Appendix 4 Summary results tables

Key to tables:

– = level of evidence uncategorised (ie lower than IV)[1]
LPS = large population study
NA = not applicable
RCT = randomised controlled trial

[1] For further information on levels of evidence, see Section 3.4

Summary table 1 — smoking and cataract

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
460	Kelly et al 2005	Systematic review (11 cross-sectional, 9 prospective cohorts, 7 case–control)	Current smokers	Smoking	Nonsmokers or never smokers	NA	I (Adequate)	19 of 27 studies found a positive association between smoking and one or more types of cataract. For nuclear cataract, 14 of 17 studies found a positive association with smoking (7 cross-sectional studies with OR/RR 1.09 to 4.4; 5 cohort studies with OR/RR 1.05 to 2.41; 2 case–control studies with OR/RR 1.68 to 1.99). This association fulfilled 5 of 6 criteria for attribution of causality (Bradford Hill criteria)[a] and the 6th criterion (biological mechanism) was also partially met as it is likely that smoking causes oxidative stress in the lens and it is known that oxidative damage is involved in cataract formation. Risk increases with number of cigarettes smoked. Ex-smokers have a lower risk than current smokers, particularly after prolonged quitting. For posterior subcapsular cataract, of 13 studies, 8 found no association, 3 cohort studies found an association for heavy smokers only and 2 studies found an association for men or ex-smokers only. For cortical cataract, of 12 studies, all found no association.	Applicable to Australian population

a Hill AB (1965). The environment and disease: association or causation. *Proceedings of the Royal Society of Medicine* 58:295–300.

Summary	Group
Smoking is strongly associated with development of nuclear cataract, possibly associated with posterior subcapsular cataract and apparently not associated with cortical cataract. Risk increases with number of cigarettes smoked. Ex-smokers have a lower risk than current smokers, particularly after prolonged quitting.	*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

Summary table 4 — smoking and glaucoma

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
695	Bonovas et al 2004	Systematic review with meta-analysis (4 cross-sectional and 3 case–control)	Current smokers in 3 countries	Smoking	Never smokers and past smokers	NA	III (Adequate)	The results of the meta-analysis suggest that current smokers are at a significantly increased risk of developing primary open-angle glaucoma (POAG). Odds ratios from a fixed-effects model were 1.37 for current smokers (95%CI 1.00 to 1.87; *P* = 0.05). In contrast, past smokers did not have a statistically significant elevated risk of POAG (pooled OR 1.03; 95%CI 0.77 to 1.38, *P* = 0.85). A Cochrane Q test has nonsignificant *P* values indicating that the results were homogeneous, although the low number of studies limited the power of this test.	Two studies that reported an RR of 1.0 were excluded because they did not include any confidence intervals and/or used a different definition of smoking from the other studies
686	Kang et al 2003	Prospective cohort	Nurses' Health Study and Health Professionals Follow Up Study	Smoking	Analysed by current smoking status, cigarettes smoked per day and pack-years of smoking	121,701 (F) 51,529 (M)	II (LPS)	In this large study, neither current smokers nor past smokers had a higher relative risk of POAG than those who had never smoked. Heavier smoking did not increase the risk. Those with more than 10 pack-years of smoking had an approximately 20% reduced risk of POAG compared to those who had never smoked.	One pack-year = 1 pack of cigarettes per day for 1 year

Summary	Group
A systematic review of cross-sectional and case–control studies showed a positive association between current smokers (but not past smokers) and glaucoma. However, a very large prospective cohort study, and two studies excluded from the systematic review, did not show this association and those with more than 10 pack-years of smoking experience had a reduced risk of glaucoma.	*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

Summary table 5 — smoking and age-related macular degeneration

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
714	Tomany et al 2004	Prospective cohort (pooling of 3 separate studies)	Adults aged 43–95 years in Australia, the Netherlands and the United States	Smoking	Past smokers or never smokers	1710 current smokers, 3732 past smokers, 3947 never smokers	I/II (LPS)	Current smoking was associated with an increased incidence of geographic atrophy and late age-related macular degeneration (AMD) (OR relative to nonsmokers 2.83 and 2.35 respectively, for past smokers 2.80 and 1.82). No significant differences were found between past smokers and those who had never smoked, suggesting that current smokers who quit can reduce their risk. A statistically significant relationship was not demonstrated separately for smoking and neovascular AMD.	Geographic atrophy is a form of dry (early) macular degeneration
720	Arnarsson et al 2006	Prospective cohort	Adults over 50 years in Reykjavik Eye Study	Smoking	Past or never smokers	846	II (LPS)	No statistically significant association was found between smoking and risk of developing late AMD — either by current/former/never smokers or by pack-years smoked, which was unexpected as the calculated baseline risk suggested that current smokers were more likely than nonsmokers to develop late AMD. However, the authors found that those who had smoked more than 20 pack-years had an increased mortality rate during the 5-year follow-up period, which may have masked the effect of smoking on both early AMD and late AMD.	One pack-year = 1 pack of cigarettes per day for 1 year
725	Clemons et al 2005	Prospective cohort	Clinic-based study in the US	Smoking > 10 pack-years	Smoking ≤ 10 pack-years	3394	II	Results suggest a relationship between smoking at baseline and the development of advanced AMD among individuals with early or intermediate AMD. Individuals with more pack-years of smoking (> 10 pack-years of smoking compared with ≤ 0 pack-years) had an increased risk of incident neovascular AMD (OR 1.55; 95%CI 1.15 to 2.09) and central geographic atrophy (OR 1.82; 95%CI 1.25 to 2.65).	

