5 Description of results

This section provides an overview of the results of the review, based on the tables shown in Chapter 4 and Appendix 4. The strength of each statement is indicated by the group designation.3

The large scope of the review meant that some complex areas were not studied in as much detail as necessary to make fully informed conclusions (eg diet and supplements). Areas where a further, more focused literature review may be helpful for the development of public health messages are discussed in Chapter 6.

For many other areas, there have been few or no primary research studies, and further studies are therefore needed to answer the relevant questions.

5.1 Eye disease

5.1.1 Smoking and eye disease

As was expected based on previous reviews, papers published from 1997 to 2006 and included in this review confirmed that smoking is associated with the development of cataract and AMD (Group 1). There is not enough data to define a threshold smoking level for this effect. See also Chapter 6 for information on family history and myopia in relation to AMD.

Results for an association with development of glaucoma are inconsistent (Group 5).

No relevant studies were found for this time period for retinitis pigmentosa (Group 7).

5.1.2 Age or ageing and eye disease

The link between ageing and macular degeneration is well established.

Papers published from 1997 to 2006 and included in this review showed that:

- the prevalence of cataract increases sharply with ageing, particularly from about 60 years onwards (in one study, prevalence increased from 1% to 12% at 65–69 years) (Group 1)
- the prevalence of primary open-angle glaucoma (POAG) appears to increase with ageing, based on cross-sectional studies; we did not find any higher level studies (eg cohort studies) of this association (Group 2)

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3 Groups are as follows (see Section 3.2 and Table 3.2 for further details):
Group 1 — Clear association/causality
Group 2 — Possible association/causality (more research needed)
Group 3 — Lack of association/causality
Group 4 — Possible lack of association/causality (more research needed)
Group 5 — Conflicting results
Group 6 — Possible protection
Group 7 — No studies
• prevalence of amblyopia increases up to three years of age and the depth also increases with age (Group 2); it is therefore important to identify young children with anisometropia so that the condition can be treated before the development of amblyopia.

• results for whether a specific age or ageing is associated with development of diabetic retinopathy are conflicting; the best quality study found for this issue (Blue Mountains Eye Study) showed no statistically significant association with ageing (Group 5); however, further research is needed on the relationship between incidence of DR with time since onset of diabetes and ongoing increase in DR with ageing in diabetic patients (see Chapter 6).

No relevant studies were found in the search period for the effect of ageing on retinitis pigmentosa or trachoma (Group 7).

As noted in Section 3.1, there is a distinction to be made between the age of onset or detection of eye disease (such as early childhood and amblyopia) and disease that occurs progressively with ageing (such as cataract and AMD). Care is needed in public health messages to distinguish these two issues (eg for diabetic retinopathy).

5.1.3 Alcohol consumption and eye disease

Papers published from 1997 to 2006 and included in this review showed that drinking increases the risk of nuclear, cortical and posterior subcapsular cataracts (Group 1). However, it is difficult to define a threshold drinking level for this effect. The lowest level that showed an effect in the included studies was 91 g pure ethanol per week (equivalent to nine Australian standard drinks).

The relationship between alcohol and AMD is difficult to evaluate due to the number of variables, including the different types and symptoms of AMD, definitions of alcohol intake and types of alcohol. However, the majority of the included literature suggests that drinking more than 6 beers per week increases the risk of developing drusen and drinking more than about 3 drinks per day, particularly of wine or spirits, is associated with development of AMD (Group 2).

Although there may be an association between alcohol consumption and diabetic retinopathy, more research is needed to clarify this effect (Group 2).

There are conflicting results on the effect that alcohol has on the development of glaucoma and its major risk factor, intraocular pressure (Group 5).

No relevant studies were found in the search period for the effect of alcohol consumption on amblyopia, retinitis pigmentosa or trachoma (Group 7).

