
6/18	.	.	.	.	13	2	13	11	3	6	48
6/12	.	.	.	.	.	.	8	14	1	12	35
6/9	.	.	.	.	.	.	.	63	1	30	94
6/7.5	.	.	.	.	.	.	.	.	3	11	14
6/6	.	.	.	.	.	.	.	.	.	200	200
Total	18	8	6	13	22	2	29	102	9	264	473

Table 8.3.7 The effect of glasses on vision in eyes in 1996. Entries are frequencies.

vision of the eye without correction	year and vision of the eye with correction					
	1993			1996		
	impaired	adequate	TOTAL	impaired	adequate	TOTAL
impaired	81	5	86	45	37	82
adequate		372	372		391	391
TOTAL	81	377	458	45	428	473

Table 8.3.8 The effect of glasses on impaired vision in eyes in 1993 and in 1996. Entries are frequencies.

blind eye without correction	year and blind eye with correction					
	1993			1996		
	no	yes	TOTAL	no	yes	TOTAL
no	417		417	451		451
yes	1	40	41	4	18	22
TOTAL	418	40	458	456	18	473

Table 8.3.9 The effect of glasses on blindness in eyes in 1993 and in 1996. Entries are frequencies.

best uncorrected vision of the subject	best corrected vision of the person								
	<6/60	6/60	6/36	6/24	6/18	6/12	6/9	6/7.5	6/6	Total
<6/60	11	.	.	.	.	.	1	.	.	12
6/60	.	7	.	.	.	.	.	.	.	7
6/36	.	.	5	.	.	.	1	.	.	6
6/24	.	.	.	4	.	.	.	.	.	4
6/18	.	.	.	.	16	.	.	.	1	17
6/12	.	.	.	.	.	14	3	.	1	18
6/9	.	.	.	.	.	.	31	.	1	32
6/7.5	.	.	.	.	.	.	.	14	.	14
6/6	.	.	.	.	.	.	.	.	119	119
Total	11	7	5	4	16	14	36	14	122	229

Table 8.3.10 The effect of glasses on subject vision in 1993. Entries are frequencies.

best uncorrected vision of the subject	best corrected vision of the person								
	<6/60	6/24	6/18	6/15	6/12	6/9	6/7.5	6/6	Total
<6/60	3	1	.	.	.	.	.	.	4
6/60	.	2	1	.	.	1	.	.	4
6/36	.	1	1	.	.	2	1	.	5
6/24	.	2	1	.	.	3	.	1	7
6/18	.	.	7	1	9	6	1	2	26
6/12	.	.	.	.	5	9	.	5	19
6/9	.	.	.	.	.	30	.	17	47
6/7.5	.	.	.	.	.	.	2	5	7
6/6	.	.	.	.	.	.	.	118	118
Total	3	6	10	1	14	51	4	148	237

Table 8.3.11 The effect of glasses on subject vision in 1996. Entries are frequencies.

subject vision without correction	best corrected subject vision					
	1993			1996		
	impaired	adequate	TOTAL	impaired	adequate	TOTAL
impaired	27	2	29	9	11	20
adequate	0	200	200	0	217	217
TOTAL	27	202	229[1]	9	228	237[2]

Table 8.3.12 The effect of glasses on impaired vision in subjects in 1993 and in 1996. Entries are frequencies.

blind eye without correction	year and blind eye with correction					
	1993			1996		
	no	yes	TOTAL	no	yes	TOTAL
no	218	0	218	234	0	234
yes	1	11	12	1	3	4
TOTAL	219	11	230[1]	235	3	238[2]

[1,2] In one subject the record of the "adequate vision" was missing and therefore the subject was omitted from the denominator in Table 8.3.8. This subject had records in the blindness category with and without correction, therefore, the subject is included in the denominator in Table 8.3.9. This explains the different denominators.

Table 8.3.13 The effect of glasses on blindness in subjects in 1993 and in 1996. Entries are frequencies.

main diagnosis (ICD9)	year 1993	1996
aftr-catar obscur vision		2
amblyopia nos		1
aphakia	1	
chorioretinal scar nos		2
corneal opacity nos		2
diabetic retinopathy nos	1	5
drusen (degenerative)		1
myopia		7
post subcap senile catar	2	4
screening-eye cond nec		1
senile cataract nec		2
senile entropion		1
senile nuclear cataract	1	9
TOTAL	5	37

Table 8.3.14 Ocular pathologies of eyes where vision could be improved from impaired vision to adequate vision with glasses in 1993 and in 1996. Entries are frequencies (aftr-catar obscur = after cataract obscuring, nos = not otherwise specified, post subcap catar = posterior subcapsular cataract, nec = not elsewhere classified).

main diagnosis (ICD9)	year 1993	1996
aphakia	1	
glaucoma nos		2
mature cataract		1
senile cataract nec		1
TOTAL	1	4

Table 8.3.15 Ocular pathologies of eyes where vision could be improved from blindness to no blindness with glasses in 1993 and in 1996. Entries are frequencies.

8.4 Description of visual acuity in eyes with diabetic retinopathy

In this section the distribution of visual acuity and each of diabetic retinopathy, maculopathy, CSME and VTR is presented in two sets of tables. The first table describes the visual acuity in all eyes of this diabetic population, as examined in the mobile clinics. While this provides important information about the burden of ocular disease in this diabetic population, it cannot provide unconfounded information about the effect of diabetic retinopathy/maculopathy/CSME/VTR on visual acuity, since these eyes may be affected by other ocular pathologies in addition to diabetic retinopathy. The second table therefore uses as its denominator only those eyes in which no ocular pathology apart from diabetic retinopathy was present.

Tables 8.4.1 – 8.4.8 show that:

- In 1993 in eyes with diabetic retinopathy 16.9% (11/65) of eyes had impaired vision compared with 7.6% (26/344) in eyes without diabetic retinopathy. The respective figures for 1996 were 10.7% (9/84) and 6.3% (24/379) (table 8.4.1).

- In 1993 the proportion of eyes with blindness was 4.6% (3/65) in eyes with diabetic retinopathy compared with 2% (7/344) in eyes with no diabetic retinopathy. The respective figures for 1996 were 2.4% (2/84) and 1.8% (7/379) (table 8.4.1).

- Table 8.4.2 shows that when diabetic retinopathy is the only significant ocular pathology 50% (22/44) of eyes had a visual acuity of 6/6 and 75% (33/44) of eyes had a visual acuity of 6/9 or better in 1993. The respective findings were 53% (36/68) and 88% (60/68) (table 8.4.2).

- In 1993 16.3% (7/43) of eyes with diabetic maculopathy had impaired vision compared with 7.7% (28/364) in eyes without maculopathy. The respective figures for 1996 were 17.9% (7/39) and 5.9% (25/423) (Table 8.4.3).

- Table 8.4.4 shows that when diabetic retinopathy was the only significant ocular pathology, in 1993, 8.8% (3/34) of eyes with diabetic maculopathy had impaired vision compared with none without maculopathy. The respective figures for 1996 were 15% (5/33) for eyes with diabetic maculopathy and there were no eyes with impaired vision without diabetic maculopathy in 1996 either (table 8.4.4).

- When diabetic retinopathy was the only significant ocular pathology in 1993, 50% (17/34) of eyes with maculopathy had a visual acuity of 6/6 and 79% (27/34) had a visual acuity of 6/9 or better. The respective figures for 1996 were 45% (15/33) and 79% (26/33) (table 8.4.4).

- In 1993 in eyes with CSME only 24% (6/25) had impaired vision compared with 21% (29/382) in eyes with no CSME. The respective figures for 1996 were 23.8% (5/21) and 6.1% (27/441). Of all eyes in the population that have CSME 52% (13/25) had a vision of 6/9 or better in 1993. The respective figure for 1996 was 67% (14/21) (table 8.4.5).

- Table 8.4.6 shows that when diabetic retinopathy was the only significant ocular pathology, in 1993, only 15% (3/20) of eyes with CSME had impaired vision compared with 23.8% (5/21) in 1996 (table 8.4.6).

- When diabetic retinopathy was the only significant ocular pathology, in 1993 25% (5/20) of eyes had a visual acuity of 6/6 and 65% (13/20) of eyes had a visual acuity 6/9 or better. The respective figures for 1996 were 33% (7/21) and 67% (14/21) in 1996 (table 8.4.6).

- In 1993, 29% (8/28) of eyes with VTR had impaired vision compared with 8% (29/381) of eyes with no VTR. The respective figures for 1996 were 24% (6/25) and 6% (27/438). In 1993 the prevalence of blindness in eyes with VTR was 7% (2/28) compared with 2% (8/381) in eyes with no VTR. The respective figures for 1996 were 8% (2/25) compared with 2% (7/438) (table 8.4.7).

- Table 8.4.8 shows that when diabetic retinopathy was the only significant ocular pathology, in 1993 only 22% (5/23) of eyes with VTR had impaired vision compared with 24% (6/25) in 1996.