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
3410	Khan et al 2006	Case–control	4 United Kingdom counties from 2001 to 2003	Current and past smoking	Nonsmoking	435 cases and 280 controls	III-3	The results were consistent with smoking being a risk factor for AMD but were not statistically significant. There was a strong association between AMD and pack years of cigarette smoking ($P = 0.002$), the odds ratio increasing with the amount smoked; for subjects with more than 40 pack-years of smoking the OR was 2.75 (95%CI 1.22 to 6.20) compared with nonsmokers. Both types of AMD showed a similar relation; smoking more than 40 pack-years of cigarettes was associated with an OR of 3.43 (95%CI 1.28 to 9.20) for geographic atrophy and 2.49 (95%CI 1.06 to 5.82) for choroidal neovascularisation. Stopping smoking was associated with reduced odds of AMD and the risk in those who had not smoked for more than 20 years was comparable to nonsmokers. The risk profile was similar for males and females. Passive smoking exposure was associated with an increased risk of AMD (OR 1.87; 95%CI 1.03 to 3.40) in nonsmokers. Conclusions: The results provide strong support for a causal relation between smoking and AMD. They also show an increased risk for AMD in nonsmokers exposed to passive smoking. Stopping smoking appears to reduce the risk of developing AMD.	Choroidal neovascularisation is a major component of the exudative (late) form of AMD

Summary	Group
In two studies, current smoking was associated with an increased risk of developing AMD compared with past smokers and never smokers. Smoking is also associated with a progression to late AMD among those with early or intermediate AMD. For one study that did not show these effects, deaths from other causes may have masked the effect.	*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

Summary table 8 —age or ageing and cataract

Paper no.	Reference	Type of study	Population/ study information	Risk factor/ indicator	Comparator	*N*	Level (quality)	Results	Other notes
1494	Cedrone et al 1999	Prospective cohort	Random population sample of people aged 45–69 years (already enrolled in a study for cardiovascular risk factors) in 1987, 7-year follow-up	Age	Reference to baseline sample without age-related cataracts Reference to follow-up sample without AR cataracts at baseline adjusted for nonresponse to follow-up	860 people at baseline; 828 of the same group at follow-up, 7 years later	II	Ageing is a 'very important risk factor' for cataract. Sex is not a risk factor. Prevalence of AR cataracts increased after the age of 60 (from 1% before 60 up to 12% in the 65–69-year-old group). Cataract prevalence in baseline sample: 3.7% (95%CI 2.7 to 5.2%). Cumulative incidence referring to baseline sample: 6.5% (95%CI 4.8 to 8.2%). Cumulative incidence referring to follow-up sample: 9% (95%CI 6.7 to 11.3%). Adjusted cumulative incidence of AR cataract: 7.6% (95%CI 5.6 to 9.5%). Although the severity of the AR cataracts among the subjects who developed AR cataracts during the 7 years of the study ($N = 54$) was related to the baseline age, it was not *significantly* related: 25 bilateral cataracts (63.5 ± 3.9 years) and 29 unilateral cataracts (61.8 ± 4.9 years).	Frequency/ rate study

Paper no.	Reference	Type of study	Population/ study information	Risk factor/ indicator	Comparator	*N*	Level (quality)	Results	Other notes
2306	Klein et al 1998	Prospective cohort	Beaver Dam Eye Study (population aged 43–84 years at baseline)	Age	NA	3684 people	II	Age at baseline was the most significant characteristic associated with the incidence of all types of cataracts studied (nuclear, cortical, posterior subcapsular) in people without diabetes. The age effect was significant for all groups (males/females with or without diabetes) ($P \leq 0.03$), apart from the left eyes of diabetic men ($P = 0.2$). Incident cortical cataract increased significantly with age in all groups ($P \leq 0.0.4$) and progression was common in all groups and increased significantly with age. Incident posterior cataract (the least frequent type) was also significantly affected by age in people without diabetes ($P < 0.001$), and progression was associated with age in all groups ($P \leq 0.01$).	Age was only one (minor) focus of the study; other risk factors; results were given for people with and without diabetes; only the latter are included in this results table

Summary	Group
Prevalence of cataract increased with ageing, particularly post-60 years when the prevalence increased from 1% to 12% at 65–69 years.	*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

Summary table 9 — age or ageing and amblyopia

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
1023	Donahue 2006	Retrospective case study (Based on large preschool screeening data)	Preschool children (0–7 years) from mass photoscreening program, with anisometropia of > 0.1 diopter	Age	Children of same age with strabismus (*N* = 562)	792	NA	Prevalence and depth of amblyopia increased with age, although prevalence did not rise significantly after 3 years of age (depth continues to rise). Younger children with anisometropia had a lower prevalence and depth of amblyopia than older children. Prevalence of amblyopia: 2 years: 40% 3 years: 65% 5 years: 76% Moderate depth: 0–1 years: 2% (had amblyopia of moderate depth) 2 years: 17% 6–7 years: 45% Severe depth: 0–3 years: rare 4 years: 9% 5 years: 14% (compared with children with strabismus: stable prevalence and depth with age).	Frequency/rate study. Baseline is children who have anisometropia so may be predisposed to amblyopia. Diagnosis depends on the type of health professional diagnosing. Some issues with sensitivity to tests and standardisation.

Summary	Group
In a case series of children with anisometropia, prevalence increased up to three years of age, and depth of amblyopia also increased with age;.	*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

Summary table 10 — age or ageing and diabetic retinopathy

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
1577	Cohen et al 1998	Retrospective cohort	People > 50 years with type 2 diabetes, and without diabetic retinopathy at baseline; ≥ 4-year follow-up	Ageing	NA	833	III-2	Age was a significant and independent predictor of development of diabetic retinopathy (DR) in the elderly. Both age of onset of diabetes and age were significantly associated ($P = 0.01$ and <0.02, respectively). 10% of people (who had not developed DR after more than 13 years since onset of diabetes) developed DR during the 4-year follow-up. People who developed DR were younger at the age of diabetes onset (48.7 ± 9 years; compared with 53.4 ± 9.9 years for those who did not develop DR); $P = 0.001$. Authors conclude that long complication-free period does not define elderly patients at lower risk of DR.	Frequency/ rate study
1579	Cugati 2006	Prospective cohort study	The Blue Mountains Eye Study (participants aged > 49 years, 6-year follow-up)	Ageing	NA	3509	II (LPS)	No significant ageing-related trend for prevalence of DR seen in either survey. Prevalence of DR was 29.4% (1992–94) and 33.4% (1997–2000). A slightly higher prevalence of mild levels of DR ($P = 0.018$) but lower prevalence of moderate–severe levels of DR ($P = 0.049$) was evident after the 5-year interval. Study cites similar figures, as well as contradictory figures, from other studies.	Frequency/ rate study

