

Appendix 1: Methods for estimating disability burden

In this section we describe our methods for calculating disability for the large number of diseases and injuries and their sequelae for which models were developed, including all those that make significant contributions to the total non-fatal burden. While this list is extensive, it is not exhaustive, and explicit models were not developed for many conditions. Table A1.1 lists the full names of many of the data sources underlying our models and our abbreviations of these names, which we use in this section for ease of reference.

Table A1.1: List of full and abbreviated names of commonly used data sources

Abbreviated name	Full name
AusDiab	The Australian Diabetes, Obesity and Lifestyle Study, 1999–2000 (Dunstan et al. 2001)
Australian dialysis and transplant data	2002 Australian and New Zealand Dialysis and Transplant Registry (McDonald & Russ 2002). The interpretation of this data is the responsibility of the authors of this report and should not be seen as the interpretation of the Australian and New Zealand Dialysis and Transplant Registry.
Australian disability survey	Survey of Disability, Ageing and Carers (1993, 1998 or 2003) (ABS 1993, 1998b, 2003b)
Australian general practitioner data	2000–01 and 2002–03 Bettering the Evaluation and Care of Health (AIHW: Britt et al. 2001)
Australian hospital data	2002–03 National hospital morbidity database (AIHW 2003a)
Australian mortality data	2003 Cause of Death dataset (ABS 2005)
Australian notification data	National Notifiable Infectious Disease Surveillance System (CDA, 2003) except for HIV/AIDS which is from the National Centre for HIV Epidemiology and Research (National Centre in HIV Epidemiology and Clinical Research, 2003)
Australian perinatal data	2002 Australia's mothers and babies and various state and territory perinatal data collections (AIHW: Laws & Sullivan 2004; Queensland Health 2004; Riley & King 2003).
Disability weight regression model	Regression model of Dutch disability weights which requires inputs of health state description based on the six domains of the EQ5D+ (p. 158 of AIHW: Mathers et al. 1999)
DisMod	DisMod version II (Barendregt et al. 2003)
GBD study	Global burden of disease and risk factors, 2000 (Lopez et al. 2006)
Low prevalence study	1997–98 Low Prevalence (Psychotic) Disorders Study (Jablensky et al. 1999)
National Health Survey	2001 National Health Survey (unless otherwise specified as the 1995 National Health Survey) (ABS 1995, 2001c)
National mental health survey	1997 National Survey of Mental Health and Wellbeing (ABS 1997)
National Trachoma Survey	1980 National Trachoma and Eye Health Program (Royal Australian College of Ophthalmologists 1980)
Previous Australian burden study	Australian Burden of Disease and Injury Study, 1996 (AIHW: Mathers et al. 1999)
Victorian birth defect data	2001–02 Victorian Birth Defects Register (Riley & Halliday 2004)
Victorian linked hospital dataset	Analyses of Victorian hospital data 1996–2002 & 2001–02 from the 2001 Victorian Burden of Disease and injury study (DHS, 2005)
Women's health Australia	Australian longitudinal study on women's health (Lee et al. 2005)

1A Infectious and parasitic diseases

Tuberculosis

We estimate the incidence of tuberculosis using Australian notification data on new cases of tuberculosis. We assume that the average duration for tuberculosis is 8 months, reflecting 6 months for the shortest treatment cycle available and another 2 months of symptoms before treatment.

Sexually transmitted diseases (excluding HIV/AIDS)

We base our incidence estimates for syphilis, chlamydia and gonorrhoea on Australian notification data. Following expert advice, we assume that annual notifications for syphilis and gonorrhoea represent all incident cases. We model syphilis using a staged approach applying proportionate distributions for primary, secondary, and tertiary syphilis from the GBD study. We adjust our estimates for chlamydia to account for under-reporting due to asymptomatic infections and the reluctance of some patients to consult general practitioners about sexually transmitted diseases. We base our incidence estimates of pelvic inflammatory disease, a complication of both chlamydia and gonorrhoea in women, on Australian hospital data. Following expert advice we adjust these estimates to account for under-identification. Common sequelae of pelvic inflammatory disease include ectopic pregnancy, chronic pelvic pain, infertility and tubo-ovarian abscess. We base our rates of complications following pelvic inflammatory disease on GBD assumptions. We adjust our incident estimates of infertility resulting from pelvic inflammatory disease for women who do not wish to have a child and therefore do not experience disability. In the absence of Australian data on 'child wish', we make this adjustment using findings from a recent German study (Stobel-Richter et al. 2005). The GBD reports urethral stricture and epididymitis as complications following chlamydial and gonorrhoeal urethritis in men. These complications were thought by experts to be rare, and so have not been included in the Australian estimates. We model disability weights and durations for syphilis, chlamydia and gonorrhoea and their sequelae using the assumptions of the GBD study.

HIV/AIDS

We model HIV as a progressive condition with four stages: (1) asymptomatic HIV; (2) symptomatic HIV; (3) AIDS prior to terminal phase; and (4) terminal AIDS. We assume that the annual number of new HIV diagnoses from Australian notification data represent all incident cases of HIV. We use the Dutch disability weights for each of the stages (stage 1 – 0.2, stage 2 – 0.31, stage 3 – 0.56 and stage 4 – 0.95) and adjust the weight for stage 1 to account for the estimated proportion of undiagnosed asymptomatic HIV cases to whom we assign a disability weight of 0 (Aalen et al. 1997). We calculate the mean durations for stages 1 to 3 using Weibull regressions of published data accounting for background mortality (Kaldor & McDonald 2003; Mocroft et al. 1997; Porter et al. 2003). This gives average durations of 30 years for the combined stages 1 and 2 and 5.5 years for stage 3. We adjust our duration estimates for stage 1 and 2 based on the assumption that an equal amount of time is

spent in each stage based on work by Aalen and colleagues (1997). In the absence of new evidence, we assume that stage 4 lasts an average 0.5 of a year.

Diarrhoeal diseases

Diarrhoeal diseases include a number of notifiable diseases as well as non-notifiable diseases. Given that notifications are generally considered a gross underestimate of the incidence for notifiable diarrhoeal diseases, and that there is often even less reliable information on the incidence of non-notifiable diarrhoeal diseases, we do not model diarrhoeal diseases using notification data or by specific cause. Instead, we derive the incidence of diarrhoea not requiring hospitalisation using annualised self-reported data from the 2001–02 National Gastroenteritis Survey (Hall & OzFoodNet Working Group 2004). We base our duration of 2 days from the findings of this survey and use age-specific weights for uncomplicated diarrhoea (average weight of 0.093) from the GBD study. We use Australian hospital data to estimate the incidence of diarrhoea cases requiring hospitalisation. We use the age-specific GBD weight for diarrhoea (0.093) since the Dutch weight is implausible. We assume 2 weeks duration for complicated diarrhoea and derive an average weight (0.42) based on 1 week of disability equivalent to the regression model of health state (323311) and 1 week of disability for uncomplicated diarrhoea.

Childhood immunisable diseases

We do not model poliomyelitis and diphtheria for 2003. This is because there were no notifications of poliomyelitis from 1993 to 2003 and only one notification of diphtheria in 2001.

Pertussis

We estimate the incidence of pertussis using Australian notification data averaged over 2000–2003, an epidemic cycle. We adjust our incidence estimates for under-reporting based on the literature (Andrews et al. 1997; Torvaldsen et al. 2002). We apply the age-specific GBD disability weights for untreated cases for pertussis (0–4 years: 0.178; 5–14 years: 0.166; 15 years or over: 0.156), since the weight for treated cases is implausible, along with the GBD duration of 1 month. Following expert advice we estimate the incidence of intellectual disability attributable to pertussis as the proportion of intellectual disability cases from the total episodes of infection for 0–4 year olds in the GBD study (that is, 0.3% of pertussis cases). We derive a disability weight (0.58) for pertussis-related intellectual disability by weighting the number of cases of intellectual disability due to infectious diseases by the level of severity (using the Dutch weights for intellectual disability).

Tetanus

We estimate the incidence of tetanus using Australian notification data and apply the GBD disability weight for 60 years or over of 0.612, and duration of 2 weeks.

Measles

We derive the incidence of measles using Australian notification data. We assume annual notifications in 2003 represent all cases of measles due to enhanced surveillance (Brotherton et al. 2004). For acute measles episodes we apply the GBD duration and disability weights (2 weeks, 0.152). We use Australian hospital data to estimate the incidence of measles sequelae. In 2003 there were no hospitalisations for measles encephalitis, and only one for sub-acute sclerosing panencephalitis. For the latter sequelae we apply the Dutch disability weight for end stage disease with a duration of 9 months.

Rubella

We derive the incidence of rubella using Australian notification data which we adjust for over-reporting. Enhanced surveillance of rubella notifications in Victoria found that 27% were laboratory confirmed (Guy et al. 2004). As there is no GBD or Dutch weight for rubella we use the measles disability weight (0.152) with a duration of one week. We use Australian notification data to derive incidence estimates of congenital defects due to rubella; there were only three such cases in 2003. The classic triad of complications associated with congenital rubella infection are cataract, heart disease, and deafness. In the absence of more specific information, we derive an average disability weight and durations to reflect each of these complications.

Haemophilus influenzae type b

We derive the incidence of *Haemophilus influenzae* type b from Australian notification data. We only model the disability associated with the following sequelae – meningitis, epiglottitis, septicaemia, pneumonia and ‘other’ using data from an Australian study (Herceg 1997). Following expert advice we assume that all cases of epiglottitis and meningitis are confined to the 0–14 year age group and pneumonia and septicaemia to the 15 years or older age group. We assume that meningitis from *Haemophilus influenzae* type b is included in the hospitalisation-based estimates of total meningitis and subtract these cases from the total incidence estimates of meningitis to avoid double-counting. We use the same disability weights and durations for these sequelae as per the previous Australian burden study.