5.1.4 Eye infections and eye disease

Papers published from 1997 to 2006 and included in this review showed the following possible associations (all Group 2):

• eye infections (conjunctivitis and toxoplasmosis) appear to be linked to cataract
• amblyopia appears to occur in some cases of eye infection (very small study)
• eye infection appears to be associated with the development of retinopathy in people with diabetes
• a range of infectious agents (eg herpes zoster, cytomegalovirus and nematodes) appear to be associated with glaucoma
• there may be a link between infection with *Chlamydia pneumoniae* and macular degeneration.

No relevant studies were found in the search period for the effect of eye infections on retinitis pigmentosa (Group 7). No studies were found for an association between trachoma and other eye infections (Group 7).

### 5.1.5 UV exposure/sunlight and eye disease

Papers published from 1997 to 2006 and included in this review indicate that exposure to medium-wave ultraviolet light (UVB) is associated with the development of cortical cataract (Group 1).

Exposure to UV in the teenage years and 30s is associated with increased risk of AMD-related pathologies (drusen and pigmentation) and early AMD, although outdoor exposures (eg working outdoors) did not increase risks (Group 2). Wearing sunglasses and hats for at least half the time was protective for people with the highest levels of exposure when measured at 10 years (but not at 5 years).

No relevant studies were found in the search period for an association between exposure to UV or sunlight and amblyopia, diabetic retinopathy, glaucoma, retinitis pigmentosa or trachoma (Group 7). Papers published from 1997 to 2006 and included in this review indicate that exposure to medium-wave ultraviolet light (UVB) is associated with development of cortical cataract (Group 1).

Papers published from 1997 to November 2008 and included in this review indicate that exposure to medium-wave ultraviolet light (UVB) is associated with development of pterygium and ocular surface neoplasia (both Group 1).

See Chapter 6 for further information about sunlight exposure and myopia.

### 5.1.6 Injuries and accidents and eye disease

Papers published from 1997 to 2006 and included in this review indicate that injuries and accidents are clearly associated with an increased risk of cataract, amblyopia and glaucoma (Group 1).

Injuries and accidents appear not to increase the risk of AMD (Group 4).

No relevant studies were found in the search period for an association between injuries and accidents and diabetic retinopathy, retinitis pigmentosa or trachoma (Group 7).

### 5.1.7 Corticosteroids and eye disease

Papers published from 1997 to 2006 and included in this review show that:
• oral corticosteroids can increase the risk of ocular hypertension or OAG in older people (Group 1)
• inhaled corticosteroids can increase the risk of glaucoma and intraocular hypertension in people who take high doses of this medication for long periods of time, or for those with a family history of glaucoma (Group 1) and may also be associated with cataracts (Group 2)
• topical corticosteroids used near the eyes may increase the risk of glaucoma (Group 2).

No relevant studies were found in the search period for an association between corticosteroids and amblyopia, diabetic retinopathy, retinitis pigmentosa or trachoma (Group 7).

5.1.8 High myopia and eye disease

Papers published from 1997 to 2006 and included in this review show that high myopia is associated with cortical, nuclear and posterior subcapsular cataract although the direction of causality is not clear (Group 1).

Myopia may also increase the risk of POAG, and of developing glaucoma early in life (Group 2).

High myopia does not appear to increase the risk of AMD (Group 4).

Due to conflicting results, it is unclear whether high myopia is associated with an increased risk of amblyopia in children, or with an increased risk of diabetic retinopathy (Group 5). However, this is a complex area of research and more detailed analysis was beyond the scope of this review.

No relevant studies were found in the search period for an association between high myopia and retinitis pigmentosa or trachoma (Group 7).

See also further information in Chapter 6 on the relationship between high myopia and retinal detachment and chorio-retinal degeneration.

5.1.9 Ocular hypertension and eye disease

Papers published from 1997 to 2006 and included in this review show that ocular hypertension (OHT) may lead to glaucoma, with even mild and moderate pressure increasing the risk (Group 2). However, in many cases of glaucoma, OHT may be normal. See Chapter 6 for further information on the relationship between OHT and glaucoma.

Due to conflicting results, it is unclear whether OHT is associated with an increased risk of cataract development (Group 5).