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
1580	Dandona et al 1999	Cross-sectional	Representative sample of an urban population in southern India of people with diabetes ≥ 30 years (all had had diabetes for at least 10 years)	Ageing	NA	1399	IV	Used multiple logistic regression to show that the odds of having DR were significantly higher in those ≥ 50 years than in those 30–49 years (OR 7.78; 95%CI 2.92 to 20.73)	

Summary	Group
There are conflicting results as to whether or not age and/or ageing is associated with development of diabetic retinopathy. The best quality study in this group (Blue Mountains Eye Study) showed no statistically significant age-related association. Further research is needed on the relationship between the incidence of diabetic retinopathy with time since the onset of diabetes and an ongoing increase in the disease with ageing in diabetic patients.	*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

Summary table 11 — age or ageing and glaucoma

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
1019	Wensor et al 1998	Cross-sectional	Residential (> 40 years randomly selected across city) and nursing home participants (aged 46–101 years) in Melbourne	Age	NA	Residential: 3271 Nursing: 403	IV	Rate of glaucoma in Melbourne rises significantly with age. Residential participants: overall prevalence rate of primary open-angle glaucoma (POAG) = 1.7% (95%CI 1.21 to 2.21). Overall, prevalence of POAG increased significantly with age, from 0.1% (40–49 year-olds) to 11.9% (90+ years). Lists other studies that support this finding (that glaucoma prevalence increases with age).	Frequency/ rate study
983	Buch et al 2001	Cross-sectional	Copenhagen City Eye Study (Elderly urban Danish population 1986–99)	Age	NA	946 (60–80 years)	IV	Glaucoma accounted for 10% of all bilaterally blind people in the study. Bilateral blindness rose significantly with age ($P = 0.02$) for all causes (age-related macular degeneration was the main cause at 60%); glaucoma was equal second with several other causes (all at 10%). Glaucoma wasn't observed as a cause of blindness in participants younger than 70 years. Despite differences in methods and locations of studies, all population-based studies conducted during the 1990s found that the prevalence of blindness increases with age, with glaucoma the primary cause of blindness in 6–12% of cases.	Frequency/ rate study Glaucoma studied as one of many factors leading to blindness

Summary	Group
Cross-sectional studies (level IV) show that prevalence of glaucoma (POAG) increases with ageing.	*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

Summary table 15 — alcohol and cataract

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
638	Hiratsuka and Li 2001	Review of epidemiological studies (including the prospective cohort studies — the Beaver Dam Eye Study and the Blue Mountains Eye Study)	Residents of Australia and the S	Alcohol	Less alcohol	NA	II (LPS)	The Beaver Dam Eye Study found that a history of heavy drinking was related to more severe nuclear sclerotic, cortical and posterior subcapsular opacities (OR 1.34, 1.38 and 1.57, respectively) (Ritter et al 1993). Munoz et al (1993) confirmed that heavy drinkers are at higher risk of posterior subcapsular opacities compared to nondrinkers (OR 4.6; 95%CI 1.4 to 15.1), and the Blue Mountains Eye Study confirmed the association of heavy drinking with increased risk of nuclear cataract (Cumming and Mitchell 1997).	Heavy drinking defined as an average of ≥ 4 drinks/day (Beaver Dam; Blue Mountains) or ≥ 91 g pure ethanol/week (Munoz et al)

Summary	Group
Drinking increases the risk of nuclear, cortical and posterior subcapsular cataracts. Both the Blue Mountains Eye Study and the Beaver Dam Study found this effect at 'heavy' drinking levels, defined as ≥ 4 drinks/day (equivalent to approximately 280 g ethanol per week). However, a further study found that drinking at lower levels (≥ 91 g pure ethanol per week) increased the risk of posterior subcapsular opacities.	*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

Summary table 17 — alcohol and diabetic retinopathy

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
745	Howard et al 2004	Systematic literature review (2 prospective cohort studies)	Results from 2 prospective cohort studies	Alcohol consumption	Less alcohol	NA	I (Good)	The 2 prospective cohort studies assessed the association between alcohol consumption at baseline and diabetic retinopathy. Moss et al (1994) found no association between alcohol consumption and the incidence or progression of diabetic retinopathy, calculated as odds per ounce (28 g) of alcohol. Young et al (1984) found an increased risk of diabetic retinopathy for those who drank ≥ 10 pints of beer/week or equivalent.	Review of the effect of alcohol use on incidence, management and complications of diabetes
743	Kohner et al 1998	Cross-sectional	The United Kingdom Prospective Diabetes Study	Alcohol consumption	NA	2694	IV	For men, increased alcohol consumption was related to more severe retinopathy ($P = 0.005$). The same effect was not seen for women although this may have been because few women involved in the study were regular or heavy drinkers.	Alcohol consumption classified into none, social, regular and heavy

Summary	Group
More research is needed on the long-term effects of alcohol consumption on the noncardiac complications of diabetes mellitus.	*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

Summary table 18 — alcohol and glaucoma

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
692	Fan et al 2004	Case–control	Adults admitted for early onset primary open-angle glaucoma (POAG) in a hospital in China	Alcohol	Less alcohol	32 (early onset POAG) 96 (controls)	IV	This study found that alcohol consumption had a protective effect against early onset POAG (OR 0.028; 95%CI 0.001 to 0.548). The authors suggest that moderate intake of alcohol dilates blood vessels and promotes outflow, thus reducing intraocular pressure (ocular hypertension is the major cause of progressive damage in glaucoma). The study defined alcohol consumption as consuming an average of at least 100 mL of liquor (equivalent to 38 g of alcohol) daily for not less than 1 year.	This study may not be generalisable to the Australian population
676	Yoshida et al 2003	Cross-sectional	People attending an annual health check-up without previous history of glaucoma	Alcohol	NA	569	IV	Alcohol consumption score was found to have a significant positive correlation with intraocular pressure in men ($P < 0.001$). Alcohol consumption was measured as 'never or seldom', 'several times per month', 'several times per week' and 'every day'.	