Meningitis

We estimate the incidence of meningitis from Australian hospital data which we adjust to avoid double-counting of meningitis from *Haemophilus influenzae* type b. We model meningitis as a progressive condition with acute episodes of one month, after effects lasting up to six months and subsequent lifelong effects, in some, for a range of conditions (including hearing loss, ventriculoperitoneal shunt, seizure disorder, less severe developmental problems, mental retardation and motor deficit and physical deformities). We make minor modifications to the assumptions in the Dutch study regarding proportions of meningitis cases progressing to sequelae and their associated disability using the results of a seven-year follow-up study of meningitis in Melbourne children (Grimwood et al. 1995) and expert opinion.

Septicaemia

We estimate incident cases of septicaemia from Australian hospital data. We do not adjust our estimates to account for meningitis-related septicaemia as Victorian data suggests that less than 2% of cases are due to meningitis. In the absence of a weight for this condition in its uncomplicated state, we use the Dutch weight for meningitis for an average duration of 1 month (Stouthard et al. 1997).

Arbovirus infections

We estimate the incidence of arbovirus infections using Australian notification data. Because there are no specific disability weights for arboviruses we use comparable weights from the Dutch study. For Ross River and Barmah Forest viruses we adjust estimates by 100% to account for under-reporting in endemic areas. We model Ross River and Barmah Forest viruses as a febrile illness in children aged up to 14 years and as an illness with acute and chronic stages for incident cases aged 15 years or over. Based on Australian literature we use the Dutch weight for influenza for children (1 month duration) and the Dutch weights for moderate rheumatoid arthritis (1 month duration) and mild arthritis (3.5 months in Ross River fever and half of this duration for Barmah Forest virus) for acute and chronic stages respectively in adults (Mylonas et al. 2002; Russell 2002). In general, arthralgia persists longer in Ross River virus infection than in Barmah Forest virus infection (Mackenzie et al. 1998; Russell & Dwyer 2000), therefore we halve the duration of the chronic phase in the latter.

We adjust notifications for dengue fever by 10% to account for under-reporting. Based on the literature we use the Dutch weight for malaria with a duration of 6 days (Russell & Doggett 1998; Solomon & Mallewa 2001). We use Australian hospital data to estimate the incidence of the rare and disabling sequelae dengue haemorrhagic fever. There were only two cases in 2003. The GBD weight for this condition appears too low and so we apply the Dutch weight for meningitis for just over 1 week.

We model the following flavivirus infections as 'other arbovirus infections': Murray Valley encephalitis, Kunjin virus infection, Japanese encephalitis, and flavivirus not elsewhere classified. In 2003 there were no notifications for Murray Valley encephalitis and only one case of Japanese encephalitis notified. We apply GDB estimates of the incidence of sequelae (episodes, cognitive impairment and neurologic sequelae), average disability weights, and duration for Japanese encephalitis to all other arbovirus infections.

Hepatitis

Hepatitis A

We estimate the total incidence of hepatitis A using Australian notification data, which we adjust for under-reporting (Amin et al. 1999). We assume that the 10% of incident cases represent prolonged hepatitis A. We assume that Australian hospital data on hepatitis A represent all cases of complicated hepatitis A. We calculate the number of incident cases of uncomplicated hepatitis A by deducting the prolonged and complicated cases from our total estimate. Due to the implausibility of the Dutch weight for uncomplicated hepatitis A we use the average GBD weight of 0.093 with a duration of 3 weeks (Amin et al. 1999). We assume that prolonged hepatitis A cases experience depression or fatigue for 6 months at disability

weight equivalent to the Dutch weight for mild depression (0.14) (McIntyre 1990; Willner et al. 1998). We assume durations of 4 weeks for children and 6 weeks for adults (Melnick 1995). We apply a severe disability weight for half of this time (DW 0.747), and the remaining time at the same weight as uncomplicated cases. This gives an average weight of 0.42.

Hepatitis B

We estimate the incidence of acute hepatitis B using Australian notification data and assume that all infections reported as incident are symptomatic.

We derive incidence estimates for acute symptomatic hepatitis B infection in infants from birth data and probabilities of perinatal transmission for 'at risk' mothers as reported by Kaldor and colleagues (1996). Based on the literature we assume a 40% probability of transmission if exposed. Using this estimate we can calculate the number of infants who would be infected in the absence of vaccination (Kaldor et al. 1996). As current vaccination coverage in children born to mothers 'at risk' is 95% (Menzies et al. 2004), we reduce the number of carriers from perinatal transmission accordingly. Similarly we adjust the number of perinatal infections for the probability of symptomatic infection which is 5% (Kaldor et al. 1996). Based on expert opinion, we assume a similar number of infections by casual contact in childhood and for males and females.

We base our estimates of chronic hepatitis B on a series of DisMod models. First we estimate the prevalence of adult carriers using an overall prevalence of 0.47% (O'Sullivan et al. 2004), a remission of 0.5%, and an overall relative risk of mortality of 1.5. Next we estimate the prevalence of adult carriers using incidence estimates of carriers from perinatal and casual childhood transmission assuming no vaccination had occurred. We then subtract the prevalence of carriers from childhood infections from the first model so we can use DisMod to derive the incidence of chronic hepatitis B infection in adults. This model assumes a steady state of hepatitis B infection in the population, with vaccination only recently affecting perinatal and childhood transmission rates. This is unlikely to reflect the pattern of disease over time, but in the absence of data on the trends over time, this was considered the most plausible method of modelling the disease following expert consultation.

We assume the average duration for an acute symptomatic episode to be 4 weeks (Lee 1997). We use the Dutch disability weight for acute hepatitis infection (0.21). We adjust the Dutch weight for chronic hepatitis B infection with active viral replication (0.36) following expert advice that only 15% of chronic cases have a symptomatic episode for 2 weeks each year (giving an average weight of 0.002). The methods we use to derive YLD for hepatitis B-related cirrhosis and liver cancer are described in the following section on hepatitis C. test

Hepatitis C

Due to the asymptomatic nature of hepatitis C infection we assume that all YLD are a result of hepatitis C sequelae, that is, cirrhosis and liver cancer.

There is a paucity of information on the occurrence of cirrhosis at a population level. Instead, we make use of estimates of hepatitis C-related cirrhosis occurrence from an Australian study which modelled the progression rates to various sequelae from hepatitis C incidence (Law et al. 2003). The major problem in estimating the occurrence of hepatitis C-related cirrhosis is the dramatic change in hepatitis C incidence over the last 5 decades, the relevant time period for the development of current cirrhosis cases. The best available approximation

of the pattern of hepatitis C epidemiology over the last 40 years is based on the pattern of injecting drug use over time (Law et al. 2003).

We make largely the same assumptions in the modelling of hepatitis C-related cirrhosis as in Law and colleagues (2003):

- 75% of people exposed to hepatitis C develop chronic infection
- an annual progression rate of 2% to cirrhosis
- a hepatitis C-related mortality rate of 1.5% following cirrhosis
- mean age of hepatitis C seroconversion among injecting drug users of 25 years
- a male to female ratio of 2:1 for persons who inject drugs and are hepatitis C-infected
- unlike in Law et al. (2003), we assume that 80% of those exposed to the hepatitis C (the estimated proportion of hepatitis C carriers exposed through injecting drug use) have a relative risk of mortality of 13 (Darke & Ross 2002) for an average of 14 years from the moment of exposure (as estimated for heroin dependence)
- background mortality is calculated from life tables constructed from Australian mortality and population data from 1950 to 2003.

Based on this model, we estimate 447 new cases of hepatitis C-related cirrhosis and 5,804 people living with cirrhosis due to hepatitis C in 2003. Next, we examine the Australian hospital data for cirrhosis. In all cases in 2003 with a stated underlying cause, 49.4% are alcohol related and 50.6% non-alcohol related. Based on expert opinion we attribute 5% of non-alcohol related cirrhosis to other causes and the remainder to hepatitis. We estimate the occurrence of cirrhosis due to other causes (that is, hepatitis B, alcohol abuse, and 'other') by adjusting Australian hospital data by the admissions-to-prevalence ratio observed in hepatitis C-related cirrhosis cases. We only give a disability weight for the last 3 years lived with cirrhosis at 0.31 (minus 2 months) and 0.84 for the last 2 months (effectively interpreting the Dutch weight for compensated cirrhosis as relevant for the time spent in decompensated cirrhosis and the decompensated cirrhosis weight of the Dutch as the weight for terminal liver failure).

In the previous study we assumed liver cancer occurred in around 19% of people with hepatitis C and hepatitis B. More recent data, including from a large multi-centre study of liver cancer patients in Europe, indicates that hepatitis B and C are responsible for around 19% and 40% of liver cancer respectively (Brecht et al. 1998; CDC 2001).

Malaria

We derive incidence estimates for malaria from Australian notification data. We model two aspects of malaria, episodes and neurologic sequelae and adopt GBD assumptions for disability weights and durations.