- When diabetic retinopathy was the only significant ocular pathology, of all eyes with VTR in 1993 22% of eyes (5/23) had a visual acuity of 6/6 and 57% (13/23) of eyes had a visual acuity of 6/9 or better. The respective findings for 1996 were 32% (8/25) and 68% (17/25) (table 8.4.8).

best corrected vision of the eye	year and diabetic retinopathy 1993						
	nil	mild	mod sev	severe	prolif	not vis	TOTAL
<6/60	7	1			2	30	40
	17.50	2.50	.	.	5.00	75.00	100.00
	2.03	1.96	.	.	66.67	58.82	8.70
6/60	6	1		2		7	16
	37.50	6.25	.	12.50	.	43.75	100.00
	1.74	1.96	.	50.00	.	13.73	3.48
6/36	7	1		1		6	15
	46.67	6.67	.	6.67	.	40.00	100.00
	2.03	1.96	.	25.00	.	11.76	3.26
6/24	6	2		1		1	10
	60.00	20.00	.	10.00	.	10.00	100.00
	1.74	3.92	.	25.00	.	1.96	2.17
6/18	17	4	1		1	6	29
	58.62	13.79	3.45	.	3.45	20.69	100.00
	4.94	7.84	14.29	.	33.33	11.76	6.30
6/15							
	.	.	.	.	.	.	.
	.	.	.	.	.	.	.
6/12	29	4	1				34
	85.29	11.76	2.94	.	.	.	100.00
	8.43	7.84	14.29	.	.	.	7.39
6/9	56	13				1	70
	80.00	18.57	.	.	.	1.43	100.00
	16.28	25.49	.	.	.	1.96	15.22
6/7.5	26	3	3				32
	81.25	9.38	9.38	.	.	.	100.00
	7.56	5.88	42.86	.	.	.	6.96
6/6	190	22	2				214
	88.79	10.28	0.93	.	.	.	100.00
	55.23	43.14	28.57	.	.	.	46.52
TOTAL	344	51	7	4	3	51	460
	74.78	11.09	1.52	0.87	0.65	11.09	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.4.1 Best corrected visual acuity by diabetic retinopathy in eyes in 1993 and in 1996 (continued next page). Entries are frequencies, row and column percentages.

best corrected vision of the eye	year and diabetic retinopathy 1996						
	nil	mild	mod sev	severe	prolif	not vis	TOTAL
<6/60	7		1		1	9	18
	38.89	.	5.56	.	5.56	50.00	100.00
	1.85	.	3.13	.	25.00	75.00	3.79
6/60	5		1			2	8
	62.50	.	12.50	.	.	25.00	100.00
	1.32	.	3.13	.	.	16.67	1.68
6/36	4		2				6
	66.67	.	33.33	.	.	.	100.00
	1.06	.	6.25	.	.	.	1.26
6/24	8	2	2			1	13
	61.54	15.38	15.38	.	.	7.69	100.00
	2.11	4.76	6.25	.	.	8.33	2.74
6/18	17	2	3				22
	77.27	9.09	13.64	.	.	.	100.00
	4.49	4.76	9.38	.	.	.	4.63
6/15	2						2
	100.00	.	.	.	.	.	100.00
	0.53	.	.	.	.	.	0.42
6/12	28	1					29
	96.55	3.45	.	.	.	.	100.00
	7.39	2.38	.	.	.	.	6.11
6/9	78	15	6	3	2		104
	75.00	14.42	5.77	2.88	1.92	.	100.00
	20.58	35.71	18.75	50.00	50.00	.	21.89
6/7.5	5	2	2				9
	55.56	22.22	22.22	.	.	.	100.00
	1.32	4.76	6.25	.	.	.	1.89
6/6	225	20	15	3	1		264
	85.23	7.58	5.68	1.14	0.38	.	100.00
	59.37	47.62	46.88	50.00	25.00	.	55.58
TOTAL	379	42	32	6	4	12	475
	79.79	8.84	6.74	1.26	0.84	2.53	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.4.1 (cont) Best corrected visual acuity by diabetic retinopathy in eyes in 1993 and in 1996. Entries are frequencies, row and column percentages.

best corrected vision of the eye	year and diabetic retinopathy 1993 mild	mod sev	severe	prolif	TOTAL
<6/60				2	2
	.	.	.	100.00	100.00
	.	.	.	66.67	4.55
6/36	1		1		2
	50.00	.	50.00	.	100.00
	3.13	.	50.00	.	4.55
6/24			1		1
	.	.	100.00	.	100.00
	.	.	50.00	.	2.27
6/18	1	1		1	3
	33.33	33.33	.	33.33	100.00
	3.13	14.29	.	33.33	6.82
6/12	2	1			3
	66.67	33.33	.	.	100.00
	6.25	14.29	.	.	6.82
6/9	6				6
	100.00	.	.	.	100.00
	18.75	.	.	.	13.64
6/7.5	2	3			5
	40.00	60.00	.	.	100.00
	6.25	42.86	.	.	11.36
6/6	20	2			22
	90.91	9.09	.	.	100.00
	62.50	28.57	.	.	50.00
TOTAL	32	7	2	3	44
	72.73	15.91	4.55	6.82	100.00
	100.00	100.00	100.00	100.00	100.00

Table 8.4.2 Best corrected visual acuity by stages of diabetic retinopathy in eyes without ocular pathology other than diabetic retinopathy in 1993 and in 1996 (continued next page). Entries are frequencies, row and column percentages.

best corrected vision of the eye	year and diabetic retinopathy 1996 mild	mod sev	severe	prolif	TOTAL
<6/60		1		1	2
	.	50.00	.	50.00	100.00
	.	3.85	.	25.00	2.94
6/60		1			1
	.	100.00	.	.	100.00
	.	3.85	.	.	1.47
6/36		1			1
	.	100.00	.	.	100.00
	.	3.85	.	.	1.47
6/24	1	1			2
	50.00	50.00	.	.	100.00
	3.13	3.85	.	.	2.94
6/18		2			2
	.	100.00	.	.	100.00
	.	7.69	.	.	2.94
6/9	11	4	3	2	20
	55.00	20.00	15.00	10.00	100.00
	34.38	15.38	50.00	50.00	29.41
6/7.5	2	2			4
	50.00	50.00	.	.	100.00
	6.25	7.69	.	.	5.88
6/6	18	14	3	1	36
	50.00	38.89	8.33	2.78	100.00
	56.25	53.85	50.00	25.00	52.94
TOTAL	32	26	6	4	68
	47.06	38.24	8.82	5.88	100.00
	100.00	100.00	100.00	100.00	100.00

Table 8.4.2 (cont) Best corrected visual acuity by stages of diabetic retinopathy in eyes without ocular pathology other than diabetic retinopathy in 1993 and in 1996. Entries are frequencies, row and column percentages.

best corrected vision of the eye	year and diabetic maculopathy							
	1993				1996			
	no	yes	not vis	TOTAL	no	yes	not vis	TOTAL
<6/60	8		30	38	7	1	10	18
	21.05	.	78.95	100.00	38.89	5.56	55.56	100.00
	2.20	.	58.82	8.30	1.65	2.56	76.92	3.79
6/60	6	3	7	16	5	1	2	8
	37.50	18.75	43.75	100.00	62.50	12.50	25.00	100.00
	1.65	6.98	13.73	3.49	1.18	2.56	15.38	1.68
6/36	7	2	6	15	4	2		6
	46.67	13.33	40.00	100.00	66.67	33.33	.	100.00
	1.92	4.65	11.76	3.28	0.95	5.13	.	1.26
6/24	7	2	1	10	9	3	1	13
	70.00	20.00	10.00	100.00	69.23	23.08	7.69	100.00
	1.92	4.65	1.96	2.18	2.13	7.69	7.69	2.74
6/18	20	3	6	29	18	4		22
	68.97	10.34	20.69	100.00	81.82	18.18	.	100.00
	5.49	6.98	11.76	6.33	4.26	10.26	.	4.63
6/15					2			2
	.	.	.	.	100.00	.	.	100.00
	.	.	.	.	0.47	.	.	0.42
6/12	29	5		34	28	1		29
	85.29	14.71	.	100.00	96.55	3.45	.	100.00
	7.97	11.63	.	7.42	6.62	2.56	.	6.11
6/9	63	6	1	70	93	11		104
	90.00	8.57	1.43	100.00	89.42	10.58	.	100.00
	17.31	13.95	1.96	15.28	21.99	28.21	.	21.89
6/7.5	27	5		32	8	1		9
	84.38	15.63	.	100.00	88.89	11.11	.	100.00
	7.42	11.63	.	6.99	1.89	2.56	.	1.89
6/6	197	17		214	249	15		264
	92.06	7.94	.	100.00	94.32	5.68	.	100.00
	54.12	39.53	.	46.72	58.87	38.46	.	55.58
TOTAL	364	43	51	458	423	39	13	475
	79.48	9.39	11.14	100.00	89.05	8.21	2.74	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.4.3 Best corrected visual acuity by diabetic maculopathy in eyes seen in 1993 and in 1996. Entries are frequencies, row and column and percentages.

best corrected vision of the eye	year and diabetic maculopathy							
	1993				1996			
	no	yes	not vis	TOTAL	no	yes	not vis	TOTAL
<6/60						1	1	2
	.	.	.	.	.	50.00	50.00	100.00
	.	.	.	.	.	3.03	100.00	2.94
6/60						1		1
	.	.	.	.	.	100.00	.	100.00
	.	.	.	.	.	3.03	.	1.47
6/36		2		2		1		1
	.	100.00	.	100.00	.	100.00	.	100.00
	.	5.88	.	4.76	.	3.03	.	1.47
6/24		1		1		2		2
	.	100.00	.	100.00	.	100.00	.	100.00
	.	2.94	.	2.38	.	6.06	.	2.94
6/18	2	1		3		2		2
	66.67	33.33	.	100.00	.	100.00	.	100.00
	25.00	2.94	.	7.14	.	6.06	.	2.94
6/12		3		3				
	.	100.00	.	100.00	.	.	.	.
	.	8.82	.	7.14	.	.	.	.
6/9	1	5		6	10	10		20
	16.67	83.33	.	100.00	50.00	50.00	.	100.00
	12.50	14.71	.	14.29	29.41	30.30	.	29.41
6/7.5		5		5	3	1		4
	.	100.00	.	100.00	75.00	25.00	.	100.00
	.	14.71	.	11.90	8.82	3.03	.	5.88
6/6	5	17		22	21	15		36
	22.73	77.27	.	100.00	58.33	41.67	.	100.00
	62.50	50.00	.	52.38	61.76	45.45	.	52.94
TOTAL	8	34		42	34	33	1	68
	19.05	80.95	.	100.00	50.00	48.53	1.47	100.00
	100.00	100.00	.	100.00	100.00	100.00	100.00	100.00

Table 8.4.4 Best corrected visual acuity by diabetic maculopathy in eyes without ocular pathology other than diabetic retinopathy in 1993 and in 1996. Entries are frequencies, row and column percentages.