No relevant studies were found in the search period for an association between OHT and amblyopia, diabetic retinopathy, retinitis pigmentosa, AMD or trachoma (Group 7).
5.1.10 Poor living conditions and eye disease

It is difficult to determine the effects of poor living conditions on eye health because of the lack of a standard classification system for socioeconomic status. Studies use a range of different socioeconomic factors (e.g., income, education, occupation, etc) to identify poor living conditions. However, papers published from 1997 to 2006 and included in this review show that poor living conditions may be linked to a higher risk of diabetic retinopathy and glaucoma (Group 2).

Due to conflicting results, it is not clear whether poor living conditions are associated with cataract and AMD (Group 5).

No relevant studies were found in the search period for an association between poor living conditions and amblyopia or retinitis pigmentosa (Group 7). There is a well-established association between poor living conditions and trachoma but no relevant studies were found in this review (indicating that this was already well established by 1996).

In retrospect, ‘poor living conditions’ may not have been the best search term for this topic and a further research of the literature about socioeconomic status and eye disease may yield further relevant information (see Chapter 6).

5.1.11 Diabetes and eye disease

Papers published from 1997 to 2006 and included in this review show that type 1 and type 2 diabetes are significantly linked with all types of cataract but, in the early stages diabetic cataract can be reversed with a change in diet and medication (Group 1).

There is a high rate of diabetes in people with trachoma, but causality is not clear because both diseases are poverty related. Diabetic retinopathy may make people more susceptible to poor visual acuity after trachoma (Group 2).

Diabetes appears not to be a risk factor for AMD (Group 4).

Due to conflicting results, it is not clear whether diabetes is associated with AMD (Group 5).

No relevant studies were found in the search period for an association between diabetes and amblyopia or retinitis pigmentosa (Group 7).

5.1.12 Heredity and eye disease

Papers published from 1997 to 2006 and included in this review show that heredity is the major factor determining cataract development (Group 1).

Development of POAG is strongly linked to heredity factors; secondary OAG less so (Group 1). However, unlike cataracts, where a simple genetic mechanism is involved, glaucoma has different phenotypes (sometimes due to cornea thickness, sometimes due to increased intraocular pressure, etc). This makes it more difficult to associate heredity with particular cases.

Also, heredity strabismus may be linked with amblyopia (Group 2).
No relevant studies were found in the search period for an association between heredity and diabetic retinopathy, AMD, or trachoma (Group 7). However, see Chapter 6 for further information on the contribution of genetic mutations to AMD.

Retinitis pigmentosa is known to be inherited.

This is a very complex area that requires further, more focused literature research to identify the contribution of genetics to eye disease (see Chapter 6).

5.1.13 Hypertension and eye disease

Papers published from 1997 to 2006 and included in this review show that hypertension is a risk factor for retinopathy in people with and without diabetes, and for glaucoma (Group 1).

Due to conflicting results, it is not clear whether hypertension is a risk factor for cataract or AMD.

No relevant studies were found in the search period for an association between hypertension and amblyopia, retinitis pigmentosa or trachoma (Group 7).

5.1.14 Squint and eye disease

Papers published from 1997 to 2006 and included in this review show that strabismus is clearly a cause of amblyopia, although there is debate about different intervention and screening programs (Group 1).

No relevant studies were found in the search period for an association between squint and cataract, diabetic retinopathy, glaucoma, AMD, retinitis pigmentosa or trachoma (Group 7).

5.1.15 Anisometropia and eye disease

Papers published from 1997 to 2006 and included in this review show that anisometropia can lead to amblyopia (Group 1). The condition is associated with the presence of cataract, although causality has not been confirmed (Group 2).

No relevant studies were found in the search period for an association between anisometropia and diabetic retinopathy, glaucoma, AMD, retinitis pigmentosa or trachoma (Group 7).

5.1.16 Cataract and eye disease

Papers published from 1997 to 2006 and included in this review show that congenital cataracts cause abnormal or reduced visual stimulation during the sensitive period of visual development, which can result in amblyopia (Group 1).