Summary	Group
There are conflicting results on the effect that alcohol has on the development of glaucoma and its major risk factor, ocular hypertension.	*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

Summary table 19 — alcohol and age-related macular degeneration

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
720	Arnarsson et al 2006	Prospective cohort	Random sample from Reykjavik Population Census for individuals 50 years or older (Reykjavik Eye Study)	Alcohol	Less alcohol	846	II (LPS)	Current alcohol consumption was found to have a moderately protective effect against the development of drusen compared with people who had never drunk alcohol (OR 0.48, 95%CI 0.28 to 0.82 for < 1 drink per month; 0.34, 95%CI 0.16 to 0.72 for > 1 drink per month). It may also increase the risk of pigmentary abnormalities, although this was not found to be statistically significant (OR 1.37, 95%CI 0.77 to 2.43 for < 1 drink per month; 1.42, 95%CI 0.69 to 2.91 for > 1 drink per month). The combined result suggested an increased incidence of age-related macular degeneration (AMD) (OR 1.65, 95%CI 1.06 to 2.56 for < 1 drink per month; 1.98, 95%CI 1.13 to 3.49 for > 1 drink per month; $P < 0.05$).	
650	Buch et al 2005	Prospective cohort	Volunteers between 60 and 80 years of age in Copenhagen (Copenhagen City Eye Study)	Alcohol	Less alcohol	301	II	Alcohol consumption of more than 250 g/week (25 standard drinks) was among the risk factors for early AMD (OR 2.9; 95%CI 1.0 to 9.2; $P < 0.01$); there was a similar trend for late AMD (OR 2.8; 95%CI 0.8 to 9.9) but this was not statistically significant.	In Australia, 1 standard drink = 10 g alcohol
736	Klein et al 2002	Prospective cohort	Beaver Dam Eye Study	Alcohol	Less alcohol	2764	II (LPS)	Total alcohol consumption at baseline was not associated with the incidence of early or late AMD or progression of AMD. Those who drank 78 g or more of alcohol as beer per week at baseline had an increased risk of developing large (greater than 250 μm in diameter) drusen compared with those who did not drink any beer (RR 1.84; 95%CI 1.07 to 3.14). In contrast, consumption of beer in the past year was not associated with the incidence of pigmentary abnormalities or exudative macular degeneration. Men who drank 56 g or more of	

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
								alcohol from liquor per week had an increased risk of developing exudative macular degeneration compared to men who did not drink liquor (RR 6.09; 95%CI 1.50 to 21.80). The same relationship was not found for women. Heavy drinking (defined as 4 or more drinks per day) was associated with a statistically significant increase in incidence of exudative AMD abnormalities, for both former heavy drinkers (RR 2.55 95%CI 1.03 to 6.34) and in current heavy drinkers at baseline (RR 6.51, 95%CI 1.41 to 30.21). This effect was maintained for both men and women in a logistic regression model in relation to smoking. There was a trend towards a reduction in the incidence of early AMD with wine consumption (< 23 g/week and ≥ 23 g/week) but the result was not statistically significant.	
761	Moss et al 1998	Prospective cohort	Beaver Dam Eye Study	Alcohol	Less alcohol	3684	II (LPS)	Men drinking 78 g/week or more of alcohol from beer had a higher incidence of early AMD than those who did not drink beer, although this trend was not statistically significant ($P = 0.08$). The incidence of soft indistinct drusen, increased drusen area and confluent drusen were associated with beer drinking in men.	
755	Cho et al 2000	Prospective cohort	Nurses' Health Study and Health Professionals Follow Up Study	Alcohol	Less alcohol	62252	II (LPS)	Compared with nondrinkers, women who drank 30 g/day or more of alcohol had an increased risk of AMD (RR 1.54; 95%CI 0.99 to 2.39, after adjustment for smoking). The same trend, although weaker, was found for men. When the two types of AMD were considered separately, the association was found for both early and dry AMD but not wet AMD. When alcohol types were considered separately beer consumption was not found to be associated with AMD. Drinking more than 2 wines per day was associated with AMD in women (RR	

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
								2.07; 95%CI 1.20 to 3.58) but not in men (RR 1.7; 95%CI 0.42 to 4.46).	
762	Obisesan et al 1998	Prospective cohort	National Health Nutrition and Examination Survey (NHANES-1)	Alcohol	Less alcohol	3072	II	A statistically significant and negative association was found between drinking wine and AMD after adjusting for the effect of age, gender, income, history of congestive heart failure and hypertension (OR 0.81; 95%CI 0.67 to 0.99).	

Summary	Group
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 that drinking more than about 3 drinks per day, particularly of wine or spirits is associated with development of AMD.	*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

Summary table 22 — eye infections and cataract

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
1437	Madhaven 1999	Cross-sectional	People with conjunctivitis in Chennai, India between 1990 and 1998 had eye swabs taken and analysed	Eye infections	NA	1061	IV	Rubella virus was found in 86 lens aspirates from eyes with congenital cataract (8.1%).	
1441	Vutova et al 2002	Prospective cohort	Infants and children with eye manifestations of congenital toxoplasmosis	Eye infections	NA	38	II (small study)	Cataract was found in 16% of children with congenital toxoplasmosis.	