best corrected vision of the eye	year and CSME							
	1993				1996			
	no	yes	not vis	TOTAL	no	yes	not vis	TOTAL
<6/60	8		30	38	7	1	10	18
	21.05	.	78.95	100.00	38.89	5.56	55.56	100.00
	2.09	.	58.82	8.30	1.59	4.76	76.92	3.79
6/60	6	3	7	16	5	1	2	8
	37.50	18.75	43.75	100.00	62.50	12.50	25.00	100.00
	1.57	12.00	13.73	3.49	1.13	4.76	15.38	1.68
6/36	7	2	6	15	5	1		6
	46.67	13.33	40.00	100.00	83.33	16.67	.	100.00
	1.83	8.00	11.76	3.28	1.13	4.76	.	1.26
6/24	8	1	1	10	10	2	1	13
	80.00	10.00	10.00	100.00	76.92	15.38	7.69	100.00
	2.09	4.00	1.96	2.18	2.27	9.52	7.69	2.74
6/18	20	3	6	29	20	2		22
	68.97	10.34	20.69	100.00	90.91	9.09	.	100.00
	5.24	12.00	11.76	6.33	4.54	9.52	.	4.63
6/15					2			2
	.	.	.	.	100.00	.	.	100.00
	.	.	.	.	0.45	.	.	0.42
6/12	31	3		34	29			29
	91.18	8.82	.	100.00	100.00	.	.	100.00
	8.12	12.00	.	7.42	6.58	.	.	6.11
6/9	65	4	1	70	97	7		104
	92.86	5.71	1.43	100.00	93.27	6.73	.	100.00
	17.02	16.00	1.96	15.28	22.00	33.33	.	21.89
6/7.5	28	4		32	9			9
	87.50	12.50	.	100.00	100.00	.	.	100.00
	7.33	16.00	.	6.99	2.04	.	.	1.89
6/6	209	5		214	257	7		264
	97.66	2.34	.	100.00	97.35	2.65	.	100.00
	54.71	20.00	.	46.72	58.28	33.33	.	55.58
TOTAL	382	25	51	458	441	21	13	475
	83.41	5.46	11.14	100.00	92.84	4.42	2.74	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.4.5 Best corrected visual acuity by CSME in eyes seen in 1993 and in 1996. Entries are frequencies, row and column and percentages.

best corrected vision of the eye	year and CSME							
	1993				1996			
	no	yes	not vis	TOTAL	no	yes	not vis	TOTAL
<6/60						1	1	2
	.	.	.	.	.	50.00	50.00	100.00
	.	.	.	.	.	4.76	100.00	2.94
6/60						1		1
	.	.	.	.	.	100.00	.	100.00
	.	.	.	.	.	4.76	.	1.47
6/36		2		2		1		1
	.	100.00	.	100.00	.	100.00	.	100.00
	.	10.00	.	4.76	.	4.76	.	1.47
6/24		1		1		2		2
	.	100.00	.	100.00	.	100.00	.	100.00
	.	5.00	.	2.38	.	9.52	.	2.94
6/18	2	1		3		2		2
	66.67	33.33	.	100.00	.	100.00	.	100.00
	9.09	5.00	.	7.14	.	9.52	.	2.94
6/12		3		3				
	.	100.00	.	100.00	.	.	.	.
	.	15.00	.	7.14	.	.	.	.
6/9	2	4		6	13	7		20
	33.33	66.67	.	100.00	65.00	35.00	.	100.00
	9.09	20.00	.	14.29	28.26	33.33	.	29.41
6/7.5	1	4		5	4			4
	20.00	80.00	.	100.00	100.00	.	.	100.00
	4.55	20.00	.	11.90	8.70	.	.	5.88
6/6	17	5		22	29	7		36
	77.27	22.73	.	100.00	80.56	19.44	.	100.00
	77.27	25.00	.	52.38	63.04	33.33	.	52.94
TOTAL	22	20		42	46	21	1	68
	52.38	47.62	.	100.00	67.65	30.88	1.47	100.00
	100.00	100.00	.	100.00	100.00	100.00	100.00	100.00

Table 8.4.6 Best corrected visual acuity by CSME in eyes without ocular pathology other than diabetic retinopathy in 1993 and in 1996. Entries are frequencies, row and column percentages.

best corrected vision of the eye	year and VTR							
	1993				1996			
	no	yes	not vis	TOTAL	no	yes	not vis	TOTAL
<6/60	8	2	30	40	7	2	9	18
	20.00	5.00	75.00	100.00	38.89	11.11	50.00	100.00
	2.10	7.14	58.82	8.70	1.60	8.00	75.00	3.79
6/60	6	3	7	16	5	1	2	8
	37.50	18.75	43.75	100.00	62.50	12.50	25.00	100.00
	1.57	10.71	13.73	3.48	1.14	4.00	16.67	1.68
6/36	7	2	6	15	5	1		6
	46.67	13.33	40.00	100.00	83.33	16.67	.	100.00
	1.84	7.14	11.76	3.26	1.14	4.00	.	1.26
6/24	8	1	1	10	10	2	1	13
	80.00	10.00	10.00	100.00	76.92	15.38	7.69	100.00
	2.10	3.57	1.96	2.17	2.28	8.00	8.33	2.74
6/18	19	4	6	29	20	2		22
	65.52	13.79	20.69	100.00	90.91	9.09	.	100.00
	4.99	14.29	11.76	6.30	4.57	8.00	.	4.63
6/15					2			2
	.	.	.	.	100.00	.	.	100.00
	.	.	.	.	0.46	.	.	0.42
6/12	31	3		34	29			29
	91.18	8.82	.	100.00	100.00	.	.	100.00
	8.14	10.71	.	7.39	6.62	.	.	6.11
6/9	65	4	1	70	95	9		104
	92.86	5.71	1.43	100.00	91.35	8.65	.	100.00
	17.06	14.29	1.96	15.22	21.69	36.00	.	21.89
6/7.5	28	4		32	9			9
	87.50	12.50	.	100.00	100.00	.	.	100.00
	7.35	14.29	.	6.96	2.05	.	.	1.89
6/6	209	5		214	256	8		264
	97.66	2.34	.	100.00	96.97	3.03	.	100.00
	54.86	17.86	.	46.52	58.45	32.00	.	55.58
TOTAL	381	28	51	460	438	25	12	475
	82.83	6.09	11.09	100.00	92.21	5.26	2.53	100.00
	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.4.7 Best corrected visual acuity by VTR in eyes seen in 1993 and in 1996. Entries are frequencies, row and column and percentages.

best corrected vision of the eye	year and VTR					
	1993			1996		
	no	yes	TOTAL	no	yes	TOTAL
<6/60		2	2		2	2
	.	100.00	100.00	.	100.00	100.00
	.	8.70	4.55	.	8.00	2.94
6/60					1	1
	.	.	.	.	100.00	100.00
	.	.	.	.	4.00	1.47
6/36		2	2		1	1
	.	100.00	100.00	.	100.00	100.00
	.	8.70	4.55	.	4.00	1.47
6/24		1	1		2	2
	.	100.00	100.00	.	100.00	100.00
	.	4.35	2.27	.	8.00	2.94
6/18	1	2	3		2	2
	33.33	66.67	100.00	.	100.00	100.00
	4.76	8.70	6.82	.	8.00	2.94
6/12		3	3			
	.	100.00	100.00	.	.	.
	.	13.04	6.82	.	.	.
6/9	2	4	6	11	9	20
	33.33	66.67	100.00	55.00	45.00	100.00
	9.52	17.39	13.64	25.58	36.00	29.41
6/7.5	1	4	5	4		4
	20.00	80.00	100.00	100.00	.	100.00
	4.76	17.39	11.36	9.30	.	5.88
6/6	17	5	22	28	8	36
	77.27	22.73	100.00	77.78	22.22	100.00
	80.95	21.74	50.00	65.12	32.00	52.94
TOTAL	21	23	44	43	25	68
	47.73	52.27	100.00	63.24	36.76	100.00
	100.00	100.00	100.00	100.00	100.00	100.00

Table 8.4.8 Best corrected visual acuity by VTR in eyes seen without ocular pathology other than diabetic retinopathy in 1993 and in 1996. Entries are frequencies, row and column percentages.

The pivotal findings of this chapter are that in diabetic subjects of Aboriginal communities of the Katherine region

- In 1993, after correction, 11.7% of subjects had impaired vision, 7.4% of subjects had monocular blindness, 4.8% of subjects had binocular blindness. The respective figures for 1996 were 3.8%, 5.9% and 1.3%.

- In 1993, of those older than 60, 28.6% of subjects had impaired vision, 14.3% of subjects were monocularly blind, 15.9% of subjects were binocularly blind. The respective figures for 1996 were 10.8%, 9.2% and 3.1%.

- In 1993 the prevalence of impaired vision and binocular blindness were much higher in males than in females. In 1996 the prevalence of impaired vision and binocular blindness were approximately the same in both genders.

- In both 1993 and 1996 almost all cases of visual impairment could have been prevented.

- The KRDRS has also shown the importance of reporting visual acuity in both subjects and individual eyes. Reporting the effect of glasses on visual acuity in the "no blindness /blindness" and "impaired / adequate vision" categories in eyes alone would have underestimated its significance in the population. On the other hand, reporting the effect of glasses on vision in eyes where vision could be improved with three or more lines would have overestimated its significance in the population.

- Visual acuity is a poor indicator for the presence of diabetic retinopathy, maculopathy CSME and VTR.

Chapter 9

9. Discussion

This chapter summarizes the principal results of the KRDRS and then discusses them within the context of other Australian studies. The third section of the discussion outlines some implications of the KRDRS for screening, health service delivery and further research.

9.1 Principal results of the KRDRS

In 1993 and in 1996 the prevalence of subject retinopathy was 18% and 21% and the prevalence of clinically significant macula edema (CSME) was 8% and 6% respectively. The prevalence of vision threatening retinopathy (VTR) was 8.5% and 6.7% and the proportion of subjects with VTR in those with retinopathy was 47% and 50%, respectively. If corrections for the effect of laser treatment were made, the adjusted figures in 1993 and in 1996 for diabetic retinopathy (19% and 22%), for CSME (9% and 11%), for VTR (10% and 12%) and for the proportion of subjects with VTR in those with retinopathy (51% and 54%) were even higher. There was an unexpectedly high prevalence of CSME in subjects with mild, moderately severe and severe BDR. In 1993 at the time of diagnosis of diabetes, 11% of subjects had diabetic retinopathy and 3% of subjects had VTR. The corresponding figures for 1996 were 9% and 2%.