Due to conflicting results, it is not clear whether incidence of cataracts or cataract surgery is linked to AMD (Group 5).
No relevant studies were found in the search period for an association between cataract and diabetic retinopathy, glaucoma, retinitis pigmentosa or trachoma (Group 7).

5.1.17 Physical activity and eye disease

Papers published from 1997 to 2006 and included in this review show that physical activity may protect against cataract and exudative AMD (Group 6).

No relevant studies were found in the search period for an association between physical activity and amblyopia, diabetic retinopathy, glaucoma, retinitis pigmentosa or trachoma (Group 7).

Physical activity is also related to UV/sunlight exposure (see Section 5.1.5).

5.1.18 Diet and eye disease

Papers published from 1997 to 2006 and included in this review show that (all Group 6):

- a diet high in fruit and vegetables, especially spinach and kale, has a modest protective effect against cataract
- nutrients such as riboflavin, thiamin, vitamin C and vitamin E may protect against cataract, although further studies are needed
- a low-fat, low-glycaemic diet high in fruit, fish and nuts may protect against the onset of AMD, although again, further studies are needed.

Prospective studies suggested that diet (specifically fatty acids and antioxidants) had neither a beneficial nor a harmful effect on the development of POAG (Group 3).

Glycaemic load does not appear to be related to cataract (Group 4).

No relevant studies were found in the search period for an association between diet and amblyopia or retinitis pigmentosa (Group 7).

More research is needed on the role of diet in eye health.

5.1.19 Nutritional supplements and eye disease

Papers published from 1997 to 2006 and included in this review show that supplements (in the form of antioxidants) do not significantly reduce the risk of glaucoma (Group 3).

Due to conflicting studies, it is not clear whether supplements (in the form of vitamins, antioxidants, lutein, zeaxanthin and zinc) have any beneficial or harmful effect on AMD (Group 5). Similarly, it is not clear whether lutein supplements have any effects on retinitis pigmentosa, although docosa-haxaenoic acid (long chain omega-3 fatty acid; DHA) supplements appear not to be beneficial for this condition (Group 5).

No relevant studies were found in the search period for an association between nutritional supplements and amblyopia, diabetic retinopathy or trachoma (Group 7).
5.1.20 Fatty acids and eye disease

Papers published from 1997 to 2006 and included in this review show that particular fatty acids can increase the risk of age-related nuclear opacities, although it is not clear whether a similar association exists for dietary fats and cataracts (Group 2). Fatty acids may lead to improvements in retinitis pigmentosa, but more studies are needed before this could be recommended as therapy (Group 2).

Due to conflicting results, it is not clear whether omega-3 fatty acids protect against glaucoma or AMD (Group 5).

No relevant studies were found in the search period for an association between fatty acids and amblyopia, diabetic retinopathy or trachoma (Group 7).

5.1.21 Obesity and eye disease

Papers published from 1997 to 2006 and included in this review suggest that:

- a body mass index (BMI) and waist-to-hip ratio within the normal range offers the lowest risk of ARM (Group 1);
  higher or lower than average BMI is a risk factor for visually significant AMD (indicating a J-shaped relationship)
- obesity is associated with an increased risk of cataract (especially posterior subcapsular cataract) (Group 2)
- abdominal obesity is a risk factor for retinopathy in people with or without diabetes, although body mass index (BMI) is not a risk factor for this condition (Group 2)

BMI may not affect the risk of glaucoma, but more research is needed to confirm this finding (Group 4).

No relevant studies were found in the search period for an association between obesity and amblyopia, retinitis pigmentosa or trachoma (Group 7).

5.1.22 Combined diet, supplements and weight, and eye disease

See Chapter 6 for further discussion diet and eye disease.