Summary	Group
Eye infections (conjunctivitis and toxoplasmosis) appear to be linked to cataract.	*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

Summary table 23 — eye infections and amblyopia

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
1466	Kunimoto et al 1998	Case series	Children up to 16 years being treated for microbial keratitis in Hyderabad India between 1991 and 1995	Eye infections	NA	107	NA	70.3% of the children aged under 10 years (37/107) had poor visual acuity outcomes, linked to amblyopia.	Amblyopia is barely mentioned as a measured outcome
1465	Chong et al 2004	Case series	Children under 16 years diagnosed with herpes simplex virus (HSV) keratitis in 1 institution in Texas, USA	Eye infections	NA	23	NA	Amblyopia occurred in 3 children (13%).	Reviewed clinical records of patients presenting with HSV keratitis and followed the results of their treatments

Summary	Group
Amblyopia appears to occur in some cases of eye infection.	*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

Summary table 24 — eye infections and diabetic retinopathy

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
1469	Dev 1999	Case series	Diabetics with endophthalmitis treated between 1992 and 1997 in Wisconsin	Eye infections	NA	11 people (12 eyes)	–	The 6 cases with no retinopathy before endophthalmitis did not go on to develop retinopathy. Of the 6 eyes, cases with pre-existing nonproliferative retinopathy, 4 showed evidence of progression within 6 months of the infection.	

Summary	Group
Eye infection appears to be associated with the development of retinopathy in people with diabetes.	*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

Summary table 25 — eye infections and glaucoma

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
1457	Thean et al 2001	Case series	People with herpes zoster ophthalmicus (HZO) and secondary uveitis seen in Victoria over a 10-year period	Eye infections	NA	34	–	56% of patients developed secondary glaucoma. 15% of all patients required surgical intervention to treat glaucoma.	
1444	De Schryver et al 2006	Case series	People from a clinic in Paris, France, referred between 2001 and 2003 for chronic uveitis associated with glaucoma; all testing negative for human immunodeficiency virus (HIV)	Eye infections	NA	5	–	All patients responded to treatment for cytomegalovirus (CMV) infection, with ocular inflammation and glaucoma being controlled. Two patients required surgery as well. After therapy was stopped, 3 cases relapsed and required further CMV therapy. Polymerase chain reaction (PCR) tests of eye tissue were positive for CMV and negative for other viruses. PCR testing was positive in the patients who relapsed once therapy stopped.	
1448	Egbert et al 2005	Case series	People at a clinic in Ghana, Africa undergoing surgery for glaucoma or cataracts had skin snip biopsies to test for nematode infection	Eye infections in glaucoma patients	Eye infections in cataract patients	286	–	Nematode infection was present in 10.6% of glaucoma patients, compared to 2.6% in cataract patients. Once adjusted for age, region and sex, subjects with glaucoma were over 3 times more likely to test positive for nematode infection than people with cataract (OR 3.50; 95%CI 1.10 to 11.18).	
1441	Vutova et al 2002	Prospective cohort	Infants and children with eye manifestations of congenital toxoplasmosis	Eye infections	NA	38	II (small study)	Glaucoma was found in 16% of children with congenital toxoplasmosis.	

Summary	Group
A range of infectious agents (eg herpes zoster, cytomegalovirus and nematodes) appear to be associated with glaucoma.	*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

Summary table 26 — eye infections and macular degeneration

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
1130	Kalayoglu et al 2003	Case–control	People with age-related macular degeneration (AMD) who had choroidal neovascular (CNV) membrane removed	Eye infections in AMD eyes	Non-AMD eyes	9 AMD, 9 non-AMD	III-3 (very small study)	*Chlamydia pneumoniae* was isolated in 4 out of the 9 AMD samples. None of the non-AMD samples had *C. pneumoniae* present. This paper refers to 2 other studies that have shown some association between *C. pneumoniae* infection and AMD: Ishida O et al (2003). Is *Chlamydia pneumonia* infection a risk factor for age related macular degeneration? *British Journal of Ophthalmology* 87:523–524. Kalayoglu MV et al (2003). Serological association between *Chlamydia pneumonia* infection and age related macular degeneration. *Archives of Ophthalmology* 121:478–482.	Performed PCR and immunohistology on samples from AMD and non-AMD eyes

Summary	Group
There may be a link between infection with *Chlamydia pneumoniae* and macular degeneration.	*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

Summary table 36 — UV and cataract

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
932	McCarty and Taylor 2002	General review of 22 epidemiological studies (mostly cross–sectional but also including prospective cohort and case–control)	Included studies from Australia and the US	UV	NA	NA	II	Most of the 22 epidemiological studies reviewed met most of the criteria for causality, supporting an association between exposure to medium-wave ultraviolet light (UVB) and the development of cortical cataract. There was also limited evidence to suggest a link between UVB and posterior subcapsular cataract.	

Summary	Group
A large number of epidemiological studies support an association between UVB and the development of cortical cataract.	*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