Trachoma

We model the disability associated with mild, moderate and severe vision impairment resulting from trachoma infection. We assume that trachoma related visual impairment is a problem only in remote Australia. We estimate the prevalence of trachoma-related visual impairment using the 1980 National Trachoma and Eye Health Program. Based on expert advice we adjust the prevalence downwards by one-third to account for observed decreases

in the prevalence of scarring stages that follow infectious trachoma since the national survey was conducted (Landers et al. 2005; Mak & Plant 2001). In the absence of more specific information we assume that mild and moderate vision loss have the same cause distribution by age as severe vision loss. We make minor adjustments to the prevalence of each stage by age to ensure plausibility and to reflect published estimates. We estimate the incidence and duration of trachoma in DisMod using our derived prevalence estimates. We initially model the prevalence of severe vision loss in DisMod assuming no remission and a relative risk of mortality of 1. We then use the incidence of severe vision loss from the DisMod output as 'mortality' in the moderate vision loss DisMod model. This takes the cases of severe vision loss out of the pool of susceptible cases for moderate vision loss and therefore gives more accurate average durations than if we were to use remission as remitted cases in the DisMod model, as the cases continue to be subject to the hazard of incidence. Similarly we use the incidence of moderate vision loss as 'mortality' in mild vision loss.

1B Acute respiratory infections

Lower respiratory tract infections

We base our incidence estimates for lower respiratory tract infections, including episodes of influenza, acute bronchitis and pneumonia, on Australian general practitioner data. For pneumonia, general practitioner data was thought to be more representative than hospital data as it should include those who do and do not go to hospital. We use the same assumptions for disability and durations as in the previous Australian burden study. GBD duration estimates were halved to 3.5 days for acute bronchitis, and left at 1 and 2 weeks respectively for influenza and pneumonia. Disability weights were derived using the regression model (influenza 0.047; acute bronchitis 0.132; pneumonia 0.373).

Upper respiratory tract infections

We base our incidence estimates for episodes of acute nasopharyngitis and acute sinusitis on annualised self-report data from the National Health Survey, while we model tonsillitis and pharyngitis using Australian general practitioner data. We use the data from the 1995 National Health Survey because the 2001 survey did not include questions on acute conditions. We adjust the tonsillitis and pharyngitis incidence estimate upwards by twofold to reflect the much higher rate (13 times) for the broader condition of 'sore throat' that was reported in the survey. We use derived weights and assume GBD durations, with minor adjustments where we consider this to be appropriate. For employed adults, the average number of days off work due to upper respiratory tract infections was around 0.5 of a day. The GBD assumed an average duration of 3.5 days. The self-report prevalence data probably includes a considerable number of minor infections with minimal disability. Hence we use days off work plus half a day on either side to give an average duration of 1.5 days for acute nasopharyngitis. For tonsillitis and pharyngitis and sinusitis we use the GDB durations of 3.5 days.

Otitis media

We model the following stages of otitis media; acute infection, bilateral chronic infection, and life-long deafness. We estimate the incidence of acute episodes using Australian general practitioner data. We assume that those who have relatively low disability do not seek treatment and base YLD estimates on treated numbers. We adjust our incidence estimates to allow for a higher rate of acute otitis media in Indigenous Australians in remote areas based on findings from the 1980 National Trachoma and Eye Health Program. We use the disability weight regression model to derive an appropriate weight (0.090) and assume a duration of 1 week.

We estimate the prevalence of chronic otitis media in non-Indigenous and Indigenous Australians from the National Health Survey for those people reporting otitis media as a long-term health problem. We assume that these estimates represent non-Indigenous Australians in all areas and Indigenous Australians in major city or regional areas. We adjust these prevalence estimates downwards to account only for bilateral cases using a ratio of bilateral to unilateral cases from the 1980 National Trachoma survey. We estimate the prevalence of bilateral chronic otitis media in Indigenous Australians in remote areas from the 1980 National Trachoma survey and assume that the epidemiology of bilateral chronic otitis media has not changed since the survey was undertaken. We derive the incidence and duration of bilateral chronic otitis media in DisMod using prevalence, a relative risk of 1 and remissions equivalent to durations of 3 months and 3 years for non-Indigenous and Indigenous Australians, respectively, based on Australian data (McGilchrist & Hills 1986). We base our estimates for permanent hearing loss resulting from acute infections on the GBD study. For chronic infection we apply the Dutch weight for early acquired mild to moderate hearing loss (0.110). For the small number of cases that experience lifelong deafness, we use the Dutch weight for early acquired severe hearing loss (0.233).

1C Maternal conditions

We base our incidence estimates for maternal haemorrhage, maternal sepsis, hypertension in pregnancy, obstructed labour, abortion and other maternal conditions on Australian hospital data. We adopt GBD methods except in the following instances. On expert advice we assume hypertension in pregnancy results in restricted activity (due to advised bed rest or hospitalisation) for 2 months at a derived weight of 0.117 (health state 122111), with 1 in 2,500 cases developing neurological sequelae. We model the sequela caesarean section with 2 weeks of disability at a derived weight of 0.349 (health state 222111). We base our incidence estimates for abortions using South Australian data on terminations of pregnancy as a proportion of total births (Chan et al. 2003). For abortion we model the disability of infertility resulting from the sequela pelvic inflammatory disease. We assume that 20% of hospitalised cases of pelvic inflammatory disease following abortion experience infertility from age at infection to post-reproductive age which we assume to be 45 years. We adjust our incident estimates of infertility, in the abortion and maternal sepsis models, for women who do not wish to have a child and who therefore do not experience disability. In the absence of Australian data on 'child wish', we make this adjustment using findings from a recent German study (Stobel-Richter et al. 2005). Although stress incontinence was considered a sequela of obstructed labour in the GBD study, most stress incontinence occurs in the absence of such a history. We therefore treat this condition as a category in its own right, classified under 'genitourinary conditions'.

1D Neonatal causes

Birth trauma and asphyxia

We estimate the incidence of mild, moderate and severe birth asphyxia using Australian hospital data. We separate the mild and moderate incident cases using data from the GBD study. We base our sequela estimates of neurological disability by severity of birth asphyxia (0% of mild, 25% of moderate and 100% of severe) on the GBD study.

We use the estimates of intellectual disability due to birth trauma from the overall calculations for intellectual disability by all underlying causes (see Section 2K). Stanley and colleagues (1995) estimated that 8% of cerebral palsy is associated with birth trauma. The balance of the incident cases of permanent disability is divided equally between deafness and seizures.

We assume that the duration of cerebral palsy without intellectual disability and severe hearing loss is the same as those with mild intellectual disability. We base the duration of seizure on life expectancy at birth assuming a twofold risk of dying to indicate a greater likelihood of premature mortality.

Low birth weight

We estimate the incidence of low birth weight ($\geq 1500\text{g}$ and $< 2500\text{g}$) and very low birth weight ($< 1500\text{g}$) in neonatal survivors using Australian perinatal data. We apply the sex distribution of low birth weight from the 2002 Victorian perinatal data to the Australian combined proportion for both sexes which we then apply to the total number of live births in Australia in 2003 (ABS 2004). We adjust our estimates of total neonatal deaths in 2003 (ABS 2005) using a proportion for those due to low birth weight. This was derived using an average of 2002 Victorian, Queensland, and South Australian data.

We assume the probability of disability among low birth weight survivors is 25% for very low birth weight ($< 1500\text{g}$) and 5% for low birth weight ($\geq 1500\text{g}$ and $< 2500\text{g}$) as per the GBD study. This corresponds to a total of 1,230 incident cases (596 males, 634 females) of disability in low birth weight survivors in 2003.

For hearing loss, vision loss, epilepsy, and other disability we distribute the incident cases of disability in low birth weight survivors to disability type from the GBD study. We use the estimates of intellectual disability due to low birth weight from the overall estimates of intellectual disability (see Section 2K). In addition we attribute 60% of total incident cerebral palsy cases (at 2.25 per 1,000 live births) to low birth weight.

Just over one half of the low birth weight survivors with permanent disability do not have severe neuro-developmental disability. In the absence of a defined disability weight for this health state we assume that these cases have a level of disability similar to the Dutch weight for permanent early childhood acquired moderate hearing loss. For all other sequelae we apply the relevant Dutch disability weight.

We assume the duration of cerebral palsy without intellectual disability, severe hearing loss, moderate vision loss, and mild permanent disability to be the same as those with mild intellectual disability. We base the duration of epilepsy on life expectancy at birth assuming a twofold risk of dying as compared to the mortality rates of the general population.

Neonatal infections

We estimate the incidence of neonatal infections from Australian hospital data. We assume 1 month of acute disability using the Dutch weight of 0.894 (same as for meningitis) for acute episodes.

The main long-term sequelae are deafness, motor deficit disability and intellectual disability. We estimate intellectual disability attributable to neonatal infections as part of overall estimates for all causes of intellectual disability (see Section 2K).

Other conditions arising in the perinatal period

Here we include YLD for intellectual disability due to other conditions arising in the perinatal period.

1E Nutritional deficiencies

Iron deficiency anaemia

We model the following levels of severity for iron deficiency: non-anaemic, mild anaemia, moderate anaemia and severe anaemia. We define anaemia in terms of blood haemoglobin levels as per the GBD study. We derive our incidence estimates for iron deficiency anaemia using DisMod. We base our prevalence estimates for mild, moderate and severe anaemia for men and women aged 25 years and above from AusDiab. For the younger ages we use a variety of Australian studies, assuming 60% of cases are mild and the remaining 40% moderate (English & Bennett 1990; Karr et al. 1996; Nguyen et al. 2004; Oti-Boateng et al. 1998; Sadler 1996;). Iron deficiency causes anaemia but people can be iron-deficient and not anaemic and vice versa. To calculate iron deficiency without anaemia, we first have to estimate the prevalence of total iron deficiency which includes those with and without anaemia. We assume prevalence estimates of 10% and 1% in children aged 0–4 years and 5–14 years respectively (English & Bennett 1990; Mira et al. 1996; Oti-Boateng et al. 1998; Rangan et al. 1998; Ranmuthugala et al. 1998; Sadler 1996), with figures for other ages taken directly from 1989 National Risk Factor Prevalence Survey – Iron status study. In the absence of population data on the overlap between iron-deficiency and anaemia, we assume half the cases with mild anaemia and all cases with moderate anaemia are also iron-deficient. For adults aged 15 years or over, we subtract the prevalence of iron deficiency combined with anaemia from the total prevalence of iron deficiency to avoid double-counting the disability. We use the same assumptions for disability and duration as in the previous Australian burden study.