Overall, the effect of diet on eye health is an extremely complex area of research. Within the scope of this review, it has not been possible to analyse all the dietary factors in detail or fully appraise the quality of individual studies

5.2 Eye injury

Papers published from 1997 to 2006 and included in this review show that the use of eye protection is associated with a marked decrease in eye injuries. Eye injury is a common hazard in certain jobs, especially for males aged between 20 and 34 years. Foreign bodies in the eye are the most common work-related injuries, followed by chemical injuries (Group 1).
Sport is also a cause of many eye injuries, particularly in young men. The largest numbers of injuries are caused by the sports that are the most popular, rather than those that are the most dangerous, although particular caution is required when playing sports involving hard, small balls. Eye injury is less likely to occur in established sports, as these sports usually have compulsory and well-designed eye protection (Group 1).

Another cause of injury is assault. The type of eye injury resulting from an assault depends on the method of assault, but blunt trauma is the most common. Most assaults, particularly chemical assaults, result in serious injury or blindness. Further studies would be required to evaluate the severity and incidence of eye trauma from assault in Australia (Group 1).

Eye injury may also be associated with activities such as walking or running near roads, which may carry a small risk due to the potential for metallic foreign bodies to be projected from the road by passing cars (Group 2).

No relevant studies were found in the search period for an association between eye injury and the home environment, alcohol or radiation (Group 7).

5.3 Refractive error

5.3.1 Age or ageing and refractive error

Papers published from 1997 to 2006 and included in this review show that myopia increases with ageing in people under the age of 30 and over the age of 70 (Group 1). Patterns of myopia development with ageing are very dependent on location (eg in Nepal almost none; in Singapore up to 90%; see Chapter 6).

Hyperopia also appears to increase with ageing until age 70. After this age, it is unclear whether the condition stabilises and then further increases with extreme old age, or whether there is a shift to myopia between 70 and 85 (Group 2). It is also not clear whether age affects the incidence of astigmatism or merely changes the type of astigmatism present (Group 5). Another form of refractive error is presbyopia, which is, by definition, caused by ageing. Hyperopia may predispose individuals to early development of presbyopia (Group 2).

5.3.2 Diabetes and refractive error

Papers published from 1997 to 2006 and included in this review show that diabetes can cause transient hyperopia. This effect is corrected when hyperglycaemia is corrected (Group 2). Diabetes does not appear to be a risk factor for myopia (Group 4). No relevant studies were found in the search period for an association between diabetes and astigmatism or presbyopia.

5.3.3 Heredity and refractive error

Papers published from 1997 to 2006 and included in this review show that high myopia may be linked to genetic factors, but more research is needed. There may also be a genetic predisposition for astigmatism (Group 2). No relevant studies were found in the search period for heredity and hyperopia or presbyopia.
See Chapter 6 for further information on the relationship between heredity and refractive errors.

### 5.3.4 Computer or TV use

Papers published from 1997 to 2006 and included in this review found that extended use of visual display units may cause a tendency toward hyperopia (Group 2). There does not appear to be an association between computer and televisions use and myopia (Group 4). No relevant studies were found in the search period for these risk factors and astigmatism or presbyopia.

### 5.3.5 Reading or near-vision work and refractive error

Due to conflicting results, it is not clear whether myopia is associated with reading or other near vision work (Group 5). No relevant studies were found in the search period for these risk factors and hyperopia, astigmatism or presbyopia.

### 5.3.6 Ocular disease and refractive error

Papers published from 1997 to 2006 and included in this review found that cataract and glaucoma may increase the risk of myopia. Untreated retinopathy of prematurity can also lead to high incidence of myopia in adults (Group 2). Grave’s ophthalmopathy may be associated with greater with-the-rule (horizontal) astigmatism. However, given the low prevalence of Grave’s ophthalmopathy, it is unlikely to be a significant risk factor for astigmatism in the general population (Group 2). Glaucoma appeared to be associated with a decrease in hyperopia (Group 6). No relevant studies were found in the search period for ocular disease or presbyopia.

### 5.3.7 Trauma and refractive error

Papers published from 1997 to 2006 and included in this review show that, although rare, a wound can lead to secondary problems such as lenticular astigmatism. No papers were found in the search period for trauma and myopia, hyperopia or presbyopia.