Summary table 40 — UV and age-related macular degeneration

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
901	Cruickshanket al 2001	Prospective cohort study	Beaver Dam Eye Study (5-year follow-up)	Sun exposure	Various variables	3684	II (LPS)	Leisure time spent outdoors between the ages of 13 and 19, and between 30 and 39, was significantly associated with the risk of early age-related macular degeneration (AMD) (OR 2.09; 95%CI 1.19 to 3.65). Job time spent outdoors and leisure time spent outdoors in winter did not increase the risk of AMD. There was a slight but nonsignificant protective effect associated with the use of hats and sunglasses (OR 0.72; 95CI% 0.5 to 1.03). There were no associations between estimated ambient UVB exposure and the incidence of early AMD. There was no association between skin sensitivity (never burns, or tans, versus burns) and incidence of AMD. This is apparently contradictory to an earlier report from the Blue Mountain Eye Study in Australia (Mitchell et al 1998) that showed an association between skin sensitivity and early AMD. People with blonde or red hair had an increased risk of early AMD (RR 1.31, 95%CI 0.96 to 1.78)	
908	Tomany et al 2004	Prospective cohort study	Beaver Dam Eye Study (10-year follow-up)	Sun exposure	Various variables	2764	II (LPS)	Participants exposed to summer sun for more than 5 hours per day during their teens, in their 30s, and at baseline examination were at a higher risk of developing increased pigmentation (RR 3.17; 95%CI 1.24 to 8.11; $P = 0.01$) early AMD (RR 2.14; 95%CI 0.99 to 4.61; $P = 0.05$). No relationships were found between UVB exposure or winter leisure time spent outdoor and the 10-year incidence and progression of AMD. For those who reported a high amount of sun exposure (teens and 30s), hat and sunglass use for at least half the time had a protective effect against the 10-year incidence of soft indistinct drusen (RR 0.55: 95%CI 0.33 to 0.90; $P = 0.05$) and retinal pigment epithelial depigmentation (RR 0.51 95%CI 0.29 to 0.91; $P = 0.02$). Those who had experienced more than 10	

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
								incidents of severe sunburn during their youth were more likely to develop ≥ 250 µm drusen by the 10-year examination (RR 2.52; 95%CI 1.29 to 1.71; *P* = 0.01).	
3397	Mitchell et al 1998	Cross-sectional	Blue Mountains Eye Study	NA		3654	IV (LPS)	Blue iris colour was significantly associated with an increased risk of both late AMD (OR 1.69) and early AMD (1.45). Abnormal skin sensitivity, either high (OR 2.54) or low (OR 2.18), was associated with increased risk of AMD.	

Summary	Group
Despite experimental evidence that the retina is susceptible to UV damage there has been no evidence that UV exposure per se is a risk for AMD. However, sunlight exposure in the teenage years and 30s is associated with increased risk of AMD-related pathologies (drusen and pigmentation) and early AMD. Other outdoor exposures did not increase risks (eg working outdoors). 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).	*Group 1* — Clear association/causality *Group 2* — Possible association/causality (more research needed) **(sunlight generally — not UV)** *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

Summary table 41 — UV and pterygium

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	N	Level (quality)	Results	Other notes
08-01	Vojnikovic et al 2007	Case-control	Mean age 65-80 years	Exposure to UV light (chronic)	Urban population compared to rural agriculturalists and fisherman. (Croatia)	480 rural, 61 urban	III-3	Prevalence of pterygium: Villagers 23% (16% in males, 7% in females), urban people 0%.	Applicable to specific population
08-02	Lu et al 2007	Prospective cohort study	Tibetan people ≥40 years	UV exposure and other factors	None	2632	II (LPS)	Prevalence of pterygium: 14.49% (95% CI 13.03 to 15.95). Risk factors: Increasing age for persons aged 70-79 years, compared with those aged 40-49 years (OR 2.0; 95% CI 1.4 to 2.8); Female gender (OR 1.6; 95% CI 1.2 to 2.0); Dry eye symptoms (OR 1.3; 95% CI 1.0 to 1.7) Seldom use of sunglasses/ crystal spectacles (OR 4.6; 95% CI 1.9 to 11.3) Seldom use of hats (OR 3.6: 95% CI 2.4 to 5.4) Lower education level (<3 years) (OR 1.6; 95% CI 1.1 to 2.4) Low socioeconomic status (OR 1.9; 95% CI 1.5 to 2.4).	Applicable to specific population

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	N	Level (quality)	Results	Other notes
08-03	Paula et al 2006	Cross-sectional study	Four Brazilian Indigenous populations	UV exposure and other factors on pterygia and cataract prevalence	Ethnic and social groups (Group 1: Fisherman, Group 2: Hunters)	624	IV	Prevalence of pterygium: Group 1 (36.6%) versus Group 2 (5.0%), significantly different (P<0.0001) Gender was not associated with pterygium (P=0.1326) and pterygium did not increase with age (P=0.8079) in either group.	Applicable to specific population
08-04	Mathur et al 2005	Cross-sectional study	Indian salt workers	UV exposure	Occupational groups (brine workers, dry salt workers, others)	865 salt workers, 304 controls	IV	Prevalence of pterygium: Brine workers (21.0%), dry salt workers (9.1%) (P<0.000001), non-salt-worker controls (9.4%) (P=0.00007). Risk factors: Exposure during 3rd decade of life (P=0.046) compared to exposure in second decade of life.	Applicable to specific population
08-05	Al-Bdour et al 2004	Case control study	Adult Jordanians presenting with pterygium	Environmental factors including sunlight exposure	Workplace location, past and current sun exposure	96 cases, 192 controls	III-3	Significant increase in risk of pterygium in outdoor workers compared to indoor workers (OR 5.47), current sun exposure (OR 3.54) and previous sun exposure (4.52).	Applicable to specific population
08-06	Saw et al 2000	Case-control study	Singaporean adult patients at the Singapore National Eye Centre	Current sunlight exposure	Past exposure to sunlight (5 years ago, 10 years ago)	61 cases, 125 controls	III-3	Risk of developing pterygia was higher for participants who had spent time in the sun five years ago (OR 1.27, 95% CI 1.06 to 1.54) and ten years ago (OR 1.31, 95% CI 1.09 to 1.57) compared to current exposure.	