The annual progression rates from no retinopathy to any retinopathy were, in subjects (eyes), 5.6% (4.2%), from no retinopathy to NPDR 5.2% (3.8%), from no retinopathy to PDR 0.4% (0.4%), from no retinopathy to VTR 1.2% (1.2%) and from no VTR to VTR 1.4% (1.3%). If the time since diagnosis was less than 4 years, the annual progression rates of diabetic retinopathy were in subjects (eyes) from no retinopathy to any retinopathy 4% (2.8%) and from no retinopathy to VTR 0.6% (0.6%). If the time since diagnosis was between 4 and 9 years, the annual progression rate of diabetic retinopathy were in subjects and (eyes) from no retinopathy to any retinopathy 11.1% (9.2%) and from no retinopathy to VTR 3.5% (3.3%).

The prevalence of impaired subject vision, monocular and binocular blindness in the KRDRS were 11.7%, 7.8% and 4.8% in 1993. The respective figures for 1996 were 3.8%, 5% and 1.3%.

In 1993 the prevalence of impaired subject vision and binocular blindness was much higher in females than in males. In 1996 the prevalence of impaired vision and binocular blindness was approximately the same in both sexes.

In 1993 in 3.5% (8/229) of subjects the vision could be improved one line, in 1.3% (3/229) of subjects by three lines after correction. The respective figures for 1996 were 30.0% (71/237) and 7.6% (18/237).

The KRDRS has also shown the importance of reporting visual acuity in subjects and individual eyes. Reporting the effect of glasses on visual acuity in the "no blindness / blindness" and "impaired / adequate vision" categories in eyes alone would have underestimated its significance in the population. On the other hand, reporting the effect of glasses on vision in

eyes only where vision could be improved by three or more lines would have overestimated its significance in the population.

Cataract was the single major cause of impaired vision, monocular and binocular blindness among diabetics in both 1993 and in 1996. In 1993 diabetic retinopathy was responsible for only 6% of impaired vision, for 0% of the monocular blindness and for 5% of binocular blindness. The respective figures for 1996 were 13%, 0% and 11%. In 1993, 5.8% of eyes could be improved from impaired vision to adequate vision with glasses. The respective figure for 1996 was 45%. The KRDRS results show that visual acuity is a poor indicator of the presence of diabetic retinopathy when diabetic retinopathy coexists with other ocular pathologies. This is also true for eyes with diabetic retinopathy as the only ocular pathology. Of those with CSME and with no ocular pathology other than diabetic retinopathy, 52% had a visual acuity of 6/9 or better in 1993. The respective figure for 1996 was 67%.

9.2 Discussion of results from the KRDRS in relation to other Australian studies

9.2.1 Diabetic retinopathy

The Katherine Region Diabetic Retinopathy Study (KRDRS) preceded the official recognition, in 1996, of diabetes as one of the National Health Priority Areas (NHPA) (Department of Health and Family Services and Australian Institute of Health and Welfare, 1997; Department of Health and Family Services and Australian Institute of Health and Welfare, 1999). Its objectives reflected the desire for efficient management and control of diabetic retinopathy in Aboriginal communities. The basic epidemiology of diabetic retinopathy in these communities was unknown. The KRDRS provides the best estimates so far of diabetic retinopathy in an Aboriginal Australian diabetic population. This is due to the rigorous methods for subject selection, outcome measurement and to the very high coverage of the target population.

There is only one study reporting the prevalence of diabetic retinopathy in the general Australian population where the method of subject selection allows comparison with the KRDRS (Mitchell 1980, 1985, 1990). In other studies the objectives, subject selection and methods used for diagnosis and reporting diabetic retinopathy make comparison with the KRDRS less applicable. Even allowing for the time difference between Mitchell's study and the KRDRS, on the basis of the prevalence figures from the KRDRS, it first appears that diabetic retinopathy affects the Aboriginal population of

Katherine region to a lesser extent than the non-Aboriginal population of Australia.

However, if the prevalences of maculopathy, CSME and VTR in the KRDRS are compared to the Newcastle or Singleton population of Mitchell's study (Mitchell, 1980; Mitchell, 1985), four important differences are seen.

First, the prevalence of maculopathy in the KRDRS was approximately twice that in Singleton (Mitchell, 1980).

Second, in the KRDRS 60% or more of the subjects with maculopathy had CSME compared to 20% of subjects in the Newcastle diabetic clinic and 50% in the Singleton Census area (Mitchell, 1980). These revised proportions in the Newcastle Study of Diabetic Retinopathy (NSDR) were 10% and 19%, respectively (Mitchell, 1985). While the prevalence of CSME by stages of diabetic retinopathy was not reported by Mitchell, in the KRDRS in 1993 34% of subjects with mild BDR had CSME compared with 16% in 1996. The prevalence of CSME in the KRDRS is the highest reported in any Australian study (8% in 1993 and 6% in 1996). There is no other study that has reported CSME by stages of diabetic retinopathy; in the KRDRS the prevalence of CSME in 1993 was 34% in subjects with mild BDR, 80% in those with moderately severe BDR, 100% in those with severe BDR. The respective figures for 1996 were 16%, 33% and 75%. This means that in Aboriginal communities CSME may appear at a very early stage of the disease giving rise to vision threatening retinopathy (VTR).

Third, the prevalence of proliferative diabetic retinopathy was found to be lower (0.9% in 1993 and 1.3% in 1996) than the figures reported in the NSDR (Mitchell, 1980; Mitchell, 1985).

Fourth, the prevalence of VTR in the KRDRS (8.5% in 1993 and 6.7% in 1996) suggests that diabetic retinopathy is as important a health issue in Aboriginal communities as in non-Aboriginal Australians (Mitchell, 1980; Mitchell, 1985).

After adjusting for previous laser therapy, the prevalence of proliferative diabetic retinopathy and VTR in 1996 was similar to that found in the NSDR (Mitchell, 1980; Mitchell, 1985).

We can also compare prevalences of maculopathy, CSME and VTR among those with retinopathy in the KRDRS and the NSDR. In 1993, in the KRDRS, of those with retinopathy 42% of subjects had CSME and 47% had VTR. The respective figures for 1996 were 28% and 32%. These figures are higher than in the NSDR where 27% of subjects with retinopathy had VTR in the Newcastle diabetic clinic. The respective figure for the Singleton Census area was 22% (Mitchell, 1985). If these proportions from the KRDRS are adjusted for the effect of laser treatment (of those with retinopathy in 1993 46% had CSME, 51% had VTR; the respective figures in 1996 were 50% and 54) it is even more evident that in Aboriginal Australians diabetic retinopathy is more likely to present with VTR than in non-Aboriginal Australians.

In the KRDRS the annual incidence of diabetic retinopathy was 5.6% in subjects (4.2% in eyes) with no retinopathy at the beginning of the study. Even allowing for the time differences between Mitchell's study and the

KRDRS it may appear that the progression of diabetic retinopathy is slower in the Aboriginal Australian population than in the NSDR (Mitchell, 1985). If however the progression of diabetic retinopathy in the KRDRS is compared to Mitchell's study three major differences are seen.
First, the annual progression rate from no diabetic retinopathy to VTR in the KRDRS was threefold that in the NSDR (Mitchell 1985). It is consistent with the high prevalence of VTR in the KRDRS.

Second, in 21% (29%) of new cases, diabetic retinopathy presented as VTR compared with 5% in the NSDR (Mitchell, 1985).

Third, the annual progression rate from mild to worse grade diabetic retinopathy in the KRDRS was 12.1% (8.8%), higher than Mitchell's figures in subjects (8%) (Mitchell, 1985).

Due to the high prevalence of maculopathy and CSME in the KRDRS study population, a description of the incidence of CSME was also warranted in those with and without maculopathy. In those without maculopathy the annual progression rate to CSME was 1.1% (1%); in those with maculopathy the corresponding figure was 4.8% (6.1%).

On the basis of the overall incidence of diabetic retinopathy it may seem that progression of diabetic retinopathy in the KRDRS is slower than in the non – Aboriginal Australian community. The short observation period in the KRDRS limits our ability to detect progression. (The observation period in the KRDRS was 3 years, less than half than that in Mitchell's study). Also, in the NSDR the incidence of diabetic retinopathy in previously unaffected subjects showed a peak late in the first decade of diabetes in the young-onset group, while in the older onset group, the peak occurred in the

second decade of diabetes. On the other hand, for progression from early to more advanced or proliferative stages of diabetic retinopathy the rate peaked in the second and third decade of diabetes (Mitchell, 1985). In the KRDRS the mean time since diagnosis in the non-lasered core population of the study was only 3.8 years compared with 9.5 years in Mitchell's cross-sectional population (Mitchell, 1980). Therefore, the overall lower incidence rate in the KRDRS may still represent a similar or accelerated progression of diabetic retinopathy compared to the non-Aboriginal Australian population. This is especially so if the high rates of progression to VTR are considered.

9.2.2 Visual acuity and ocular pathology

Potentially, the results of the diabetic Aboriginal population of the KRDRS should be discussed in the context of our knowledge of visual impairments in:

- the general Aboriginal Australian population (studies from the National Trachoma and Eye Health Program (RACO, 1980)) and the Anungu Pitjantjatjara (Stocks, 1994; Stocks, 1997)
- the diabetic Aboriginal Australian population (from studies other than the KRDRS)
- the general Australian population (Newland, 1996; Taylor, 1997; Wang, 2000)
- the diabetic Australian population.

Since there are no reports on visual acuity in the diabetic Australian population (Aboriginal and non-Aboriginal) apart from the KRDRS, results from KRDRS can only be discussed in relation to the general Aboriginal Australian population and to the general non-Aboriginal Australian population. Studies in the general (non-Aboriginal) Australian population have described visual acuity in subjects aged 40 and over (Taylor, 1997), 49 and over (Wang, 2000) and 50 and over (Newland, 1996).

9.2.2.1 Comparison of results with the general Aboriginal population

The NTEHP's findings were reported in 13 zones of rural Australia (RACO 1980). These zones were chosen because of relative similarity in geographic, climatic, industrial and demographic conditions (RACO 1980). The NTEHP found that there was a similar pattern of disease across communities within each zone (RACO 1980). Visual impairment in the NTEHP was most prevalent in Zone 2 which includes the Katherine region of the Northern Territory (RACO 1980). This suggests that results from the KRDRS should be discussed in terms of the prevalence of visual impairment in Zone 2 of the NTEHP. However, to indicate the burden of disease in the KRDRS study population and induce action among health professionals (as the overall results from the NTEHP did in the 1980s) the visual impairment in the KRDRS will also be discussed in the light of the overall visual impairment in the NTEHP. Also, not all results from the NTEHP were reported by zones (RACO 1980).