2F Malignant neoplasms

As in the previous study, the basis of YLD estimation for malignant neoplasms is a series of models of disease progression developed by the Dutch burden of disease study team for 26 cancers for which they determined disability weights (Stouthard et al. 1997).

The disease model commences with an initial phase of diagnosis and primary therapy, with a duration of up to 12 months. After this, cases are classified as those who will and will not

be cured. Those who will be cured enter a phase of up to 5 years after which they are considered cured and have (with some exceptions as discussed below) no further cancer-related disability. Those who will not be cured enter a phase (of variable length) of remission followed by a phase of disseminated carcinoma (lasting 12 months or less), then a terminal phase (lasting 1 month) and death.

We allocate a Dutch weight to each of these phases. Where no Dutch weights were available for a specific cancer site, we extrapolate weights based on the cancer that it most resembles. The Dutch study did not derive a weight for the terminal phase of any of the cancers, so we use instead the Dutch weight for general end-stage disease.

We modify this general model to each cancer site with results from studies in the peer-reviewed literature and input from local clinicians to reflect local treatment practices.

Disease incidence data

The primary source of cancer incidence data is the AIHW & AACR National Cancer Statistics Clearing House database (AIHW & AACR 2001). This database records all cancer cases (except non-melanoma skin cancer) notified in Australia from 1982 to 2001. Cancer incidence rates in the Australian population change very slowly (AIHW et al. 2005). We apply the 2001 age- and sex-specific cancer incidence rates to the 2003 Australian population counts to estimate the 2003 cancer incidence.

Two exceptions to this approach are breast cancer and non melanoma skin cancer. The breast cancer disease model requires details of size of tumour at diagnosis which are not available from the Clearing House database. Instead, we extrapolate the proportion of new cases in each size category from 2001 BreastScreen Australia data (AIHW 2005a) and all incident cases from the AIHW breast cancer size and nodal status report for 1997 (AIHW et al. 2001). We then apply these proportions to the 2003 incident cases projected from the Clearing House database. Non-melanoma skin cancer is not a notifiable disease in Australia and so is not within the scope of the Clearing House database. Instead, we extrapolate the incidence from the results of a 2002 Australian population survey of the incidence of non-melanoma skin cancer (NCCI 2003).

Cure rate and mean survival time

To estimate the cure rate and mean time to death for those not cured for each cancer we assume a Weibull distribution for the time from diagnosis to death and apply a non-linear model to the survival curves for each cancer (Verdecchia et al. 1998). We base the survival curves on all cases recorded in the Clearing House database with a diagnosis date between 1982 and 1997 which we follow-up for death until the end of 1999 (AIHW & AACR 2001).

We base the durations of the initial treatment, disseminated and terminal stages separately for each cancer, using Dutch study assumptions, peer-reviewed literature and input from local clinicians. For those not cured, we base duration of the remission stage as the total average time to death (estimated from the Weibull model) less the sum of the other stages. For those cured, we base the duration of the stage following initial treatment as 5 years less the duration of the initial treatment stage.

Again, breast cancer and non melanoma skin cancer are the two exceptions to this approach. Since the Clearing House database does not record tumour size, we base the survival times and cure rates on an analysis of breast cancer cases by tumour size published by the South

Australian Cancer Registry (South Australian Cancer Registry 2000). Because there are no national data on non melanoma skin cancer we estimate survival times and cure rates using assumptions modelled from published studies.

Long-term sequelae of cancer

The model for cancer in the previous Australian burden study assumed, with the exception of bone cancer, that cancer sufferers have no further burden following cancer cure. However, there are some cancers that are likely to have major sequelae causing long-term burden following successful treatment. The GBD study included long-term sequelae for colorectal cancer, breast cancer, female reproductive cancers and male genitourinary cancers. In addition, we include removal of one eye for eye cancer, removal of the larynx for larynx cancer, amputation for bone cancer and long-term brain injury for brain cancer. These sequelae and their associated severity weights are listed in the table below (Table A1.2).

We estimate cancer-related rates of amputation, stoma, mastectomy, larynx, eye removal and infertility from Australian hospital data. We estimate infertility rates from cancer-related hysterectomies and assume these only apply to survivors under 40 years of age. We derive impotence and incontinence rates from a review of the literature. Results published in the literature note the similarity between the effects of treatment for brain cancer and other forms of traumatic head injury, so we assume that the rates of long-term brain injury from brain cancers are the same as the equivalent rates for head injury.

We use the GBD disability weights for stoma, mastectomy, infertility, impotence and incontinence. For the disability associated with removal of an eye, amputation, and long-term brain injury we use comparable weights from the Australian study for long-term weight for an injury to an eye, major amputation and long-term effects of a brain injury in a non-fatal accident or injury, respectively. For removal of the larynx we assume that the Dutch weight for mild hearing loss, which is defined as ‘some difficulty in actively participating in a conversation with one or more persons’, is appropriate.

Table A1.2: Extra sequelae for cancer model

Site/sequelae	Proportion of survivors with sequelae (%)	Severity weight
Colorectal cancer—stoma	0.09	0.21
Bone & connective tissue—amputation	0.08	0.30
Breast cancer—mastectomy	0.51	0.09
Female reproductive cancer—infertility	Cervix: 0.46	0.18 (ages under 40)
	Uterus: 1.00	
	Ovary: 0.64	
Male genitourinary cancer—impotence and incontinence	Prostate: 0.53 Bladder: 0.12	0.20
Brain cancer—long-term brain injury	0.05	0.35
Eye cancer—removal of an eye	0.45	0.30
Larynx cancer—removal of the larynx	0.35	0.04

2G Other neoplasms

Benign neoplasms are not notifiable in Australia. As a result we base our incidence estimates for uterine myoma and benign brain tumour on Australian hospital data.

Specifically, for uterine myoma we use the numbers of myomectomies and hysterectomies for fibroids. We assume that surgical treatment is undertaken for all cases of rapidly growing or large tumours and myoma-related symptoms. We assume a six month pre-operative state equivalent to the GBD weight for chronic pelvic pain and an additional three-week post-operative state equivalent to laparotomy (derived weight of 0.349 for health state 222211). Based on expert advice, we assume reproductive disability occurs in 3% of hysterectomy cases to whom we apply the GBD weight for infertility. We assume the additional burden associated with menorrhagia in undiagnosed women is included in our YLD estimates for this condition under the 'other genitourinary' category.

Our model for benign brain tumour is based on the model for malignant brain tumours where we model the disease in stages for survivors (diagnosis and initial treatment, and post-curative treatment) and non-survivors (diagnosis and initial treatment, pre-terminal and terminal). We adjust our incidence estimates on the assumption that 20% of hospitalisations are readmissions (Jaaskelainen 1986; Simoca et al. 1994). We base our survival estimates on Australian mortality data and assume successfully treated cases recover normal efficiency (Steiner et al. 1998) with a period of 'worry' after treatment of 2 years. In the absence of specific disability weights, we use those for malignant brain tumours.

2H Diabetes

Diabetes cases

We estimate the incidence of insulin dependent diabetes mellitus (Type 1) from the National Diabetes Register (AIHW 2003b). We use DisMod to estimate prevalence and duration,

assuming no remission and age-specific risks of dying for all diabetes from the Asia Pacific Cohort Studies Collaboration – a meta-analysis of 24 cohort studies from Asia, Australia, and New Zealand that assessed the effects of diabetes on the risks of major cardiovascular disease and death (Woodward et al. 2003). We estimate the incidence of non-insulin dependent diabetes mellitus (Type 2) for ages less than 25 years from the National Diabetes Register. We estimate the incidence of Type 2 diabetes for ages 25 years and above by subtracting the prevalence of Type 1 diabetes from the total prevalence of diabetes from AusDiab and then deriving incidence and duration in DisMod including an annual trend for the period 1980–1999 for incidence of 2.5% for males and 1.5% for females (Dunstan et al. 2002). There is no direct measurement of the trend in incidence/prevalence of Type 2 diabetes in Australia. Instead, we analyse the historical trend in diabetes mortality (which is relatively ‘flat’) and assume that this reflects the net effect of an increase in incidence and a decrease in case-fatality which in turn we assume to be equivalent to the trend in cardiovascular disease case-fatality (as the main causes of death in people with diabetes are of cardiovascular origin). Thus, we also incorporate a 20 year trend for the case-fatality rate (–2% annual for males and –1% for females). We then project incidence and case-fatality forward to the year 2003 using the same trends as above and enter these into a DisMod model for total diabetes for 2003.

We subtract out those with diabetic nephropathy to avoid double-counting as the Dutch disability weight for diabetic nephropathy includes the disability associated with diabetes per se. We use the Dutch disability weight for an uncomplicated diabetes case (0.070).

