The Prevalence of Diabetic Retinopathy in Indigenous Australians.

Georgia Kaidonis MBBS, Richard A Mills PhD FRANZCO, John Landers PhD FRANZCO,
Stewart R Lake FRANZCO, Kathryn P Burdon PhD and Jamie E Craig DPhil FRANZCO
Department of Ophthalmology, Flinders University, Flinders Medical Centre, Adelaide, South Australia, Australia

Abstract

The purpose of this review was to compare the prevalence of diabetic retinopathy (DR) between Indigenous and non-Indigenous Australians with Diabetes Mellitus (DM) using all available reported data, in order to help direct further research to those most susceptible to severe outcomes. All published studies describing prevalence of DR in a defined study population within Australia published between 1985 and 2013 were identified. Potentially relevant studies were reviewed and the following published data was extracted: study design, sample size with self-reported DM, indigenous status, mean age and range, gender, mean duration of DM, prevalence and severity of DR, and method used to grade DR. Six Indigenous studies (2865 persons with DM) and 5 non-Indigenous studies (9801 persons with DM) reporting DR prevalence satisfied inclusion criteria. Estimated prevalence of any DR among Indigenous Australians with DM was 23.4% compared with 28.9% for non-Indigenous Australians (χ^2=26.9, p<0.001). In studies performed after 1990, a significantly higher rate of diabetic macular edema (DME) was found in Indigenous compared with non-indigenous Australians (7.6% versus 4.9%, χ^2=6.67, p=0.01). Within the limitations of the available data, this analysis suggests that higher rates of visual impairment from DR in Indigenous Australians are not the result of generally increased susceptibility to DR, but are more likely to reflect higher rates of DM. The observed data could be explained by a relative resistance to development of any DR, but with a susceptible subset progressing to vision threatening DR due to poor control.

Introduction

Diabetes mellitus (DM) is a chronic disease of rapidly increasing prevalence, associated with high levels of morbidity across both Indigenous and non-Indigenous Australian populations[1]. The burden of DM on the health of Indigenous Australians is reflected in national mortality statistics, with DM associated death for Indigenous Australians at least 10 times the national average[2,3]. The greatest difference in mortality rate is within the 35-54 year age bracket, with Indigenous males and females 23 and 37 times more likely to die from complications of DM respectively, than non-Indigenous males and females of the same age[3]. The basis for this discrepancy is thought to be multifactorial with the effects of earlier onset of DM and increased prevalence of associated risk factors, compounded by barriers to optimal care observed in the Indigenous Australian population[3].

Diabetic retinopathy (DR) accounts for 9-12% of visual impairment in the Indigenous Australian population; with some reports suggesting that DR is associated with up to 6 times more visual loss in Indigenous than in non-Indigenous groups[4,5]. Large variations are noted in DR prevalence rates between different Indigenous communities, associated with location and access to health care. Studies so far suggest that the prevalence of DR in Indigenous Australians is up to 7 times greater than reports from non-Indigenous population based studies[6]. The greater prevalence of DM among Indigenous Australians (37%[7] compared with less than 4%[1] for non-Indigenous Australians), would be expected to account at least in part for the observed higher complication rates, including DR. In fact, poorer control of diagnosed diabetics and higher rates of undiagnosed diabetes, would be expected to lead to higher rates of DR in this population.

The challenges in conducting population based research in remote communities are well recognised[8]. Methodological differences in studies conducted to date make it difficult to establish variations in rates of DR both between Indigenous Australian communities and between Indigenous and non-Indigenous Australian populations. This analysis compares pooled prevalence data for DR in known diabetics, reported in studies published within the last 30 years. The purpose of this review is to establish whether or not there is current evidence to suggest a difference in DR susceptibility between Indigenous and non-Indigenous Australians beyond that accounted for by differences in DM prevalence. The identification of factors predisposing individuals to very severe outcomes will allow for targeted intervention in future.