There is another study that will be referred to in the discussion. Stocks' study (Stocks,1994; Stocks, 1997) was carried out in the Anungu Pitjantjatjara, an area that covers the Northwest corner of South Australia. This area was part of Zone 1 of the NTEHP (RACO, 1980).

9.2.2.1.1 Visual impairment

In 1993, the prevalence of visual impairment in the KRDRS was 11.7%, more than three times than in the corresponding zone of the NTEHP (3.8%). In 1996 the prevalence was the same (RACO 1980). In 1993 the prevalence of visual impairment in the KRDRS was higher than the unadjusted 3.8 % prevalence reported by Stocks et al (1997), in 1996 the prevalence of visual impairment was less than Stocks' unadjusted result. In the KRDRS there was an 8% decrease in visual impairment in the 1996 study population. In both years of the study females were more likely to present with visual impairment, similar to Stocks et al's study (Stocks, 1997).

In the KRDRS the prevalence of binocular blindness was 4.8% in 1993 and 1.3% in 1996. The prevalence for 1993 was much higher than the overall prevalence of blindness in the NTEHP (1.5%) (RACO, 1980), the prevalence found by the corresponding area in the NTEHP (3.5%) (RACO, 1980), the prevalence reported by Taylor in Central Australia (1.8%) (Taylor, 1977) or the prevalences reported by Stocks et al in the Anungu Pitjantjatjara (1.5% and 0.9%, respectively) (Stocks, 1994; Stocks, 1997). The prevalence of binocular blindness in the KRDRS in 1996 was less than

the overall prevalence found by the NTEHP (1.5%) (RACO, 1980), less than that reported by Stocks et al in the Anungu Pitjantjatjara land (Stocks, 1994) and less than that reported in the Zone 2 of the NTEHP (RACO, 1980) but more than Stocks' adjusted figure. In 1993, similar to Stocks et al's study (Stocks, 1994) and to the NTEHP (RACO, 1980) females were more likely to present with impaired vision and binocular blindness than males.

The overall prevalence of monocular blindness in the KRDRS (7.4% in 1993 and 5.9% in 1996) was higher than in the NTEHP (prevalence of monocular blindness was 4.7% in Zone 2, the overall prevalence was 2.7% in the NTEHP) (RACO, 1981). In the KRDRS a higher proportion of subjects with monocular blindness had adequate vision in their other eye (76% in 1993 and 92% in 1996) than in the NTEHP (64%) (RACO, 1980). The different blindness criteria used do not explain the higher proportion of subjects with adequate vision in their other eye. Also, adequate vision in the other eye of monocularly blind subjects were not reported by zones in the NTEHP.

9.2.2.1.2 Comparison of visual impairment by age and sex

In 1993, in the KRDRS the prevalence of impaired vision and blindness was higher in females; in 1996 the prevalence of impaired vision and blindness was approximately the same.

A higher prevalence of blindness in females was found in trachomatous areas in Tanzania (Turner, 1993) and in Nepal (Brilliant, 1985). In the

Australian context one explanation may be that attendance by Aboriginal women for consultations with white men can be difficult, and it is preferable for women to treat women and men to treat men (Edwards, 1990). Gender roles in society also have implications for knowledge about access to health resources with women having less access than men to preventive and curative services (Brilliant, 1985). One possible explanation of the change in the prevalences by sex (especially in the 60+ age group) may be that the active search for the visually impaired in the KRDRS has overcome the barriers that prevented women from accessing ophthalmic services.

Prevalences of impaired vision, monocular blindness and binocular blindness similar to the KRDRS can be found in a non-Aboriginal Australian population that is approximately 30 years older than the study population of the KRDRS (Newland, 1993; Taylor, 1997; Wang, 2000). On a population level, Indigenous Australians demonstrate "accelerated ophthalmological ageing".

In the KRDRS, in the 60+ age group, the prevalence of visual impairment, monocular and binocular blindness was 28.6%, 14.1% and 15.9% in 1993. These figures were less relative to the Zone 2 of the NTEHP (the respective figures were 24.9%, 21.5% and 25.2%) or than the overall prevalences in the NTEHP (22.7%, 19.3% and 18.9%, respectively).. However, similar to Stocks et al findings (Stocks, 1994) it must be concluded that further improvements in this age group will need to occur.

9.2.2.1.3 Comparison of ocular pathology causing visual impairment

The high proportion of those with cataract in the visually impaired group was surprising. The prevalence of cataract in the binocularly and monocularly blind group was nearly the same in both 1993 and in 1996 (50% and 49% respectively) as in Taylor's Central Australian Aboriginal population (Taylor, 1977) 20 years prior to the KRDRS. Diabetic retinopathy was responsible for less than 10% of visual impairment in both 1993 and in 1996. As in the NTEHP (Taylor, 1977) or in the Pitjantjatjara land study (Stocks, 1994) nearly all blindness in the KRDRS could have been prevented. The literature suggests that isolation and restricted access to services contribute to the prevalence of cataract (Brilliant, 1985) and trauma related blindness (Schwab, 1990).

9.3 The effect of refraction on visual impairment

The effect of refraction on visual impairment in eyes was different in the KRDRS in 1993 and in 1996. This may have been due to the increase in myopic prescriptions (which were not due to high BSL) and also to the less severe ocular pathologies present in the 1996 study population. The presence of myopic prescriptions without any other pathology present is unusual in light of Taylor's observations regarding hypermetropia in Aboriginal people (RACO, 1980).

The KRDRS has also shown the importance of reporting visual acuity in subjects and individual eyes. Reporting the effect of glasses on visual

acuity in the "no blindness / blindness" and "impaired / adequate vision" categories in eyes alone would have underestimated its significance in the population. On the other hand, reporting the effect of glasses on vision in eyes only where vision could be improved with three or more lines would have overestimated its significance in the population.

The proportion of subjects who improved their vision by one line in the KRDRS (3.5% in 1993 and 30% in 1996) was much lower than in the Baltimore Eye Survey (Tielsch, 1990) (54%), in the Blue Mountains Eye Study (Attebo, 1996) (45%) or in the Melbourne Visual Impairment Project (Liou, 1999) (10%). The proportion of subjects in the KRDRS who improved their vision 3 or more lines after refraction was much lower in the KRDRS in 1993 (1.3%) than in the Baltimore Eye Survey (Tielsch, 1990) (7.5%) or in the Blue Mountains Eye Study (Attebo, 1996) (13%). In the KRDRS in 1996 the proportion of subjects who improved their vision three or more lines after refraction was the same (7.6%) as in the Baltimore Eye Survey (Tielsch, 1990) (7.5%) but less than in the Blue Mountains Eye Study (Attebo 1996) (13%).

9.4 The effect of diabetic retinopathy on vision

The visual acuity of a diabetic Australian population has not been described previously. In Aboriginal Australians visual acuity (with or without ocular pathologies other than diabetic retinopathy) was a poor indicator for diabetic retinopathy, maculopathy, CSME or VTR. Since CSME is a main feature of diabetic retinopathy in this Aboriginal diabetic population, it is of particular importance that, when diabetic retinopathy was the only

significant ocular pathology, 65% of eyes had a visual acuity of 6/9 or better in 1993 and 67% in 1996. The similar figures for VTR were 47% and 78 % respectively. These figures are similar to Klein's (Klein 1984) Harding's (Harding 1995) report and they show that measurement of visual acuity has very little to add to the detection of diabetic retinopathy.

In 1993 in this diabetic Aboriginal Australian population diabetic retinopathy was responsible for 6% of visual impairment and for 5% of blindness in eyes. The respective figures for 1996 were 13% and 11%. This is in sharp contrast to studies where visual acuity and ocular pathology are described in "first world" diabetics.

A comparison of blindness in the KRDRS and other non-Aboriginal studies shows that in 1993 the prevalence of blindness in the KRDRS (4.8%) was higher than in non - Aboriginal diabetic populations. In 1996 the prevalence of blindness in the KRDRS (1.3%) was approximately the same as in non-Aboriginal diabetic populations. In Klein's study (1984) the prevalence of blindness in the older onset group was 1.6%, in Prasad's study 1.13% (Prasad 2001) and in Fong's study 1.5% (Fong 2001).

A major difference is seen in the proportion of blindness due to diabetic retinopathy in non-Aboriginal populations and in the KRDRS. The proportions of blindness due to diabetic retinopathy was 86% in Klein's study (Klein 1984), 32% in Prasad's study (Prasad 2001) and 42.7% in Fong's study (Fong 2001). This is in sharp contrast with the KRDRS. \

9.5 Comparison with Australian studies

The results from the KRDRS have already been compared to the non-Aboriginal population during presentation of the results. For convenience I summarize the findings and refer the reader to the set of study hypotheses I proposed in Chapter 2.

In Chapter 3 Section 2.4 results from the NSDR (Mitchell 1980, 1985, 1990) have been presented in great detail due to their significance. For comparison with the KRDRS his modified prevalence figures (Mitchell 1985) are used. According to this, the prevalence of ophthalmoscopic retinopathy, proliferative diabetic retinopathy, vision – threatening maculopathy and VTR were 49%, 7%, 10% and 13% in the Newcastle diabetic clinic. The respective figures for the Singleton Census area were 36%, 3%, 8% and 8%, respectively (Mitchell 1985). While confidence intervals were not provided in his study, they can be calculated.

1. The prevalence of diabetic retinopathy in subjects of the known Aboriginal diabetic population of Katherine region was 18% in 1993 and 21% in 1996 (RR = 1.3, 95%CI = 0.9, 1.8; P = 0.16). If corrections for the effect of laser treatment were made, these figures were even higher (19% and 22%, respectively)(RR= 1.2; 95%CI = 0.9, 1.7; P = 0.25). In the NSDR Mitchell found a prevalence of 49% (95%CI = 0.46, 0.52) in the diabetic clinic in Newcastle and 39% (95%CI = 0.27, 0.46) in the Singleton census area (Mitchell, 1980). On the basis of these prevalence figures from the KRDRS it appears that diabetic retinopathy affects the Aboriginal population of Katherine region to a lesser extent than the non-Aboriginal Australian population.