Complications from diabetes for which we calculate YLD include retinopathy, cataract, glaucoma, renal failure, neuropathy, peripheral vascular disease, diabetic foot, amputations, ischaemic heart disease and stroke.

Retinopathy

We estimate the prevalence of mild and moderate vision loss from proliferative diabetic retinopathy in the Melbourne Visual Impairment Project (Weih et al. 2000). Experts confirmed that most retinopathy is treated before it leads to more serious vision loss. Therefore we estimate the incidence and duration of diabetic retinopathy in DisMod from the prevalence estimates from the Melbourne project, assuming no remission and twice the excess risk of mortality as for all diabetes. We base the proportion of cases due to Type 1 and Type 2 diabetes on the ratio of expected cases derived from modelling data on the progression of proliferative diabetic retinopathy from time of diagnosis (NHMRC 1997b; Tapp et al. 2003b). The Dutch disability weights for mild and moderate vision loss apply.

Cataract and glaucoma

We estimate the proportion of YLD from cataract and glaucoma attributable to Type 1 and Type 2 diabetes using population attributable fractions. We base the risks of cataract and glaucoma in diabetics from the Blue Mountain Eye Study (Mitchell et al. 1997) and use severity distributions from the Melbourne Visual Impairment Project (Weih et al. 2000).

Renal failure

We estimate the incidence of diabetes-related renal failure using 2002 data from the Australian dialysis and transplant data. We use DisMod to estimate the average duration for people on dialysis, assuming a case-fatality rate reflecting observed deaths from the register. We base our annual remission estimates on observed transplant data: 85% in Type 1 diabetes cases aged 0–85 years or over for males and females combined; 6% in Type 2 cases under 65 years for males and females combined; and 0% in Type 2 cases aged 65 years or over for males and females combined. We use the Dutch disability weight for diabetic nephropathy (0.29). We estimate YLD for transplant patients assuming a case-fatality ratio reflecting observed deaths from the register and 3% ‘remission’ due to graft failure (as these patients return back to the pool of dialysis cases). We assume a high disability weight (0.29) for the first 6 months following the transplant and a GBD weight of 0.11 thereafter.

Neuropathy

Tapp and colleagues provide estimates of diabetic neuropathy prevalence by time since diagnosis (2003a). We estimate by linear regression an annual increment in prevalence, which we then apply to survivors of incident cases of Type 1 and Type 2 diabetes by age as they progress to other age groups. Based on the Rochester Diabetic Neuropathy Study only 15% of Type 1 and 13% of Type 2 cases with diabetic neuropathy are symptomatic, of which 6% of Type 1 and 1% of Type 2 are severely affected (Dyck et al. 1993). The disability weight for Type 1 is 0.099 using the disability weight regression model (health state: 111111 – 85%; 222221 – 9%; and 222331 – 6%) and for Type 2 is 0.074 (using the corresponding percentages of 87%, 12% and 1%).

Peripheral vascular disease

Tapp and colleagues provide estimates of peripheral vascular disease incidence and prevalence (Tapp et al. 2003a). We assume that only those with claudication are symptomatic. We estimate by linear regression an annual increment in the prevalence of diabetes-related peripheral vascular disease in order to derive incidence, similar to the approach for diabetic retinopathy. In the absence of Dutch or GBD disability weights for this condition we derive a weight of 0.19 using the disability weight regression model. Remission from surgery by vascular grafts is assumed to be 20%.

Amputation and diabetic foot

We estimate the incidence of diabetes-related amputations from Australian hospital data. We use GBD disability weights for these conditions and base our durations and proportions treated on expert opinion. We use amputation rate data for diabetics with foot ulcers from the Diabetes Research Foundation (Yue & Molyneaux 2005). From 1994–2005 the amputation rate for diabetics with foot ulcers was 5.3%. We calculate an average duration of 8.9 months after fitting a log normal function to follow-up data on the duration of foot ulcers. As there is no Dutch disability weight, we apply the GBD weight of 0.113.

Ischaemic heart disease and stroke

We estimate the proportion of ischaemic heart disease YLD attributable to Type 1 and Type 2 diabetes using a population attributable fraction based on prevalence and the relative risk (2.0 and 2.5 for males and females respectively) of dying from ischaemic heart disease and stroke (2.0 for both males and females) amongst diabetics from the Asia Pacific Cohort Studies Collaboration (Woodward et al. 2003).

21 Endocrine and metabolic disorders

Haemolytic anaemia

We use Australian hospital data to estimate the incidence of hereditary haemolytic anaemia, assuming that annual admissions at age 0 years represent incidence. We model beta thalassaemia and 'other' haemolytic anaemia separately to account for different durations. We assume that the average duration for beta thalassaemia is 35 years based on a USA review (US Preventive Services Taskforce 1996) and we assume that the life expectancy of persons with 'other' haemolytic anaemia is the same for sickle cell anaemia, that is, around 25 years lower than the population average. In the absence of specific weights we use the GBD weight for very severe anaemia (0.25) and severe anaemia (0.09) for beta thalassaemia and other haemolytic anaemias respectively.

Other non-deficiency anaemia

We model the disability associated with aplastic anaemia and autoimmune anaemia. We base the prevalence of aplastic anaemia on Australian hospital data. We derive incidence and duration in DisMod using prevalence data, Australian mortality data where aplastic anaemia was an underlying condition and a remission of zero. We estimate the incidence of autoimmune anaemia using hospital data and assume that the average duration is 3 months. In the absence of a specific weight for other non-deficiency anaemias we use the GBD weight for very severe anaemia (0.25).

Cystic fibrosis

Massie and colleagues (2000) found the incidence of cystic fibrosis in Victoria over a 9-year period to be 3.5 per 10,000. This estimate is very similar to information from Queensland and Western Australia (Bower et al. 2004; Queensland Health 2004). We apply the Victorian estimate to the whole of Australia. We estimate the duration of cystic fibrosis in DisMod using the above incidence, no remission, and an age- and sex- specific risk of mortality from a patient-based USA study (Kulich et al. 2003). There is no disability weight for cystic fibrosis available. As obstructive lung disease is a major sequela, and the disease is progressive and fatal, we use the disability weight for severe chronic obstructive pulmonary disease (0.53).

Haemophilia

We base our estimate of the incidence of moderate and severe haemophilia on Australian data (Street & Ekert 1996). We do not model mild cases of haemophilia since we assume they have zero disability, as bleeding only occurs as a result of injury. We use the same assumptions about severity distribution, duration and disability weights as the previous Australian burden study.

2J Mental disorders

The 1997 National Survey of Mental Health and Wellbeing, including the child mental health and low prevalence disorder components, remains the only population-based data source for our estimates of most mental disorders (ABS 1998a; Jablensky et al. 1999; Sawyer et al. 2000).

Table A1.3 summarises the mental disorders for which we calculated YLD, along with the sources of data on which our incidence estimates are based.

Table A1.3: Sources of data for mental disorders

Data source	Mental disorder
National Survey of Mental Health and Wellbeing 1997	Depression & anxiety; bipolar disorder; most substance abuse (alcohol, sedative and cannabis drug dependence or abuse); and borderline personality disorder
Low Prevalence (Psychotic) Disorders Study	Psychotic disorders
Child and Adolescent Component of the National Survey of Mental Health and Wellbeing 1997 (Sawyer et al. 2000)	Childhood disorders (separation anxiety disorder, attention-deficit hyperactivity disorder)
Epidemiological Study—National Drug and Alcohol Research Centre Technical Report No. 198 (Degenhardt et al. 2004)	Heroin dependence
Alcohol and Other Drug Treatment Services National Minimum Data Set (AODTS-NMDS) collection < www.aihw.gov.au/drugs/datacubes/index.cfm > (accessed 15 December 2005)	Stimulant dependence
Reviews of epidemiological studies	Eating disorders (anorexia nervosa and bulimia nervosa), autism, and Asperger's syndrome

Depression & anxiety, substance abuse (excluding heroin and stimulant dependence), borderline personality disorder and bipolar disorder

While the data sources have remained mostly the same as were used for the previous Australian burden study, there are a number of key methodological changes. First, we have grouped all anxiety disorders (panic, agoraphobia, social phobia, generalised anxiety disorder, obsessive-compulsive disorder, post-traumatic stress disorder and separation anxiety disorder) and the unipolar depressive disorders (major depression and dysthymia) that were previously modelled separately into a single disease category. This is based on the argument that the high degree of comorbidity and the similarity in psychological and drug treatment means that all these disorders can be considered as part of the same entity, with a continuum between mostly depressed to mostly anxious (for example (Andrews et al. 1990;

Andrews & Slade 2002). The advantage of this approach is that it takes away some of the difficulties of dealing with the frequent comorbidity among these disorders.

Second, disability weights for all conditions derived from the national mental health survey continue to be based on the mental component score of the SF-12 but for this update we calculate a per unit change in disability weight for each unit change in the mental component score and apply this to all disorders. Dutch disability weights exist for mild, moderate and severe depression as well as for six different anxiety disorders for a combined mild-moderate state and a severe state. Assuming that mild, moderate and severe are 1, 2 and 3 standard deviations, respectively, below the population mean of the mental component score we sought a mathematical function that best describes the range of disability weights. A second-order polynomial function gave the best fit. This transformation of categorical weights into a continuous scale allows us to calculate a disability weight for each respondent in the survey. Any mental component score value greater than the population mean of 52 is set to 0 and the weight for a mental component score of 20 is taken as the highest disability weight even if the mental component score is lower (this is done because otherwise the lowest mental component scores would correspond with a disability weight of greater than 1).