### 5.3.8 Other risk factors and refractive error

No relevant studies were found in the search period for an association between refractive error and alcohol consumption, eye infections, UV damage, antidepressants, antihistamines or warm climate (Group 7).

See Chapter 6 for further information about the effect of light on development of myopia.

### 5.3.9 Diet and refractive error

Papers published from 1997 to 2006 and included in this review found that the link between a high glycaemic diet and myopia remains a theory, but would be an interesting area for future research.
See further information in Chapter 6 about the relationship between myopia and diet.

No relevant studies were found in the search period for an association between diet and hyperopia, astigmatism or presbyopia (Group 7).

5.3.10 Fatty acids and refractive error

Papers published from 1997 to 2006 and included in this review found no relevant studies for an association between fatty acids and myopia, hyperopia, astigmatism or presbyopia (Group 7).

5.3.11 Obesity and refractive error

Papers published from 1997 to 2006 and included in this review found that obesity may be a risk factor for hyperopia, although more research is required in this area.

No relevant studies were found in the search period for an association between obesity and myopia, astigmatism or presbyopia (Group 7).

5.4 Eye infections

5.4.1 Do infection control measures reduce the incidence of eye infections?

The only papers published from 1997 to 2006 relevant to this question and included in the review were those relating to trachoma. In the case of this disease, there is conflicting evidence as to whether infection control measures such as insecticide sprays, antibiotics, health education and face washing reduce the incidence of trachoma.

Further research is needed to determine whether specific infection control measures reduce the incidence of eye infections.

5.4.2 What impact does contact lens use have on incidence of eye infections?

The only papers published from 1997 to 2006 relevant to this question and included in the review were those showing an increased risk of acanthomoebic keratitis in those using contact lenses (Group 1).

See Chapter 6 for information on the emerging practice of orthokeratology.

5.4.3 Does education on use and misuse of contact lenses affect incidence of eye infections?

The only paper published from 1997 to 2006 relevant to this question and included in the review was a randomised controlled trial (RCT) that showed that education did not significantly increase compliance with correct contact lens use in lens users. No studies looking specifically at incidence of eye infections were found.
Further research is needed to determine whether education on use and misuse of contact lenses reduces the incidence of eye infections.

5.5 Eye tests

The questions asked were:

- Do regular eye tests reduce the incidence of eye disease?
- What is the optimal frequency of eye tests for each age group?
- What are the risks and benefits of different frequencies of eye test?

Papers published from 1997 to 2006 and included in this review revealed two important studies on population-based screening of asymptomatic people:

- a 2006 Cochrane review, which found that community-based screening of asymptomatic older people did not result in improvements in vision
- a cohort study in Melbourne found that eye examinations at five-year intervals only yielded a low number of people (maximum of 0.88%) with asymptomatic vision loss; others had noticed a change in vision, and about one-third of those with vision loss had a family history of eye disease.

Section 5.5.2 describes studies for people with diabetes. No studies were identified that allowed assessment of eye testing in other specific populations identified in the scope of this review (such as different age groups, or Aboriginal and Torres Strait Islander people; see Section 1.1).

Two further Cochrane reviews found that there are no RCTs to show the effectiveness or otherwise of population-based screening for glaucoma, or for amblyopia in childhood. Further research is needed, particularly in relation to eye testing for glaucoma, and for the effectiveness and frequency of screening in childhood for amblyopia (see Chapter 6).

5.5.2 People with diabetes and eye tests

Papers published from 1997 to 2006 and included in this review revealed one systematic review of adequate quality that examined eye testing for people with diabetes. This review concluded that it is effective to screen and treat early diabetic retinopathy; however, only a small portion of screened patients benefit from this intervention. The optimal frequency of screening has not been well studied. Several consensus reviews from overseas recommend different schedules.

Further research is needed to determine the optimal screening program. For example, should high-risk patients be screened more often? Should low-risk patients or those with negative test results be screened less often?