2. The prevalence of diabetic maculopathy in subjects of the known Aboriginal diabetic population of Katherine region was 13% in 1993 and 10% in 1996 (RR = 0.8; 95%CI= 0.5, 1.2; P = 0.29). If corrections for the effect of laser treatment were made, these figures were even higher (14% in 1993 and 13% in 1996; RR = 0.9; 95%CI = 0.6, 1.3; P = 0.56). The prevalence of maculopathy is unclear in Mitchell's study (see Chapter 3 Section 2.4 for details) (Mitchell 1980, 1985).

   The prevalence of clinically significant macula edema in subjects of the known Aboriginal diabetic population of Katherine region was 7.5% in 1993 and 5.9% in 1996 (RR = 0.8, 95%CI = 0.4, 1.4). If corrections for the effect of laser treatment were made, the respective figures were 9% and 11% (RR = 1.2; 95%CI = 0.7, 1.9; P = 0.49). If Mitchell's modified figures are used, in the NSDR the prevalence of "vision threatening maculopathy" was 5% (95% CI = 0.04, 0.06) in the Newcastle diabetic clinic and 7% (95%CI =0.02, 0.12) in the Singleton Census area. The overall prevalence rate of "vision threatening maculopathy" in his study was 0.05 (95%CI = 0.04,0.06) (note that the Singleton Census area there were only 99 known diabetics). Therefore, in the KRDRS study subjects were more likely to present with clinically significant macula edema than subjects with "vision-threatening maculopathy" in the NSDR.

3. The prevalence of vision threatening retinopathy in subjects of the known Aboriginal diabetic population of Katherine region was 8.5% in 1993 and 6.7% in 1995 (RR = 0.9, 95%CI = 0.5, 1.5; P value = 0.59). If corrections for the effect of laser treatment were made the respective figures were 10% and 12% (RR 1.2, 95%CI = 0.8, 2.0; P = 0.35). Mitchell found the prevalence of VTR 13% (95%CI = 0.11,0.15) in the

Newcastle diabetic clinic and 8% (95%CI = 0.03,0.13) in the Singleton census area (Mitchell 1985). Even if the 95%CI from Mitchell's study includes the point prevalences from the KRDRS, results from the KRDRS represent a more severe presentation of diabetic eye disease due to the much shorter duration of diabetes in the KRDRS (see Chapter 5 for details).

While Mitchell did not report the prevalence of CSME by stages of diabetic retinopathy, the KRDRS found that of those with mild BDR 34% of subjects had CSME in 1993 and 16% in 1996 (RR = 0.5, 95%CI = 0.2, 1.3; P = 0.14). The respective figures for moderately severe BDR were 80% and 33% (RR = 0.4, 95%CI = 0.2, 0.9; P = 0.03), for severe BDR were 100% and 75% (RR = 0.7, 95%CI = 0.4, 1.3; P = 0.29), for proliferative diabetic retinopathy 0% and 33%. These figures show a more advanced form of diabetic retinopathy in the Aboriginal population, especially if the short time since diagnosis of diabetes is considered. (Regarding the time since diagnosis of diabetes see Chapter 5 Section 2).

4. In 1993, the prevalence of subject retinopathy was 21% in the non-traditional environment and 18% in the traditional living environment (RR = 0.8; 95%CI = 0.3,2.1, P = 0.70). The respective figures for 1996 were 29% and 19% (RR = 0.7, 95% CI = 0.4,1.2; P = 0.17). After corrections for the effect of laser treatment were made, the respective figures for 1993 were 21% and 19% (RR = 0.9, 95% CI = 0.4,2.3; P = 0.83), for 1996 32% and 20% (RR = 0.6; 95%CI = 0.4, 1.1; P = 0.10).

5. In 1993, the prevalence of diabetic retinopathy was 18% in major centers and 16% in other communities. After corrections for the effect

of laser treatments were made, the respective figures were 20% and 18%. It was shown, that in 1993 in both living environments approximately half of those with diabetic retinopathy presented with sight-threatening changes.

In 1996, the prevalence of diabetic retinopathy was 25% in major centers and 13% in other communities. After corrections for the effect of laser treatment were made, the respective figures were 25% and14%. It has been shown that in 1996, those subjects with diabetic retinopathy living in other communities were more likely to present with vision threatening changes.

6. The annual incidence of diabetic retinopathy was 5.6% in subjects with no retinopathy at the beginning of the study (95%CI = 3%, 10%). This figure on its own may represent a figure lower to Mitchell's study (8%) (Mitchell, 1985). (The confidence intervals for incidence rates cannot be calculated from Mitchell's study, since only an approximate follow up period is given.) However, the progression from no retinopathy to VTR was threefold in the KRDRS (1.2%, 95%CI = 0.2%, 3.8%) than in Mitchell's study (0.4%) (Mitchell 1985). Even if the 95% CIs around the point estimates in the KRDS involve Mitchell figure, the annual incidence of VTR in the KRDRS represents an accelerated progression of the disease due to the shorter observation period and the shorter time since diagnosis in the KRDRS than in Mitchell's study (Mitchell 1980, 1985).

7. If the annual incidence of diabetic retinopathy is examined the main difference between major centers and other communities was that none of the subjects progressed to clinically significant macula edema

who were living in major centers. Of those with no CSME at the beginning of the study 3.7% (95%CI = 0.8%, 10.8%) progressed to CSME annually.

8. The KRDRS found that diabetic retinopathy was responsible only for 6% of impaired vision in 1993, and for 13% in 1996. The main cause of impaired vision was cataract in both 1993 (67%) and in 1996 (58%). There are no reports on the visual acuity and ocular pathology of a known diabetic non – Aboriginal Australian population

9. The KRDRS found that 52% of eyes with clinically significant macula edema had a vision better than 6/12 in 1993 and 67% in 1996. In eyes with no ocular pathology other than diabetic retinopathy the respective figures were 65% and 67%.

9.6 Implications of the KRDRS

9.6.1 Implications for screening

From the literature review it can been seen that while there are reliable screening methods available to detect diabetic retinopathy, in the general Australian population (Aboriginal and non-Aboriginal) there is poor (Harper 1995, Taylor 1997) or "suboptimal" compliance with screening in the community (Bamroongsuk et al 2002).

The purpose of screening for diabetic retinopathy is to detect retinopathy at an early stage of the disease when timely intervention can reduce visual loss. The current NHMRC screening guidelines (National Health and Medical Research Council, 1997) for diabetic retinopathy are supported by the KRDRS on the basis of the prevalences of diabetic retinopathy and VTR by age and time since diagnosis, the prevalence of diabetic retinopathy and VTR at the time of diagnosis of diabetes (see Chapter 6) and the progression of diabetic retinopathy in the study population (see Chapter 7). The OATSIH report described the barriers to screening for diabetic retinopathy such as the "possible deficiencies in primary health care recall systems, in general practitioners' examination skills or in the referral systems", and also names barriers specific to Aboriginal communities such as "distance from facilities, faltering referral systems and cross-cultural barriers" (OATSIH, 2001). On the basis of the barriers to screening for diabetic retinopathy in Aboriginal communities the necessity for annual screening in the communities as suggested by the OATSIH guidelines (OATSIH, 2001) is supported by the findings from the KRDRS. The best method of identifying cases in Aboriginal populations however requires further consideration.

The NHMRC and OATSIH guidelines fell short of the reality of everyday clinical practice in traditional Aboriginal communities (National Health and Medical Research Council, 1997; OATSIH, 2001). While they recognized differences in attitudes of various Aboriginal communities towards accepting medical care, they did not go far enough in their conclusions about its significance in the provision of medical care to these communities. Despite Taylor's and the NTEHP's description of the importance of personal relationships in providing eye care in Aboriginal

communities 25 years ago (Taylor, 1977; RACO, 1981), in the NHMRC and OATSIH guidelines identification of cases and their treatment are not separated (National Health and Medical Research Council, 1997; OATSIH, 2001). It was assumed in these reports that case identification inevitably leads to treatment and therefore to prevention of blindness. Case identification may be followed by treatment in some (Aboriginal and non-Aboriginal) communities in Australia, but due to the cultural differences between health regions and individual communities caution must be exercised when generalizing this concept to all Aboriginal communities. Aboriginal communities change with time in many ways including in their attitudes to health services. Therefore it must be emphasized that the recommendations below are based on the experience of the KRDRS between 1993 and 1996.

The current guidelines for Aboriginal communities suggest annual screening using fundus camera and immediate referral of subjects with VTR to ophthalmologists (OATSIH, 2001). Non-urgent referral is suggested for subjects with a visual acuity of less than 6/12 and no diabetic retinopathy, with unexplained visual loss or ungradable photos. When macula edema is the only sign of CSME, it is undetectable without a good quality stereoscopic photograph and the presence of CSME is not inconsistent with a visual acuity of 6/6 (Early Treatment Diabetic Retinopathy Study Group, 1985; Harding, 1995).

If the OATSIH guidelines were applied to the study population of the KRDRS (without allowances being made for ungradable photos) in 1993, 8.5% of diabetics in Katherine region would have required a referral to Darwin for laser treatment. In addition, 11.4% of those living outside Katherine would have required a referral to Katherine for ophthalmic

examination since they did not have VTR, but their visual acuity was less than 6/12. In 1996, when the Mobile Eye Unit had access to a portable laser, 17% of the diabetic population outside Katherine would have had to have been referred to the central hospital in Katherine for examination or treatment. If an ungradable rate of 13% is assumed (on the basis of the MVIP (Keeffe, 1997)) in 1993 approximately between 20% (if all non-gradable photographs had a visual acuity of less than 6/12) and one-third (if all non-gradable photographs had a visual acuity of better than 6/12) of the study population living outside Katherine would have had to have been referred to a central hospital for review (Darwin or Katherine). Under those circumstances, in 1996 the proportion of subjects needing to be referred to Katherine would have been between 17 and 30 % of those living outside the regional center. (It may be argued that in the Aboriginal population from the MVIP (Keeffe, 1997) the rate of ungradable photographs is lower than it would be in those Aboriginal patients living in remote communities in the Katherine region.) These percentages do not include those subjects from the community who are not diabetic but would need assessment for other ocular conditions.