Third, to deal with comorbidity, we apportion the disability weights calculated in the National mental health survey equally between the comorbid mental health diagnoses for each individual. In the previous Australian burden study we did the correction for comorbidity at the level of the number of people affected and hence reported lower than actual numbers of incident and prevalent cases.

Our general model for these conditions derives incidence figures from the National mental health survey prevalence figures, using DisMod and assuming appropriate remission rates and relative risks of mortality from a meta-analysis (Harris & Barraclough 1998). We use the proportion of one-year prevalent cases reporting symptoms in the previous two weeks as an approximation of the proportion of time with symptoms and thus assume that all these conditions have a chronic nature with periods of remission in between.

For children aged 5–17 years, we use prevalence estimates for depression and anxiety from the Child and Adolescent Component of the national mental health survey (Sawyer et al. 2000). In DisMod we use a remission rate of 0.043, a pooled estimate from follow-up studies of people with various anxiety disorders (Steketee et al. 1999; Wewetzer et al. 2001; Yonkers et al. 2003) and an increased relative risk of mortality of 1.5, a value in between the range of meta-analysis estimates reported for anxiety and depressive disorders (Harris & Barraclough 1998).

The prevalence estimates for bipolar disorder in the previous Australian burden study were based on the international literature. This was because the prevalence figures from the National mental health survey were considered inaccurate due to a technical problem during the conduct of the survey. Subsequently Mitchell and colleagues (2004) have re-analysed the data and defined the prevalence of 'euphoric hypomanic/manic syndrome'. They argue that with this definition around 95% of cases of bipolar disorder are captured. For the current estimates we use the same definition and adjust the 12-month prevalence by 100/95. In DisMod we use a remission rate of 0.035 calculated from a follow-up study (Angst & Preisig 1995) and an increased relative risk of mortality of 1.96 in men and 1.76 in women (Harris & Barraclough 1998).

In this study we include all personality disorders – rather than borderline personality disorder only – but limit our estimates to those without any comorbid mental disorders. The

proportion of comorbidity between personality disorders and other mental disorders is so high that we argue that in most cases it ought to be seen as a risk factor rather than a separate condition. However, in order to capture all disability from mental disorders we include a category 'isolated personality disorder'. The remission estimate of 17% is consistent between two follow-up studies (Grilo et al. 2004; Zanarini et al. 2003). The relative risk of mortality is 1.84 (Harris & Barraclough 1998).

In the previous Australian burden study, estimates for alcohol use disorder were made separately for alcohol dependence and harmful alcohol use, and then presented as one disease category. In the current update we combine the two categories and create one DisMod model based on 12 month prevalence of any alcohol use disorder in the National mental health survey. The two other parameters in DisMod are a remission rate of 23.7% calculated from a two-year follow-up study (Booth et al. 2001), and an elevated mortality risk of 1.8 in males and 3.84 in females (Harris & Barraclough 1998).

For cannabis dependence, we assume a remission of 8% (Swift et al. 2000) and no excess risk of mortality. There are no follow-up studies of people with sedative dependence. We use the same remission as in the cannabis model and apply an excess mortality risk of 2.1 reported for 'legal' drug use (Harris & Barraclough 1998).

Heroin dependence and harmful use

Household surveys are likely to underestimate the true prevalence of heroin use (differential response between users and non-users and a greater proportion of users not living in households). Instead, we use higher estimates of regular heroin users based on triangulation between five data sources: ABS opioid deaths, ambulance attendances for drug overdose in New South Wales, New South Wales Health heroin pharmacotherapy client database, New South Wales data on arrests for drug offences, and data from the Alcohol and Drug Information Service on calls related to heroin use (Degenhardt et al. 2004). While the detailed comparison of databases was done for New South Wales, extrapolations were made for all jurisdictions by extrapolation of relationship between numbers under treatment or in contact with police and opioid mortality figures from New South Wales and the opioid deaths in each jurisdiction.

In the previous Australian burden study, we assumed very high remission after age 45 years to reflect the low prevalence of heroin use. However, expert advice that this is a cohort effect rather than a high remission effect explains the drop in prevalence at older ages. In current estimates we 'allow' DisMod to build up prevalence figures at older ages.

Back projection methods by the National Drug and Alcohol Research Centre assumes a risk of dying from overdose of 0.8% per year (Law et al. 2001). We assume a case-fatality rate of 1% to account for raised mortality from other causes. The overall relative risk calculated in DisMod is of the same order of magnitude as reported elsewhere (AIHW: Ridolfo & Stevenson 2001; Darke & Ross 2002). The disability weight for heroin dependence of 0.27 was derived by Victorian mental health experts for the previous Australian burden study and is close to the GBD disability weight estimate of 0.252.

Stimulant dependence

We decided to use treatment figures rather than the estimates of prevalence of stimulant dependence from the National mental health survey as there has been a marked increase in

the use of stimulants since 1997 and the survey results show an erratic age pattern as only few cases were identified. Instead we estimate the prevalence of stimulant dependence from the number of closed treatment episodes in 2002–2003 where the principal drug of concern was listed as amphetamines (Alcohol and Other Drug Treatment Services National Minimum Data Set) collection. We inflate these figures by 5.5 as described by McKetin and colleagues (2005).

We estimate remission by first entering prevalence, a relative risk of 0 and a case-fatality rate of 0, into DisMod. We thus get DisMod to produce an estimate of remission that best replicates the age pattern of prevalence. The average remission across all ages was 12%. We then run the DisMod model again with same prevalence, this remission rate and a relative risk of 2.1 for excess mortality as reported for 'legal drug use' (Harris & Barraclough 1998).

We derive a disability weight for stimulant dependence as we have done for all other conditions in the National mental health survey and thus assume that the same average severity found among the lower number of cases with stimulant dependence in the survey reflects that of all cases in the population.

Psychotic disorders

Estimates for psychotic disorders are based on prevalence from the Low Prevalence (Psychotic) Disorders Study conducted in Australia in 1997 as part of the National Survey of Mental Health and Wellbeing. This survey measured an overall estimate of 4.7 per 1,000 population. The low prevalence study suffered from a low response rate by general practitioners contacted in the study areas and therefore under-represented people with psychotic disorders who are solely managed by their general practitioner (Lewin & Carr 1998). Before conducting further analysis, we adjust upwards to one in three the number of people in the survey who are wholly treated by a general practitioner and adjust downwards by a factor of 0.841 to reflect only those with schizophrenia and related diagnoses and not those with a diagnosis of bipolar or affective psychosis. Annual remission is based on a number of longer term studies and is set at the median of the reported rates (1.5%) (Ciompi 1980; Harding et al. 1987; Harrison et al. 2001; Helgason 1990; Huber et al. 1980). We derive incidence and duration figures from DisMod using a 54% higher risk of mortality overall for people with schizophrenia (Harris & Barraclough 1998), with an age pattern imposed by the relative frequency by age that schizophrenia is mentioned in death records. The DisMod incidence output indicates that almost all psychotic disorders have their beginning in late adolescence or early adulthood, with a small second peak in post-menopausal women. We assume that the average time spent in psychosis is 30% (Leff et al 1992). We use a composite weight based on 30% of the GBD weight for psychosis corresponding to the estimated time spent in this state and 70% of the treated weight ($0.3 \times 0.627 + 0.7 \times 0.351 = 0.434$). The low prevalence study reported a higher proportion (61%) of people with a psychotic disorder having current delusions or hallucinations. It also stated that 86% are taking prescribed medication and that 83% of the total reported that their psychotic symptoms respond to pharmacological treatment. The first finding would indicate that our composite disability weight is too low but the second finding would support a lower weight. For the Assessing Cost-Effectiveness (ACE)–Mental Health study, disability weights for each individual in the low prevalence study were estimated using a sliding scale between the highest and lowest of Dutch disability weights for schizophrenia and anchoring individuals on this scale based on their score on the diagnostic interview for psychosis disability module that was included in the survey (Haby et al. 2004). The mean disability weight across the sample using this

method is 0.39. We decided to continue to use the 0.434 disability weight as in the previous Australian burden study.

Eating disorders

Estimates for bulimia are based on a prevalence rate of 0.7% among Swiss 14–17 year old females (Steinhausen et al. 1997). This is the mid-point in the range of prevalence between 0.5% and 1% reported from more rigorous epidemiological studies (Gilchrist et al. 1998). We calculate a remission rate of 0.21 from figures reported in a review of follow-up studies (Keel et al. 1999). We derive incidence and duration estimates for women from these figures using DisMod, assuming the age at onset is between 14 and 29 years with no increased risk of mortality. Estimates for anorexia are based on a 0.5% prevalence among females older than 15 years (Gilchrist et al. 1998; Keel et al. 1999) and a remission rate of 0.11 calculated from a follow-up study (Strober et al. 1997). We use DisMod to derive incidence and duration estimates for women from these figures, assuming the age at onset is between 14 and 29 years with an increased annual risk of mortality of 0.59% (Sullivan 1995). We assume the incidence in males is 10% of the rate in females. We use the Dutch weight of 0.28 for both types of eating disorder.

Childhood disorders

Australian prevalence data for childhood attention deficit with hyperactivity disorder come from the Child and Adolescent Component of the 1997 National Survey of Mental Health and Wellbeing (Sawyer et al. 2000). We define attention deficit with hyperactivity disorder to include children with a diagnosis on the survey and whose parents report the child having more emotional or behavioural problems than have other children of the same age. The estimates of burden of attention deficit with hyperactivity disorder were derived from prevalence rates of 6% in male children, 3% in female children, 2% in male adolescents and 1% in female adolescents. Our incidence figures were derived from DisMod, assuming an age at onset of 3–6 years and a remission rate of 0.15 (Hill & Schoener 1996). To reproduce the prevalence pattern we use a higher remission rate of 0.25 in adolescents aged 10–19 years and 0.3 thereafter. We assume no increased risk of mortality. We use the Dutch weights for both mild and moderate-to-severe attention deficit with hyperactivity disorder (0.02 and 0.15), and weight these by the severity distribution found in the 1997 survey to derive a composite disability weight.