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	N	Level (quality)	Results	Other notes
08-07	McCarty et al 2000	Prospective cohort study	Nine randomly selected population clusters from Melbourne; 14 nursing homes within 5km radius of the 9 Melbourne clusters; 4 randomly selected rural clusters. Age range 40 to 101 years.	Lifetime exposure to sunlight	Location, past and current sun exposure	5147 (2850 female)	II	Prevalence of pterygium: Melbourne residents (1.2%) Nursing home residents (1.7%) Rural residents (6.7%) Overall weighted population prevalence (2.83%, 95% CI 2.35 to 3.31%). Risk factors: Age (OR 1.23, 95% CI 1.06 to 1.44) Male sex (OR 2.02, 95% CI 1.35 to 3.03) Rural residence (OR 5.28, 95% CI 3.56 to 7.84) Lifetime ocular sun exposure (OR 1.63, 95% CI 1.18 to 2. 25). Attributable risk of sunlight and pterygium: 43.6% (95% CI 42.7 to 44.6).	Australian population
08-08	Threlfall et al 1999	Case-control study	Cases had surgical removal of pterygium, control subjects had ENT procedure	Exposure to sunlight	Presence of pterygium versus no pterygium.	150 cases, 135 controls	III-3	Risk factors for pterygium: Sun exposure (highest exposure quartile) (OR 4.0, 95% CI 1.6 to 10.9) Estimated daily ocular solar radiation dose (highest exposure quartile) (OR 6.8, 95% CI 2.6 to 19.7)	Australian population
08-9	Tang et al 1999	Cross-sectional study	Postmen in Central Taiwan	Outdoor work (exposure to sunlight)	Indoor work (low occupational sunlight exposure)	394 postal workers (248 postmen, 146 office workers)	IV	Prevalence of pterygium: Postmen (7.3%.) Significantly different to office workers (P<0.05). When cumulative sun exposure increased by one unit (one year x hour/day) pterygium risk increased by 0.8%.	Applicable to specific population

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	N	Level (quality)	Results	Other notes
08-10	Khoo et al 1998	Case control study	Singaporean patients at Singapore National Eye Centre with pterygium	Sunlight exposure (outdoor workers)	Indoor work (low sunlight exposure)	61 cases, 125 controls	III-3	Risk of pterygium: OR 7.0 (95% CI 3.2 to 15.3); AOR 4.2 (95% CI 1.7 to 10.1).	
08-11	Sekelj et al 2007	Prospective cohort study	Patients with primary and recurrent whose pterygia were surgically removed	Exposure to sunlight	Patients with primary and recurrent pterygium not exposed to sun after surgical removal of pterygia	19 cases (38 eyes); 10 controls (20 eyes)	II	Pterygia recurrence rate: Exposed group (27%) Unexposed group (10%)	

Summary	Group
Current and past exposure to UV light increases the population prevalence of pterygia and the risk of developing pterygia. Exposure to UV light also increases re-development of pterygia after surgical removal.	**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**

Summary table 42 — UV and ocular surface neoplasms

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	N	Level (quality)	Results	Other notes
08-12	Kiire and Dhillon 2006	Systematic review (2 retrospective cohort studies; 2 case-control studies)	Patients with conjunctival tumours, Africa, US, Thailand	Sun exposure			Adequate Level III-2/III-3 studies	One study showed that the incidence of squamous cell carcinoma (SCC) of the eye fell by 49% for every 10-degree increase in latitude from >12 cases/million/year in Uganda to 0.2 cases/million/year in the UK. A retrospective cohort study showed an SCC of the conjunctiva incidence rate of 0.03/100000, with 5-fold higher rates among males and whites. Positive association between UVB exposure and SCC rates (beta = 2.25; r = 0.58). In one case-control study, conjunctival cancer was associated with HIV infection (OR 10.1, 95% CI, 5.2 to 19.4; P<0.001); risk increased with time spent in cultivation/direct sunlight (P= 0.05); risk decreased with decreasing age at leaving home (P=0.05). 1 small study did not show a link between sunlight exposure and risk of conjunctival intraepithelial neoplasia.	
08-13	Gallagher and Lee 2006	General review of adverse effects of UV (brief description of 7 studies from 1988 to 2003)	Patients with conjunctival tumours,	UV/sunlight			NA	Authors concluded that there is evidence for a probable relationship between with solar UVR and squamous intraepithelial neoplasm of the conjunctiva or cornea, although one or more co-factors may be necessary (eg smoking, HPV). This tumour more common in people with fair skin.	

08-14	Basti and Mascai 2003	General review of ocular surface squamous neoplasia (OSSN) (brief description of 10 studies from 1950–1997)					NA	UVB is a major risk factor for OSSN. Other risk factors included pale skin, pale iris, time spent outdoors and distance from equator. Causal evidence comes from mutation studies that show an increased prevalence of *P53* mutations.	
08-15	Ateenyi-Agaba et al 2004	Case-control study	Patients with squamous cell carcinoma, controls with benign conjunctival lesions in Uganda, Africa	Exposure to sunlight		21 cases, 22 controls	III-3	P53 mutations were found to be associated with SCC cases, particularly CC-->TT transitions, which is the signature mutation for solar UV damage. The prevalence of CC-->TT transitions was the highest reported in any cancer type and matched that of skin cancers in subjects an inherited disease causing hypersensitivity to UV damage. These results indicate at the molecular level a causal role of solar UV rays in the aetiology of SCC of the eye.	Paper in German

Summary	Group
Some epidemiology studies indicate that ocular surface neoplasms may be associated with exposure to UVB/sunlight. A causal effect of UV is substantiated by molecular evidence of UV-induced mutations. Other risk factors include fair skin, light coloured iris, HIV infection, HPV infection and smoking.	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