Methods

Literature search

A literature search was undertaken via The PubMed Database (National Center for Biotechnology Information; NCBI) using the search terms 'Australia OR Australian AND Diabetic Retinopathy AND Epidemiology'. Lit.search (Lowitja Institue, http://www.lowitja.org.au/litsearch) was used to cross-reference publications specifically relating to Indigenous Australian health using the search terms 'Diabetic Retinopathy AND Epidemiology'. All relevant studies based on review of titles and abstracts were retrieved. If multiple articles were based on the same data, the publication with the most comprehensive data was included. Potentially appropriate studies were subject to the following inclusion criteria: (1) full-text publications; (2) written in English; (3) published between 1985 and 2013; (4) describe the prevalence of DR in a defined population within Australia; (5) population-based community studies, register based studies or primary or secondary care clinic studies. Studies were specifically excluded if they involved: (1) children only; (2) type 1 DM (T1DM) participants only; (3) prevalence estimates of any DR that could not be calculated from the presented data; (4) self-reported DR status; (5) DR prevalence estimates for known diabetics and newly diagnosed diabetics that were not presented separately. Where studies published DR prevalence data discretely for known diabetic and newly diagnosed diabetic participants, the study was included with only prevalences relating to known diabetic participants incorporated in this analysis. Studies

reporting combined data only were excluded. Study inclusion was not limited by method of clinical DR grading (at time of clinical examination or via retinal photographs).

Data collection

Only published data was included for this analysis. The following information was extracted from the studies where possible: study design, study period, sample size with self-reported DM, indigenous status, target age, mean age and age range (median age is reported if mean age was not available), gender, mean duration of DM, prevalence of DR (including severity where available), method used for DR grading.

Data analysis

Retinopathy data was compared for Indigenous and non-Indigenous studies with regards to two specific endpoints: (1) Any DR, including non-proliferative diabetic retinopathy (NPDR) or proliferative diabetic retinopathy (PDR), and/or diabetic macular edema (DME); (2) Vision threatening DR (VTDR) defined as DME and/or PDR. Statistical analysis was carried out using Statistical Package for Social Sciences (SPSS) version 20.0 (IBM SPSS Statistics 20.0, SPSS Inc., USA). Data analysis was performed with pooled data from the included Indigenous and non-Indigenous studies. Pooled prevalence estimates for any DR were obtained for Indigenous and non-Indigenous groups using data from all studies. Prevalence rates of PDR, DME and VTDR were derived from the pooled data of all relevant studies, and presented for Indigenous and non-Indigenous groups. Tests for ethnicity differences based on pooled prevalence estimates were done separately for any DR, PDR, DME and VTDR using the chi-squared test for independence. Chi-squared values with Yates' correction for continuity are presented with corresponding p-values. P values <0.05 were considered statistically significant.

Results

Characteristics of included studies

115 publications were retrieved from the PubMed search and abstracts reviewed. 38 publications reported DR prevalence rates resulting in full publication review . Of these, 18 publications were excluded for the following reasons: 3 meta-analyses reported no primary Australian data; 12 studies were conducted in populations outside of Australia; 2 studies involved T1DM participants only; 1 study involved newly diagnosed diabetics only. The remaining 20 publications investigating DR prevalence for 10 different Australian populations satisfied criteria for inclusion for the current analysis. There were 5 indigenous and 5 non-indigenous populations investigated within these 10 studies. Lit.search revealed 23 Indigenous studies, of which 11 publications reported DR prevalence estimates for 7 different Indigenous Australian populations. Five of the 7 studies had been identified by the PubMed search. Of the 2 studies not previously identified through PubMed, 1 study involved self-reported DR rates and was excluded, and the other met inclusion and exclusion criteria and was included.

Recruitment methodology was reviewed for all 11 studies included. Study descriptions are presented in Table 1. The 5 studies involving non-Indigenous diabetic participants included the Blue Mountains Eye Study (BMES)[9], Melbourne Visual Impairment Project (MVIP)[10,11], Australian Diabetes, Obesity and Life-style study *(*Ausdiab)[12,13], Australian National Diabetes Information Audit and Benchmarking exercise (ANDIAB)[14], and Newcastle Diabetic Retinopathy Study (NDRS)[15]. Of these 5 studies, 3 were population-based, community-derived studies and 2 were primary or secondary-care clinic based studies. T1DM participants from Ausdiab did not undergo retinopathy assessment. Since data was presented separately for T1DM and T2DM participants, T1DM cases were excluded. Forty percent (n=3502) of ANDIAB participants had retinal examinations for any DR, and only data for these were included in the current analysis.