On the basis of the foregoing discussion we have to conclude that the current OATSIH guidelines (OATSIH, 2001) are not feasible in the Katherine region of the Northern Territory since they place an unnecessary burden on the regional health services.

First, these guidelines present an unnecessary burden on the Patient Assisted Travel Scheme (PATS). The PATS was funded by the Commonwealth Department of Health and it had an annual limit for the region. Therefore, careful planning was required to maximise the benefits it can provide.

Second, these guidelines also require the staff of the department to spend a considerable amount of time to organise travel and treatment for those who could be reviewed in the communities. This time could be used in a more effective way by the nursing staff.

Therefore, the current guidelines must be reexamined and a more cost – effective way of service delivery must be established.

Data from the literature also show that using fundus cameras for screening of diabetic retinopathy, without good quality stereoscopic photographs, will miss cases with macula edema alone as sign of CSME and testing of visual acuity has nothing to add to the diagnosis of CSME (Early Treatment Diabetic Retinopathy Study Group, 1985; Harding, 1995).

The data above show that (on the basis of the current recommendations (OATSIH, 2001)) screening for diabetic retinopathy with a fundus camera is a poor substitute for a personal visit from an ophthalmologist into Aboriginal communities. This is due to the large proportion of subjects with a visual acuity of less than 6/12, the proportion of the ungradable photos and to other ocular conditions present in non-diabetics in the communities. It is true that the presence of ocular pathology requiring laser treatment can be detected even if the slide is ungradable (Karagiannis, 1996). The other two arguments however on their own are enough to support an annual community visit by an ophthalmologist.

While a detailed cost benefit analysis would have to be the subject of a further study, it seems more beneficial for the patient (and more cost effective for the health department) if regular visits are made to Aboriginal

communities by an ophthalmologist and cases requiring treatment (diabetic and non-diabetic) are identified by a visiting ophthalmologist in the communities similar to the teams of the NTEHP (1980) and later to that of the Mobile Eye Unit. This method of community screening would also ensure good compliance with treatment. A similar program is already successfully operating in South Australia.

There are new methods on the horizon for servicing remote areas, including digital imaging and telemedicine (Lin, 2002; Constable, 2000) and there are successes with their use in the provision of services for remote areas in Queensland (Blackwell, 1997). While telemedicine ophthalmology has a role to play in servicing remote areas its use is influenced by many factors such as the spectrum of ocular disorders in the communities, the cultural environment, the concept and beliefs of the individual communities.

Lin had published a paper recommending the use of single-field nonmydriatic monochromatic digital fundus photography with remote interpretation for retinopathy screening and demonstrated that it had superior screening parameters to indirect ophthalmoscopy through dilated pupil (Lin, 2002). Klein and Klein's Editorial correctly drew attention to functions other than the screening tool that must be considered when providing diabetic eye care in the population (Klein, 2002). A visit to the ophthalmologist for screening may establish trust with the patient early during the course of the disease. This encourages the patient to be more compliant with the management of the condition and allows the ophthalmologist to have a role in diabetes education. The detection of other treatable ocular disorders like glaucoma and cataract, which are

more frequent in persons with diabetes, could also be carried out without delay.

In Aboriginal communities a recommendation from a Health Worker (or, in most communities due to the frequent changes in staff, from a previous patient or a nursing sister) may promote the establishment of trust with the communities, health care providers and the individual patients.

In summary, the diversity of the Aboriginal population, the culture, the living environment, the spectrum of other ocular conditions necessitates the establishment of best screening practices for diabetic retinopathy in each region and, if there are major differences between Aboriginal communities of a region, in each community. What is relevant for the clinicians working in any community or region is the need to establish a good working relationship with communities, health care providers and individual patients so that the patient will accept all the knowledge, skills and goodwill of the clinician. From the public health perspective an effective screening program and a skilled clinician are necessary conditions for the control of diabetic eye disease. They are not sufficient however unless these conditions are met with community acceptance and individual trust that is essential to treat the relevant condition in order to avoid blindness.

9.6.2 Implications for health services

The previous section proposed annual screening for diabetic retinopathy among diabetics in Aboriginal communities. Diabetic eye care can only be carried out efficiently in the framework of the general ophthalmic care of the community and that the process should be appropriate and sympathetic to the needs of the particular communities.

This section will address several issues: the technical difficulty of laser treatment in Aboriginal patients in Katherine region, the diagnosis of diabetes in Aboriginal communities and the future service needs of the communities. It will also illustrate the need for an efficient data management center so that community programs can be individualized.

9.6.2.1 Laser treatment in Aboriginal communities in the KRDRS

It has been shown in the previous section that the selection of screening methods has an impact on the acceptance of laser treatment in Aboriginal communities.

This section outlines other factors specific to Aboriginal communities that influence the acceptance of laser treatment.

The Mobile Unit gained access to a portable laser in 1995. Due to the limitations of the funds available for patient travel the Mobile Eye Unit was

under great pressure to carry out as many laser treatment as possible in the communities. It was feasible in major centers where the unit spent several days (sometimes a week), since it allowed a sufficient number of sessions to successfully complete the treatment. In Borroloola, Lajamanu, Wurli-Wurlinjang Health Center nearly all patients had most of their treatment in the respective communities.

In smaller communities there was hardly sufficient time for treatment to be completed and the patients had to travel to a major center to complete treatment. Movements of Aboriginal communities are determined by their traditions. Therefore, not necessarily the closest communities were selected as the place for treatment but the ones most acceptable to Aboriginal people. Also, in some Aboriginal communities the treatment of young males may be associated with shame and embarrassment that prevented treatment to be completed in the communities.

Since laser treatment was carried out in a dark room, it was threatening for many Aboriginal patients from remote communities. The study personnel made a conscious effort to make the patient feel comfortable. This was achieved by treating them as if they had come to a meeting with another community member by offering them a cold drink or a cup of tea, and, in case of a long waiting time, some food. (The long waiting time was usually due to poorly dilating pupils.) Blankets were often required since Aboriginal people soon became cold in the building. Whenever it was possible an Aboriginal Health Worker (preferably of the same gender) was present to support the patient.

The high prevalence of CSME in the Aboriginal diabetic population made it imperative to achieve patient co-operation during treatment. In the first

session sometimes more than 30 minutes was taken up familiarizing the patient with the importance of the chin and front positions. The deep-seated eyes and the prominent frontal ridge did not make the use of contact lenses easy. A common finding was that when the first shot of laser was fired, the patient jumped, moving away not only from the preferred eye position but also from the slitlamp despite special instructions prior to the laser treatment on what to expect. Access to an indirect delivery system (that could have allowed panretinal photocoagulation to be carried out without the use of contact lenses) would have made laser treatment much easier.

Compliance with the necessary follow-up after laser treatment was excellent in the KRDRS. This was due to the increased awareness of health service providers and patients about eye health and the personal knowledge of the communities by members of the Mobile Eye Unit.

The road to a successful laser treatment in Aboriginal communities is much more complex than in non-Aboriginal Australians. Beyond the requirements of trained health professionals willing to take up the challenge of servicing these communities, the screening program, treatment and follow-up must take into consideration the special needs of these communities in building a good personal relationship that is the basis of any successful program in Aboriginal communities. The extent to which this takes time and effort from practicing clinicians is yet to be recognized in measuring efficiency and in funding arrangements for rural eye health programs.

9.6.2.2 Screening for diabetes in Aboriginal communities in the KRDRS

It is not the purpose of this thesis to consider diabetes-related variables in any detail. Variables such as age, time since diagnosis and composition of the diabetic population are only mentioned (see Chapter 5) to the extent they are relevant for the descriptive epidemiology of diabetic retinopathy. Analysis of these variables did raise issues relating to the diagnosis of diabetes in Aboriginal communities.

From 1990 onwards a great emphasis was placed on opportunistic screening for diabetes in Aboriginal communities in Katherine region. The KRDRS has shown that the start of the opportunistic screening campaign had no impact on the random blood sugar levels diabetes was diagnosed with (see Table 5.2.14). Burns has shown that 2100 Aboriginal people serviced by the community health center in Maningrida (East Arnhem region of the Northern Territory) made 26500 presentations per year to the regional health center (Burns, 1998). This study shows that there was ample opportunity for opportunistic screening annually in this Aboriginal population. Since it is most likely that Aboriginal people make at least one annual presentation to the health centers and since health professionals in the communities are highly focused on diabetes it is very unlikely that the very high random blood sugar levels at diagnosis were due to detection bias. In the Australian Diabetes Screening Study, of those diagnosed with diabetes using random BSL measurement, only about 14% of subjects were diagnosed with a random BSL 11.5 mmol/l or higher (Welborn, 1997), compared with 85% of subjects in the KRDRS in both 1993 and 1996.

These results raise the question of whether diabetes in Aboriginal people presents almost subacutely with immediately high random blood sugar levels at diagnosis, or whether despite the vigilance of health service providers in the communities, the Community Health Centers are in a position to diagnose diabetes at an early stage of the disease. Further studies are warranted to answer this question.

9.6.2.3 Future service needs

The KRDRS has shown that those diagnosed with diabetes between 1994 and 1996 were on average five years younger and were diagnosed with diabetes on average two years earlier than those in the 1993 study population. In the newly diagnosed group of the non-core population of 1996 the proportion of males and females were nearly equal compared with a male population of only 30% in 1993. Of those in 1993 diagnosed after 1990 (greater emphasis on opportunistic screening) 32% were younger than 40 at the time of diagnosis compared with 47% of those diagnosed between 1994 - 1996.

These figures suggest that diabetes presents at an earlier age in the newly diagnosed population of the non-core group (diagnosed between 1994 – 1996) than those diagnosed with diabetes between 1990-1993 in the study population of 1993.
The younger age at diagnosis of those diagnosed with diabetes between 1994-1996 suggests that they may be exposed to the pathologic changes of diabetes for a longer period of their lives. As time since diagnosis is a

risk factor for the development of diabetic retinopathy, the burden on ophthalmic services will increase.

At the time of the KRDRS diabetes had already been incorporated into the women's health program in the Northern Territory. The increased proportion of males in the newly diagnosed group and the higher annual progression rates of diabetic retinopathy in males suggest the need to incorporate diabetes education and control in the men's health program as well.