Summary table 43 — injuries/accidents and cataract

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
778	Brouzas et al 2003	Case series	People admitted to a hospital in Athens, Greece, with elastic cord-induced ocular injuries over a 67-month period from October 1996	Injuries and accidents	NA	28	–	Cataract developed in 7.1% of patients (2/28). The most common injury was hyphaema (71.4%).	Frequency/ rate study
771	Ariturk 1999	Case series	Children (under 20 years) admitted with ocular trauma to a hospital in Samsun, Turkey between November 1983 and October 1996 Reviewed history of patients with ocular trauma to see what kind of trauma, treatment, follow up, etc	Injuries and accidents	NA	138	–	Blunt trauma accounted for 17.4% of injuries and perforating trauma 82.6%. Cataract was one of the most frequent complications of blunt trauma (12.5% of cases) and occurred after 24.6% of perforating trauma cases.	Frequency/ rate study
824	Wong et al 2002	Case–control Appear to have used both cross-sectional and case–control analysis	People with a history of ocular trauma taken from the Beaver Dam Study	Injuries and accidents	People with no history of ocular trauma taken from the same group	4926	III-3/IV	People with a history of ocular trauma were more likely to have cortical (OR 1.5; 95%CI 1.0 to 2.2) and posterior subcapsular (OR 1.7; 95%CI 1.0 to 3.1) cataracts, compared to people without a history of trauma. These associations were stronger for people with previous trauma caused by a blunt object (OR 3.3; 95%CI 1.6 to 6.9) for cortical cataract, and (OR 4.1; 95%CI 1.5 to 10.8) for posterior subcapsular cataracts.	Frequency/ rate study

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
834	Lithander 1999	Cross-sectional	Random selection of schoolchildren given visual screening from grades 1–6 in Oman	Injuries and accidents	NA	6292	IV	12 out of 6292 children had poor visual acuity due to trauma; of those 12 children, 4 developed cataract.	Frequency/ rate study

Summary	Group
Cataract can be caused by ocular trauma.	*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

Summary table 44 — injuries and accidents and amblyopia

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
834	Lithander et al 1999	Cross-sectional	Nationwide survey of primary schools in Oman	Injury	NA	6292	IV	The study detected several children with untreated traumatic cataract from injuries incurred several years before the study, allowing amblyopia to develop.	
803	Jeng et al 2001	Case report	Cases of penetrating ocular injury due to toy ninja stars	Injury	NA	2	–	A 3-year-old boy suffered a large corneal laceration and traumatic cataract after trauma from a toy ninja star. After a penetrating keratoplasty he required ongoing treatment for amblyopia. The authors note that many toys can cause injury but sharp pointed projectiles are particularly dangerous.	
829	Capone 2003	Case series	Infants with amblyogenic vitreous and/or subinternal limiting membrane haemorrhage from shaken baby syndrome managed by lens-sparing vitrectomy	Injury	NA	11 eyes	–	Dense vitreous haemorrhage may cause amblyopia, either from visual deprivation or anisometropia. Vitreous haemorrhage may require more time to resolve spontaneously in infants, and because infants can develop amblyopia within days or weeks the authors recommend lens-sparing vitrectomy.	

Summary	Group
Ocular injuries and accidents can cause young children to develop amblyopia, either from visual deprivation or anisometropia. These injuries can have a wide range of causes, including dangerous toys and shaken baby syndrome.	*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

Summary table 46 — injuries and accidents and glaucoma

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
854	Girkin et al 2005	Retrospective cohort	Data from the United States Eye Injury Registry on people who had suffered ocular contusion	Injury	Various	6021	III-2	The risk of developing posttraumatic glaucoma following contusion was associated with increased age (OR 1.02; 95%CI 1.02 to 1.03), poor visual acuity (OR 1.92; 95%CI 1.19 to 3.10), iris injury (OR 1.60; 95%CI 1.05 to 2.44), lens injury (OR 1.86; 95%CI 1.11 to 3.11), hyphema (OR 2.23; 95%CI 1.40 to 3.54) and angle recession (OR 1.71; 95%CI 1.00 to 2.90).	
782	Cavallini et al 2003	Case series	People presenting to a clinic in Italy with injuries from wine bottle corks	Injury	NA	13	–	All people had closed globe injuries, with anterior chamber hyphema the most frequent injury. Post-traumatic glaucoma was one of the resulting complications.	
848	Duiguid and Leaver 2000	Case report	Person with giant retinal tears following deliberate gouging in a rugby game	Injury	NA	1	–	A person presented with giant retinal tears which he had suffered as a result of deliberate gouging during a game of rugby. After surgical treatment he developed complications including glaucoma.	
805	Kenney and Fanciullo 2005	Case report	Person with blunt ocular trauma resulting from airbag injury	Injury	NA	1	–	A person who sustained blunt ocular trauma from the deployment of an airbag during a motor vehicle accident later developed complications, including traumatic/inflammatory glaucoma.	
847	De Leon-Ortega and Girkin 2002	Background	NA	NA	NA	NA	NA	Glaucoma is a possible complication of ocular trauma. Traumatic glaucoma is a multifactorial condition and can result from a variety of mechanisms including closed globe trauma (early or late onset) and open globe trauma (with or without an intraocular foreign body).	

Summary	Group
Glaucoma can occur as a complication of ocular trauma by a number of mechanisms. The risk of developing post-traumatic glaucoma after contusion is associated with increased age, poor visual acuity, iris injury, lens injury, hyphema and angle recession. The original trauma may be the result of any type of injury, including sporting injuries and automobile injuries.	*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

Summary table 47 — injuries and accidents and macular degeneration

Paper no.	Reference	Type of study	Population/ study information	Risk factor	Comparator	*N*	Level (quality)	Results	Other notes
884	Klein et al 2005	Prospective cohort	Beaver Dam Eye Study (5-year follow-up)	X-ray exposure	Less exposure	3 684	II	No evidence to link macular degeneration to X-ray exposure	
888	Shiuey and Lucarelli, 1998	Case study with historic control group	Patients with traumatic hyphema treated in a Massachusetts hospital between 1991 and 1995 had visual acuity measured and followed up	Traumatic hyphema treated in emergency dept	Historic control group treated as inpatients for the same condition between 1986–89	154	–	Only one patient developed macular degeneration as a result of traumatic hyphema	Study not set up to study AMD

Summary	Group
Macular degeneration does not appear to be linked to ocular injury.	*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