Autism is part of pervasive developmental disorders; the other important condition in that category is Asperger's syndrome, which was described at about the same time as autism. Autism is characterised by the triad of language or communication impairment, social impairment and behavioural impairment (obsessions, rituals). However, Asperger's syndrome has only the latter two components and is not associated with intellectual disability, as is the case with 80% of autistic children. Behavioural problems are a predominant feature in children with Asperger's syndrome.

We derive the incidence of autism and Asperger's syndrome from an Australian study with data from treatment and educational support services in Western Australia and New South Wales. We assume no remission and an elevated risk of mortality as reported by Shavelle and colleagues (2001). We use the average duration of mild intellectual disability and the Dutch disability weight of 0.55 for autism, and for Asperger's syndrome an estimated weight

of 0.25 based on expert advice that the condition is worse than moderate to severe attention deficit with hyperactivity disorder but much less severe than autism.

2K Nervous system and sense organ disorders

Dementia

A door-to-door population-based two-phase investigation method (screening followed by detailed neurological examination by a psychiatrist) is the most accurate epidemiologic approach to estimate the epidemiology of dementia and Parkinson's disease (Benito-Leon et al. 2004).

We base our estimates of the prevalence of dementia for people aged 65 years or over on a recent European meta-analysis of population-based door-to-door studies conducted by the Neurologic Diseases in the Elderly Research Group (Lobo et al. 2000). We proportionately redistribute the one-third of cases that constitute 'other or mixed type' to Alzheimer's and vascular dementia. We estimate the prevalence of dementia below the age of 65 years from a recent UK study of patients aged 30–64 years (Harvey et al. 2003).

We use relative risks of mortality for Alzheimer's disease and vascular dementia from a survival study of incident cases that controlled for comorbidity (Aguero-Torres et al. 1999). The estimated mortality risk for all dementia from this is comparable to the results of the meta-analyses of dementia prevalent cases and survival (Dewey & Saz 2001; Jagger et al. 2000). We prefer using the former because it provides type-specific survival data.

We derive incidence and duration using DisMod, based on the aforementioned representative population-based studies of prevalence, assuming no remission and relative risks from the incident-based survival study. This model gives average durations across all ages for both sexes of around 4 years which was in keeping with the literature on the survival of prevalent cases (Aguero-Torres et al. 1999; Helmer et al. 2001). We model dementia as a progressive illness and discount the latter stages back to incidence of disease. We use the disability weights derived by the previous Australian burden study (which combined the Dutch weights with a severity distribution from a European population-based cohort study).

Epilepsy

We base our incidence estimates for primary epilepsy on the 1980–84 Rochester Epidemiology Project medical record linkage system (Zarrelli et al. 1999). We use these incidence estimates, assuming no differentials by sex, with age-specific remissions (Annegers et al. 1979) and an overall standardised mortality ratio of 1.3 (Tomson 2000) to derive estimates of incidence and duration using DisMod. We use the Dutch disability weight for epilepsy (0.110).

Parkinson's disease

We only explicitly model primary Parkinson's disease (ICD-10 code G20). We assume that secondary Parkinsonism is accounted for under other relevant disease categories.

We base our estimates of the prevalence of Parkinson's disease from a recent European meta-analysis of population-based door-to-door studies conducted by the Neurologic Diseases in the Elderly Research Group (de Rijk et al. 2000). There are no sex differences in the prevalence of Parkinson's disease. We do not use the Australian studies on the prevalence of Parkinson's disease since we consider them to be outliers; they give prevalence estimates two to three times higher than most of the literature (Chan et al. 2001, 2005).

We base our relative risk of mortality on the meta-analysis of prevalent cases of Parkinson's disease and survival undertaken by the Neurologic Diseases in the Elderly Research Group (Berger et al. 2000). We plot and fit the risk of mortality by age using an exponential trendline to smooth the irregular pattern by age.

We derive incidence and duration using DisMod assuming no remission and a relative risk of mortality for males and females of 3.1 and 1.8 respectively, resulting in average durations of 4.5 and 9.8 years. These durations are broadly consistent with durations reported in the literature (Elbaz et al. 2003; Fall et al. 2003; Herlofson et al. 2004; Hughes et al. 2004; Morgante et al. 2000).

We derive disability weights from an Australian patient-based cohort study (Hely et al. 1999) reporting on the distribution of Hoehn and Yahr stages (which corresponds with the descriptions of the severity states of Parkinson's disease for which Dutch disability weights are available) and survival at each 2-year interval. We model Parkinson's disease assuming that all cases start with mild symptoms and progress over time to moderate and then severe symptoms over time. From simple linear regression lines we derive an annual increase in those with moderate and severe symptoms. The proportion of cases over time who are in the moderate category is the balance between those moving from mild to moderate and those exiting moderate by shifting to the severe category. For each age group we calculate the average disability weight during the estimated average duration. As severity progresses with time since incidence and younger age groups have longer durations, disability weights are highest in the younger age groups.

Motor neurone disease

We base our incidence estimates for motor neurone disease on Australian mortality data. We assume that incident cases equal annual deaths due to motor neurone disease. Our estimates for males and females are consistent with international literature for males (Chancellor et al. 1993). We assume average durations of 2.9 years for those aged 0–64 years and 1.9 years for people aged 65 years or over. We base our duration assumptions on Australian and international literature (Forbes et al. 2004; Sach 1995). In the absence of a specific disability weight we use the Dutch weight for progressive multiple sclerosis (0.67).

Multiple sclerosis

We estimate the prevalence of multiple sclerosis using 1981 and 1996 estimates of multiple sclerosis for some Australian states and territories (Barnett et al. 2003; Simmons et al. 2001) with extrapolations based on latitudinal differences for jurisdictions with no estimates. We assume that changes over time represent improvements in identification rather than changes in epidemiology. We derive incidence and duration using DisMod assuming no remission and age and sex specific case-fatality rates based on a 25-year New Zealand cohort study (Miller et al. 1992).

In 10.8% of patients the disease has a progressive course from the onset (Roxburgh et al. 2005). The median time it takes to reach an Expanded Disability Status Scale score of 6 (equivalent to having to use a cane) in those with a relapsing-remitting course is 30 years (Tremlett et al. 2006). We use the Dutch weights for relapsing-remitting (0.33) and progressive (0.67) phases and assume that those with relapsing-remitting disease have 30 years at the lower disability weight and the remainder at the higher disability weight level.

Huntington's chorea

Huntington's chorea is modelled in DisMod using prevalence from the literature (McCusker et al. 2000), assuming no remission and mortality data. We assume a duration of 20 years for the younger age groups and apply the durations from DisMod for ages 65 years or over. Assuming similar progression of disease as in Parkinson's, we adopt the weights for the three stages of this disease.

Muscular dystrophy

For muscular dystrophy in males, we use the average incidence rates from New South Wales, Victoria, Queensland, Western Australia and the Australian Capital Territory (Cowan et al. 1980; Emery 1991). The incidence for females is calculated by applying the sex ratio from mortality data. In the absence of specific weights for this condition, we assume the initial symptomatic phase is similar to the initial stage of Parkinson's disease, the phase in which walking becomes impossible is similar to that of paraplegia, and the final stage is equivalent to quadriplegia.

Vision loss

Our incidence estimates for vision loss are based on the results of the Melbourne Visual Impairment Project, which assessed visual acuity and the prevalence by cause of mild, moderate and severe visual impairment in a sample representative of Victorians (Weih et al. 2000). For glaucoma, refraction errors, macular degeneration and the category 'other vision loss', we derive incidence and duration of related visual impairment using DisMod, assuming no remission and a relative mortality risk of 1. For glaucoma we use Dutch disability weights for mild, moderate and severe vision loss to derive a composite disability weight from the severity pattern across all ages (as the age-specific data are based on small numbers). For macular degeneration, refraction errors and 'other vision loss' we derive age-specific disability weights.

We estimate the incidence of mild and moderate cataract-related vision impairment using Australian hospital data assuming that 50% of surgically corrected cases had vision loss in both eyes prior to operation for 1 year on average and that 90% of cases are mild and 10% are moderate. We estimate the prevalence of un-operated cataracts as the difference between the prevalence of cataract-related visual impairment estimated by the Melbourne study and the number of surgical corrections. This leads to a small estimate of un-operated cataracts in the elderly over 80 years of age. We use this to estimate the incidence of un-operated cases of cataract-related severe vision loss in DisMod, assuming no remission and a relative risk of 1.5. For cataract-related vision loss at ages 0-14 years we assume duration of 2 years and for ages 15 years or over we assume a 1-year duration. Incident cases of un-operated cataract were assumed to be prevalent cases waiting on average 1 year for cataract surgery. We use

Dutch disability weights for mild and moderate cataract-related vision loss. For severe cataract-related vision loss we estimate a combined disability weight using the Dutch weights for each of the stages along with prevalence data from the Melbourne study to derive combined stages age-specific disability weights. The proportion of glaucoma and cataract-related vision loss attributable to diabetes is then determined from relative risks from the Blue Mountain Eye Study (Mitchell et al. 1997) and only non-diabetes-related vision loss is included in the YLD estimates for these categories.