Before and during the KRDRS diabetes was an important topic of discussion among health professionals in the Northern Territory. It was argued by some that emphasis on diabetic retinopathy as a major health project in the communities was not warranted since Aboriginal people had high mortality rates, some of which was due to the macrovascular complications of diabetes. It was argued that Aboriginal diabetics do not live long enough to develop microvascular complications of diabetes, even though there was no published evidence to support this. The high prevalence of VTR in both 1993 and in 1996, the annual progression rates comparable to Mitchell's non-Aboriginal Australian population (Mitchell, 1990) despite much shorter duration of diabetes in the KRDRS suggest otherwise. Therefore, despite the increased mortality risk in Aboriginal diabetics (International Diabetes Institute, 1998) the microvascular complications of ophthalmic disorders present before diabetics die from other diabetes-related complications.

It is impossible to plan clinical services for diabetes and diabetic retinopathy in the communities without understanding the epidemiology of the disease. While there may have been databases available in the clinics,

the KRDRS has shown inadequacies of these individual databases because of failures in their maintenance registering patient movements, deaths, etc. There was no register of Aboriginal eye health in the Northern Territory prior to the establishment of the KRDRS and therefore there was no possibility of prompt analysis of the clinical data in Aboriginal communities. It was not possible to provide up-to-date information for planning ophthalmic services. It was a failure of the KRDRS that despite good results in service provision (Taylor, 1997), the health administration of the Northern Territory could not be persuaded of the value of the database and the information it can provide despite Taylor's recommendation to establish a national database on Aboriginal eye health (Taylor, 1997). Since ophthalmic services to remote communities cease in the beginning of November and do not start until the following March due to the wet season this time could well be used for the analysis of data and planning of services for the next year.

Prompt analysis of data from the KRDRS would have shown that in Lajamanu, Ngukurr and Wurli-Wurlinjang diabetics need special attention since in these communities the retina of a large number of diabetics could not be visualized. It would also have shown that of those with diabetes in the major communities Wurli-Wurlinjang has the highest proportion of subjects with retinopathy. In Borroloola in both years the proportion of diabetics was relatively low compared to the regional total, the proportion of those with diabetic retinopathy was the highest compared to the regional total. In 1993 only 11% of diabetics were living in Borroloola. Between 1994 and 1996, 19% of the newly diagnosed diabetics came from this community. It came as a surprise that 12% of the newly diagnosed diabetics between 1994 and 1996 came from Barunga where only 6% of diabetics of the region were living in 1993. With no change in disease

surveillance this finding warrants further investigation and suggests a need to focus on prevention programs in these communities. The prompt analysis of data could also have justified to the department the time and funds consumed by visits to smaller communities that are affected by diabetic retinopathy.

The foregoing discussion shows that the establishment of a database is of vital importance to target the individual service needs of Aboriginal communities. This would take the "guess-work" out of planning and delivering services and allow the health dollar to stretch further. An ophthalmic database could be linked to a centre with an already existing infrastructure necessary for the efficient and prompt analysis of data.

In summary, on the basis of the results from the KRDRS it is most likely that the burden on ophthalmic services will increase in Katherine region. These data have also shown that ophthalmic care in communities can only be planned using a well-managed database that will allow services to efficiently target the individual needs of Aboriginal communities.

9.7 Further research

While the KRDRS provided a wealth of information regarding diabetic retinopathy in Aboriginal communities it has left some unanswered questions that warrant further research. Some of this research could use already existing data, some will need to collect new data.

9.7.1 Research based on existing data

The following research could be carried out by further analysis of existing data:

- risk of having diabetic retinopathy and the risk of progression
- description of performance indices of fundus photography in screening for diabetic retinopathy
- cost-benefit analysis
- description of general medical characteristics of the diabetic Aboriginal population
- the prevalence and incidence of visual impairment in Katherine region.

9.7.2 Research based on new data

First, the whereabouts of the non-core population of 1993 is uncertain. Following this group would have provided valuable information, however the busy schedule of the Mobile Eye Unit and the cultural sensitivity of Aboriginal people regarding issues of death did not allow follow up of these subjects using medical records.

Further research regarding the diagnosis of diabetes in the Aboriginal population is required. The KRDRS has shown that the random blood sugar level at diagnosis among diabetic Aboriginal people is extremely high compared to that of the Australian population despite the vigilance of

health professionals in the communities. The KRDRS does not provide an answer for the unusually high random BSL at diagnosis of diabetes. It may be that diabetes presents subacutely in the communities with high random BSL already at the time of diagnosis or perhaps health centers do not diagnose diabetes early due to the mobility of the population.

The progression of diabetic retinopathy in Aboriginal communities could be explored further. The KRDRS was hampered by small numbers in the non-lasered core population and by a relatively short observation period. Larger, longer-term studies are needed to fully describe current progression rates. Data from the KRDRS might usefully be pooled with further studies.

There have been numerous studies showing that impaired vision increases the risk of five-year mortality. In the KRDRS the visual impairment in the diabetic Aboriginal population is comparable to the general Australian population approximately 30 years older. The life expectancy in Aboriginal Australians is on average 20 years less than in non-Aboriginal Australians. It is unclear, if the level of visual impairment represent premature ageing in the KRDRS study population. This requires further studies.

It is evident from the findings of this study that the Mobile Eye Unit in the Katherine region should be revived and in 2006/2007, 10 years after the KRDRS, review of the communities should be carried out again. This may lead to a better understanding of trends in eye disorders and a provision of better services in the communities.

9.8 Conclusions and recommendations from the KRDRS

Conclusions:

The KRDRS has shown that diabetic retinopathy affects Aboriginal Australians at least to the extent of non-Aboriginal Australians. It is especially so if the high prevalences of clinically significant macular edema and vision threatening retinopathy are considered after a short duration of diabetes. The high annual incidence rates of clinically significant macular edema and VTR after a short duration of diabetes suggest an accelerated progression of the disease.

More than 90% of visual impairment, monocular and binocular blindness in the diabetic Aboriginal population are preventable. The major cause of blindness was cataract at both cross-sections of the study; diabetic retinopathy was responsible for only a small proportion of visual impairment.

Visual acuity is a poor indicator of the presence of diabetic retinopathy, clinically significant macular edema and vision threatening retinopathy.

The current OATSIH guidelines (OATSIH 2001) regarding detection and management of diabetic retinopathy in Aboriginal Australians are impractical and not feasible. The shortfalls of these recommendations reflect the lack of data on (diabetic and non diabetic) ocular conditions in Aboriginal communities and the separation of case finding and case management in Aboriginal Australians.

Recommendations:

1. Screening for diabetic retinopathy should be part of the general ophthalmic care of the community. This care should be provided at the standards as in other parts of Australia.
2. Screening should be carried out using methods that ensure identification of cases as well as provision of further treatment.
3. Due to the differences between communities and between regions the state branches of the Royal Australian College of Ophthalmologists should establish the best methods for screening and treatment reflecting community needs and community acceptance. These methods should be regularly evaluated and modified according to changes in communities.
4. As in the National Trachoma and Eye Health Program, the Royal Australian College of Ophthalmologists should be given the necessary means to provide services to these communities. Currently, ophthalmologists who are called upon to provide service to remote communities are placed at considerable financial disadvantage if they do so.
5. The method of laser treatment should be carried out in a non-threatening environment where the patient and service providers feel comfortable. Since treatment may involve several sessions and a longer stay of patients away from their community, it is imperative to reexamine the current travel arrangements for patients.
6. Evidence based individualised ophthalmic services should be established in Aboriginal communities. To achieve this, as suggested previously (Taylor, 1997), a national database on Aboriginal eye health must be created.

7. Further analysis of the database from the work of the Mobile Eye Unit must be carried out. It is essential to gain better understanding of eye disorders in Aboriginal communities.
8. The detailed documentation on methods, procedures, criteria recorded in this thesis should be used for comparison with further research.
9. A multidisciplinary approach is required to prevent the further rise of diabetes and its complications in Aboriginal communities.

Chapter 10

10. References

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Chapter 11

Appendix 1

SURNAME 1 :
CHRISTIAN NAME1:
D.O.B.1:
Medicare no1:
Settlement:
HRN DRWN:

SURNAME2:
CHRISTIAN NAME2:
D.O.B.2:
Medicare no2:

DATE SEEN:
HRN GOVE:

SEX:
HRN KATH:

Region: 1. Darwin region 2. Gove region 3. Katherine region

Ethnicity: 1.traditional Aboriginal pt in traditional communities
2.non-traditional Aboriginal pt in traditional communities
3.traditional Aboriginal pt in non-traditional communities
4.non-traditional Aboriginal pt in non-traditional communities
5.Asian pt 6.European pt

Uncorrected visual acuity right eye:

Uncorrected visual acuity left eye:

Full visual acuity right eye:
Full visual acuity left eye:

PEH:

PMH

IDDM: NIDDM: DB diagnosed in: DB diagnosed by: Hypertension:
Diabetes medication: 1.diet only 2.oral 3. insulin
IOP right eye: IOP left eye:

Anterior segment:

Posterior segment:

C/D ratio right eye: C/D ratio left eye::

Db retinopathy right eye: 1. mild BDR 2. moderately severe BDR
3.severe BDR 4.proliferative BDR

Db retinopathy left eye 1. mild BDR 2. moderately severe BDR
3.severe BDR 4.proliferative BDR

maculopathy right eye: maculopathy left eye:

previous laser treatment for proliferative retinopathy right eye:
previous laser treatment for proliferative retinopathy left eye:

previous laser treatment for maculopathy right eye:
previous laser treatment for maculopathy left eye:

cataract type right eye: 1. cortical 2.subcapsular 3.nuclear
grading: 1.nil 2.moderate 3. severe 4.aphake 5. pseudophake

cataract type left eye: 1. cortical 2. subcapsular 3. nuclear
grading: 1. nil 2. moderate 3.severe 4.aphake 5.pseudophake
snowflake right eye: snowflake left eye:

random BSL (if myopic): spot urine dipstick protein:
spot urine dipstick glucose: spot urine dipstick ketones:
BP systolic sitting: BP systolic standing:
BP diastolic sitting: BP diastolic standing:
Body weight(kg): Body height (m):