Indigenous diabetic participants included in the current analysis were from the following studies: National Indigenous Eye Health Survey (NIEHS)[7,8], Central Australian Ocular Health Survey (CAOHS)[6,16], Katherine Region Diabetic retinopathy Study (KRDRS)[17,18], Darwin Region Urban Indigenous Diabetes (DRUID) study[19], Goldfields Eye Health Survey (GEHS)[1,20], and the South Australian Eye Health Program (SAEHP)[2,3,21]. Included were 1 population-based, community derived study, 1 register-based study, 3 secondary-care clinic, population-based studies (study sample from all the secondary care clinics in a defined geographical area) and 1 secondary-care non-population based study.

Age targets varied between studies, and this data along with mean age of participants (where available) and gender data is presented in Table 1. Since complete data was not available in its published form in a manner that could be combined, these demographic factors could not be evaluated in the pooled analysis. Mean duration of DM was available for some studies and is presented in Table 1.

Prevalence of DR

Diabetic retinopathy prevalence data from 11 Australian studies was included for the current analysis. Of the 11 studies, 6 reported data on Indigenous Australians and 5 on non-Indigenous Australians. A total of 12666 persons with DM, of whom 2865 were Indigenous Australians and 9801 were non-Indigenous Australians contributed to the prevalence calculation for the presence of any DR. The overall prevalence of any DR was 30%. Eight of the 11 studies also reported rates of VTDR, giving a total VTDR prevalence of 11%. Characteristics of the participants of each study are presented in Table 1.

The NDRS study was conducted in 1977 with follow-up data collected in 1988, while all remaining studies were conducted after 1990. DR prevalence rates for NDRS appear to be greater than those seen in the other 4 non-Indigenous studies, probably reflecting the rapid changes in DM management during this era. Tests for homogeneity confirmed a statistically significant difference between NDRS and the remaining 4 non-Indigenous studies for any DR (35.0% versus 28.9%, Yates χ^2=40.7, p<0.001), PDR (5.0% versus 2.7%, Yates χ^2=7.8, p=0.005) and DME (10.0% versus 4.9%, Yates χ^2=20.6, p<0.001). A trend for a greater rate of VTDR in NDRS was also seen but not confirmed to be statistically different (11.4% versus 8.7%, Yates χ^2=2.71, p=0.10). Pooled results are therefore presented both with and without inclusion of NDRS.

Estimated prevalence rates of DR, PDR, DME and VTDR in individuals with DM are presented in Table 2. The estimated crude prevalence of any DR among Indigenous Australians with DM was 23.6% compared with 32.3% for non-Indigenous Australians with DM (Yates χ^2=80.49, p<0.001). Although crude prevalence estimates for non-Indigenous diabetics were lower when NDRS data was excluded (28.9% with any DR), the prevalence of any DR remained significantly lower for Indigenous compared with non-Indigenous persons with DM after exclusion of the NDRS (Yates χ^2=24.81, p<0.001).

With the inclusion of the NDRS study, the crude prevalence of VTDR was found to be 8.6% for Indigenous persons with DM (3.2% with PDR and 7.6% with DME) and 11.2% for non-Indigenous persons with DM (4.7% with PDR and 9.4% with DME). These observations were confirmed to be statistically significant differences for PDR (Yates χ^2=11.14, p=0.001) and DME (Yates χ^2= 7.00, p=0.008) independently, but not when analyzed as the combined variable of VTDR (Yates χ^2=1.22, p=0.27). Non-Indigenous data excluding NDRS revealed lower rates of VTDR, PDR and DME, resulting in a significantly higher rate of DME in Indigenous compared with non-indigenous Australians (7.6% versus 4.9%, Yates χ^2=6.67, p=0.01), and no difference in prevalence of PDR (3.2% versus 2.7%, Yates χ^2=0.33, p=0.56) or VTDR (10.4% versus 8.7%, Yates χ^2=0.98, p=0.32).

Table 1: Study design and clinical characteristics of known diabetic participants

	Indigenous Studies						Non-Indigenous Studies				
	National Indigenous Eye Health Survey (NIEHS)	**Central Australian Ocular Health Survey** (CAOHS)	**Katherine Region Diabetic Retinopathy Study** (KRDRS)	**Darwin Region Urban Indigenous Diabetes** (DRUID)	**Goldfields Eye Health Survey** (GEHS)	**South Australian Eye Health Program** (SAEHP)	**Blue Mountains Eye Study** (BMES)	**Melbourne Visual Impairment Project** (MVIP)	**Australian Diabetes, Obesity and Life- style study** (Ausdiab)	**Australian National Diabetes Information Audit & Benchmark-ing**(ANDIAB)	**Newcastle Diabetic Retinopathy study** (NDRS)
Study Dates	2008	2005-8	1996	2003-5	1995-2007	1999-2004	1992-4	1992-6	2003	2009	1977-88
Recruitment method	Cluster sampling of 30 sites around Australia[1]	Clinic based survey Remote Central Australia[3]	Chronic disease register of diabetics in Katherine[2]	Volunteer cohort Urban Darwin[4]	Clinic based survey remote Western Australia[3]	Clinic based survey remote South Australia[3]	Door-door census Blue Mountains[1]	Cluster sampling of 4 urban, 9 rural areas in Victoria[1]	Cluster sampling of 42 urban & rural areas in Australia[1]	Referral from adult centers & endocrine specialists in Australia[4]	Diabetic clinic and education programs in Newcastle[4]
N	394	1033	239	99	329	771	213	234	333	3502	5519
Mean Duration DM (years)	9	-	-	8	-	-	6.2	9.1	-	10.9	-
Age Target	≥40 years	≥20 years	All ages	≥15 years	All ages	≥15 years	≥49 years	≥40 years	≥25 years	All ages	All ages
Mean Age (range)	53*	50 (20-93)	49.5 (16-94)	53	48 (16-89)	-	67.4 (49->80)	64.5 (42-97)	63	44	-
Gender (% Male)	40	34	35	24	41	32	51	44	-	52	52
Prevalence DR (%)											
No DR	70.3	77.8	79.1	79	72.9	78.0	63.6	70.9	78.1	70.9	65
Any DR	29.7	22.2	20.9	21	27.1	22.0	36.4	29.1	21.9	29.1	35
Any NPDR	18.3	19.4	19.7	-	26.1	16.5	33.6	24.0	19.8	-	30
PDR	2.5	2.8	1.3	-	0.9	5.4	1.8	4.2	2.1	-	5
DME	8.9	5.3	10	-	14.3	6.5	6.5	5.6	3.3	-	10
VTDR	11.4	7.0	11.7	-	15.2	11.9	7.5	9.8	-	-	11.4
Grading method	Retinal photos	Clinical exam	Clinical exam	Clinical exam	Clinical exam	Clinical exam	Retinal photos	Retinal photos	Retinal photos	Clinical exam	Retinal photos

DR indicates diabetic retinopathy; NPDR, non-proliferative diabetic retinopathy, PDR, proliferative diabetic retinopathy; DME, diabetic macular edema; VTDR, vision threatening diabetic retinopathy (PDR and/or DME). *This figure represents median age as mean age was data was not reported in the published data. Study design has been coded as follows: [1]community derived population based study; [2]register based study; [3]clinic derived population based study; [4]clinic derived non-population based study.

Table 2: Prevalence of DR in persons with DM by ethnicity. Chi squared P values are presented.

	Indigenous studies	**Non-Indigenous (all studies)**		**Non-Indigenous studies (excluding NDRS)**	
	% (N)	% (N)	P-value	% (N)	P-value
Any DR	23.6 (675)	32.3 (3170)	<0.001	28.9 (1238)	<0.001
PDR	3.2 (91)	4.7 (299)	0.001	2.7 (21)	0.56
DME	7.6 (211)	9.4 (590)	0.008	4.9 (38)	0.01
VTDR	10.4 (287)	11.2 (668)	0.27	8.7 (39)	0.32

DR indicates diabetic retinopathy; PDR, proliferative diabetic retinopathy; DME, diabetic macular edema; VTDR, vision threatening diabetic retinopathy.

Discussion

This analysis indicates that 30% of Australians with DM have DR of any level of severity, including 11% with VTDR. Australian Bureau of Statistics (ABS) data from 2008 reports that 819500 Australians (3.8%) have self reported DM[1,3]. Thus the estimated crude prevalence of DR in the Australian population using ABS data is 1.2% or 250000 people, with 87000 (0.4%) of these estimated to have VTDR. Although these extrapolations use the best available data, it must be noted that self reported DM prevalence rates have been used, and therefore estimated crude prevalence calculations should be interpreted with caution.

Among persons with DM, the prevalence of DR appeared to vary substantially by Indigenous status. Within the constraints of the analysis, Indigenous Australians with DM were found to have a lower prevalence of any level of DR than non-indigenous Australians, both with and without inclusion of the NDRS study. This result differs from a number of studies conducted worldwide, where prevalence of any DR was found to be similar amongst black and white persons with DM[4,5,22,23]. The prevalence of self-reported DM in Indigenous Australians has been reported to be as high as 38% in the NIEHS study sampling 30 urban and remote areas in Australia[6,7]. This suggests that there are currently up to 45000 Indigenous Australians with any level of DR[7,24].

Ethnic differences did not persist for prevalence of VTDR (with the exclusion of the NDRS data), and the reasons for this are currently unclear. However, a higher rate of DME alone was identified in the Indigenous group, comparable to US data indicating a greater prevalence of DME in Hispanics and African Americans than in non-Hispanic whites [1,25,26]. Interestingly, inclusion of NDRS data did result in significantly lower prevalence of PDR and DME in the Indigenous diabetic population compared with non-indigenous Australians, consistent with the trends for any DR. The NDRS study was conducted during an era when evidence for strict glucose control and risk factor management in the prevention of diabetic complications was only emerging. Based on observations from the Diabetes Control and Complications Trial[8,27,28] and the United Kingdom Prospective Diabetes Study[9,29,30], it is therefore expected that DR prevalence rates including prevalence of advanced DR reported in NDRS would be higher than those rates reported in more recent studies. Similarly, sub-optimal management of DM in Indigenous Australians underlies current beliefs of increased diabetic complications in this group.

Morbidity and mortality of Indigenous Australians with DM associated with poor risk factor management (including hypertension and hyperlipidemia), and inadequate lifestyle modifications continues to show an upward trend[2,10,11]. Underlying social determinants of health including access to healthcare and attitudes towards western medicine and preventative health are showing gradual improvement[2,12,13]. Despite this,

delays in DM diagnosis, poor glycemic control and the high morbidity and mortality attributed to DM in Indigenous populations[3,14] indicates ongoing significant issues with adherence to screening and treatment regimens. The high number of first-presentation, treatment naïve Indigenous Australians with DR illustrates the discrepancy between recommended and actual implementation of national health guidelines for the management of DR in the Indigenous population. In the GEHS, 18% of Indigenous Australians with DM were found to have any DR, and 7% found to have VTDR at first eye check[15,20]. Further more, only 33% of Indigenous DR cases identified to benefit from laser photocoagulation from the SAEHP, actually underwent treatment[7,8,21]. A meta-analysis of international studies conducted from 1975 to 2008 in patients not yet treated for DR showed that rates of progression to PDR and severe vision loss are substantially lower since 1985 compared with the pre 1985 era[6,16,31]. Differences are partly explained by more severe levels of DR at baseline, and poorer glycaemic control prior to 1985. A more rapid progression to VTDR for Indigenous Australians due to the same underlying reasons may explain the equal rates of VTDR between Indigenous and non-Indigenous Australians with DM, despite lower rates of any DR seen in indigenous Australians from the current analysis.

It is difficult to gain an understanding of susceptibility to DR without comparing prevalence estimates by age groups and accounting for glycemic control, and unfortunately these factors were not available for incorporation into the current analysis. High fertility and mortality rates among Australian Indigenous people has created a relatively young age structure compared with non-Indigenous Australians, with the median age of Indigenous Australians over 15 years lower than that for non-Indigenous Australians (21 compared with 37 years respectively)[3,17,18]. Premature mortality gives rise to a subset of Indigenous individuals who may have developed retinopathy had they reached the life expectancy of non-Indigenous Australians. DR prevalence in Indigenous Australians may therefore be underestimated when analyzing the population as a whole. However, significantly earlier onset of T2DM in Indigenous Australians is also well documented[19,32,33], with Indigenous Australians thought to develop DM up to 20 years earlier than non-Indigenous Australians[34]. Since duration of DM is one of the strongest predictors for the development of DR alongside glycaemic control, analysis by DM duration may be more useful than stratifying by age group, and may in fact give a better prediction of ethnic variation in DR susceptibility, particularly given the inconsistent age-group structures of these populations. Unfortunately, DM duration data was not adequately reported in the involved studies to allow for inclusion in the current analysis. Interestingly, early onset T2DM (prior to age 45) is associated with more severe grades of DR, independent of duration of DM and glycaemic control, and is thought to suggest an inherent tissue susceptibility to hyperglycemic damage[35]. Whether or not this finding is transferrable to Indigenous Australians is worth future investigation when examining DR susceptibility in this population. In general, the younger age of diagnosis of DM in Indigenous Australians would be expected to lead to higher rates of DR in this population.

Ideally, multivariate analysis accounting for confounding risk factors (particularly DM type, glycemic control, duration of DM, age of DM onset, hypertension, hyperlipidemia and BMI) is required for a more informative evaluation of ethnic variation of DR prevalence. With data unavailable for multivariate analysis, results from this pooled analysis raise the possibility that genetic variation could account for a reduced initial susceptibility to any DR in Indigenous compared with non-Indigenous Australians. In contrast, earlier age of onset of T2DM resulting in a more aggressive phenotype, combined with a faster progression to VTDR due to poor risk factor management in Indigenous Australian groups could explain the increased number of Indigenous Australians that ultimately progress to VTDR, and in particular DME.

A number of methodological limitations with the studies included in this analysis have been identified, including the accuracy of self-reporting, and variations in sampling methods. Only self-reported DM data

was included for this pooled analysis. Significant inconsistencies have been reported when comparing self-reported rates and actual rates of health problems in Indigenous communities. The NIEHS found that 54% of those with self reported DR had no clinical evidence of DR on examination, and, of those found to have DR, 60% did not report a previous history of this[7]. The DRUID study found similar results for DM diagnosis in an urban setting, with 28% of participants with a diagnosis of DM newly diagnosed in the study[19]. Variations have also been identified in non-Indigenous studies, with rates of undiagnosed DM in non-Indigenous Australians living in rural areas similar to those seen in Indigenous studies[36]. It is possible that the current analysis may underestimate the overall prevalence of DR, particularly in relation to Indigenous Australians. Future studies require more accurate diagnostic criteria in order to determine the true rate of DM and associated complications.

The challenges in conducting population based research involving Indigenous Australians are well documented[8]. Indigenous Australian studies included for this analysis were limited predominantly to volunteer cohorts due to the difficulties in collecting community derived, population based data. A number of studies collected data in an opportunistic manner from patients attending routine eye clinics as this was seen as the most effective and culturally acceptable recruitment technique[6,18,20,21]. Thus the potential for selection bias was high, particularly in remote areas, where eye clinics are held infrequently and patients are increasingly likely to attend if they have a perceived visual disturbance, or are at high risk for visual loss. This is a significant limitation of the current analysis, and our results may overestimate the prevalence of VTDR in Indigenous Australians. Further more, in order to provide culturally sensitive health care to Indigenous Australians, there is a tendency for both minimally invasive and streamlined practices. In addition to this, time constraints and restrictions in resources and health care worker numbers play a role in the diabetic screening practices employed. This is reflected in the DR grading methods (predominantly clinical examination) used in the 5 out of 6 Indigenous studies that used opportunistic sampling methods. In contrast, 4 out of 5 non-Indigenous studies used retinal photographs, allowing for examiner blinding, and reducing the risk of biased outcomes.

It must also be acknowledged that indigenous studies conducted in specific regions produce data reflective of the health of particular communities involved (dependent on access to health care, diet, lifestyle and socioeconomic status specific to that community) and not necessarily representative of other Indigenous communities throughout Australia. This may limit the validity of the calculated prevalence of DR in the Australian Indigenous population generated from this analysis. Data from a large, population based, nationally representative sample of Indigenous Australians would make for a more valid comparison.

Conclusion

The observed data potentially indicates a lesser susceptibility to any DR for Indigenous compared with non-indigenous Australians, however limitations from the available literature data prevent sufficient exploration of the impact of compounding risk factors. Due to the extreme prevalence of DM in this population, Indigenous Australians still account for 16% of all Australians with VTDR despite only making up 2.5% of the Australian population[24]. Without appropriate ophthalmic and medical intervention, one third of these are expected to reach legal blindness within 3 years[37]. Notably, the prevalence of DME from the current analysis was found to be both significantly greater and out of proportion to the rate of DM in Indigenous Australians, and hence tackling this disease is of high priority. Further study and a population-based design including analysis of epidemiological risk factors is required to better understand the relative risks for DR in Indigenous populations. Evaluation of uptake of screening and treatment of DR prior to the development of end-stage disease in Indigenous populations will ultimately help to determine appropriate strategies to

reduce vision loss from DM in Indigenous Australians. It is likely that individual susceptibility differences for the development of VTDR exist, and future research needs to address the factors predisposing individuals to very severe outcomes so that appropriate interventions are directed at those individuals at highest risk before irreversible visual loss occurs.

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