Hearing loss

We model hearing loss as a progressive condition with mild (25–34 dB and 35–44 dB), moderate and severe stages so that prevalent cases with moderate or severe impairment are regarded as incident cases of mild impairment at an earlier age. We use survey prevalence data from South Australia (Wilson et al. 1999), initially modelling the prevalence of severe hearing loss, no remission and a relative risk of 1 in DisMod. We use incidence of severe hearing loss from the DisMod output as ‘mortality’ in the moderate hearing loss DisMod model; this takes the cases of severe hearing loss out of the pool of susceptible cases for moderate hearing loss and hence gives more accurate average durations than if remission were used as remitted cases in the DisMod model, as the cases continue to be subject to the hazard of incidence. Similarly, we use incidence of moderate hearing loss as ‘mortality’ in mild hearing loss (35–44 dB) and incidence of mild hearing loss (35–44 dB) as ‘mortality’ in mild hearing loss (25–34 dB). From examination of the prevalence data by level of severity and age, and assuming that all cases progress from the mildest to most severe category, it seems reasonable to assume that on average progression to the next severity level occurs at 5 year intervals between mild (25–34 dB) and mild (35–44 dB), and at 10 year intervals from mild (35–44 dB) to moderate and moderate to severe. From the cross-sectional data on prevalence it is not possible to estimate these progression times exactly. However, to be consistent with other disease models where subsequent severity levels for the same health state are discounted back to first incidence, we apply a 25-year lag for severe hearing loss, 15 years for moderate and 5 years for the mild (35–44 dB) categories. Dutch weights of 0.04, 0.12 and 0.37 apply for mild (35–44 dB), moderate and severe hearing loss, respectively. For the mild (25–34 dB) category we assume a disability weight of 0.02, half that of the mild (35–44dB) category.

Intellectual disability

Intellectual disability is categorised into the following levels: mild, moderate, severe and profound, with intelligence quotient (IQ) ranges of 50–69, 35–49, 20–34, <20 respectively. This categorisation is based on the Dutch disability weight criteria.

We estimate the incidence of mild-moderate and severe intellectual disability using the Intellectual Disability Exploring Answers Database, a Western Australian population-based dataset of children with intellectual disability identified through disability and educational services between 1983–1996. We adjust the severity distribution of incidence data to account for unspecified cases and redistribute cases so that the severity level as defined by IQ is comparable to the Dutch disability weight criteria. Then we extrapolate incident cases by the two severity levels (mild-moderate and severe) to four levels of severity (mild, moderate, severe and profound) using the average severity distribution from two Australian studies (Einfeld & Tonge 1996; Wellesley et al. 1992). We assume that because neither study

recruited cases from school services, mild cases were underestimated and base our estimation of mild cases on the balance of the mild-moderate category. This gives the following proportionate distribution of incident cases by severity: mild (76%), moderate (14%), severe (7%) and profound (3%).

In order to derive plausible durations of intellectual disability by the four stages of severity we calculate the proportional difference in life expectancy by level of severity of intellectual disability in comparison to the life expectancy of the general population from a 35-year Finnish follow-up study (Patja et al. 2000).

We model the incidence and duration of intellectual disability in DisMod assuming that 90% of intellectual disability, based on Australian population data, occurs in the first year of life and the remaining 10% occurs in the 1–4 age group, no remission, and a relative risk of mortality that gives an average duration by severity level based on the extrapolation of Finnish data to the 2003 Australian life table.

We do not include the YLD for intellectual disability as a discrete category in the main listings of this burden study. Instead, incident cases of intellectual disability are attributed to underlying causes (such as congenital disorders, epilepsy, autism, perinatal conditions, meningitis, brain tumours and cerebral palsy) using findings from the Australian Child to Adult Development Study, a longitudinal study of behavioural and emotional problems in 429 young people with intellectual disabilities. We use data from two publications of this study to produce the proportionate distribution of the underlying cause of intellectual disability by severity level and sex (Mowat et al. unpublished; Partington et al. 2000). We calculate YLD for each underlying cause using incidence and duration derived from DisMod and the Dutch disability weights for mild, moderate, severe, and profound intellectual disability.

Migraine

We base our prevalence estimates for migraine on the National Health Survey data and our incidence estimates for migraine on international data (Stewart et al. 1991). We estimate the incidence and duration of migraine in DisMod using prevalence, incidence, and a case-fatality rate of zero. Within DisMod, we use manual smoothing to extrapolate incidence to older ages. We assume that 20% of cases receive treatment in developed countries. We assume that the average duration for untreated and treated episodes is 24 hours and 6 hours respectively. We derive average disability weights for untreated and treated models using frequency, severity, and disability weight data from *Global burden of migraine in the year 2000* (Leonardi & Mathers 2003).

2L Cardiovascular disease

Ischaemic heart disease

Three health states are modelled separately for ischaemic heart disease: angina pectoris, acute myocardial infarction and heart failure. We model the incidence of angina pectoris as the number of admissions to hospital without any mention of angina in any previous admission in 15 years of linked hospital records in Western Australia (Department of Health of Western Australia et al. 2005; Holman et al. 1999) and adjust by the ratio of admissions for

angina pectoris between Western Australia and the whole country. We model angina pectoris pre- and post-myocardial infarct together. The duration is determined in DisMod, assuming remission estimated from the number of revascularisation procedures from Australian hospital data and age- and sex-specific case-fatality rates calculated over the period 1998–2003 in 'prevalent cases' of angina pectoris (that is, anyone with an admission for angina pectoris since 1988 and still alive over the follow-up period).

Assuming that about half of the declining ischaemic heart disease mortality reflects change in the case-fatality rate rather than incidence (Unal et al. 2004), we apply half of the ischaemic heart disease mortality trend observed over the period 1979–2003 in DisMod to incidence and the other half to case-fatality.

We assume 95% of angina is experienced at the mild-moderate level with the corresponding Dutch disability weight of 0.08, and the remaining 5% with a weight of 0.57.

For people discharged alive following acute myocardial infarction in 2003, we calculate a period of 3 months of disability at the GBD treated disability weight of 0.395.

Heart diseases resulting in heart failure

Population-level prevalence or incidence information on heart failure is absent in Australia and scarce elsewhere. In 2001, by extrapolation from US studies a rough estimate was made of about 300,000 prevalent cases of heart failure in Australia (Krum 2001). Complicating factors in the estimation of heart failure prevalence are that estimates from other countries and different time periods may not apply to the current Australian situation. Ischaemic heart disease is the underlying cause of heart failure in the majority of cases, and there has been a steady decline in the risk of ischaemic heart disease since the early 1970s combined with improved survival due to improvements in therapeutic options. The first would cause a reduction in prevalence, while the latter would lead to higher prevalence. It is not clear what the net effect of these two influences would be on the prevalence of heart failure.

Using hospital data is also not straightforward as the current wisdom is that there has been a change in the case load of people presenting to tertiary health facilities with this condition, following the wider use of improved pharmacological treatment combinations since the 1990s, resulting in a greater proportion of cases being successfully treated in primary care. Nevertheless, our model for heart failure starts with a description of the epidemiology of hospitalised heart failure, for which we have extensive information from Western Australia. From the linked data set of all hospitalisations and deaths in this state, we identify people who presented to hospital with heart failure (either as a primary diagnosis or as an associated condition) at any time in the period 1990–2003. To derive case-fatality, we calculate the number of years lived between 1998 and 2003 by anyone who had ever been admitted with a diagnosis of heart failure since 1990. The case-fatality rate was then taken as the number of deaths over person-years of follow-up in 5-year age groups after subtracting out the background mortality.

The complete descriptive epidemiology in this group is derived in DisMod from incidence and case-fatality, the third parameter being zero remission (that is, people do not recover from heart failure). We include in this model a declining trend in case-fatality over the last 10 years of 3% per year for males and 1% per year for females (derived from our survival model), and a 2% decline in incidence per year for both males and females over the last 35 years. This latter figure is half the annual decline we observe for ischaemic heart disease mortality over this period, ischaemic heart disease being the major driver of heart failure

risk. The other half of the decline in ischaemic heart disease mortality we assume to be due to improvements in case-fatality (see above) (Unal et al. 2004).

There is little information on the incidence of heart failure in the community (that is, not yet diagnosed cases and those diagnosed but treated in the primary care setting without requiring hospitalisation). We assume that this group has less severe disease with better survival compared to their hospitalised counterparts. We also assume that when they die, it is less likely that heart failure will be mentioned as the underlying cause of death. We have data on the number of hospitalised cases of heart failure who died with heart failure as the underlying cause of death and we know the overall number of deaths coded to heart failure. Assuming that the linkage of hospital and death records in Western Australia is complete, we then assume that the balance of heart failure coded deaths occur in never-hospitalised cases of heart failure. In the absence of data to characterise the never-hospitalised cases of heart failure we make two assumptions. First, to account for lower severity we assume that their case-fatality rate is lower by 25%. Second, we assume that deaths in non-hospitalised heart failure cases are 25% less likely to be coded to heart failure.

Among hospitalised cases that die, the probability of receiving an underlying cause of death code of heart failure (428 in ICD-9 and I58 in ICD-10) is 3.6% in males and 5.3% in females. If non-hospitalised cases are 25% less likely to be assigned a code of heart failure the percentage of total excess deaths coded to heart failure would be 2.7% in males and 3.9% in females. From this we can derive the total number of deaths due to heart failure in non-hospitalised cases (3,199 in males and 3,958 in females over the period 2001–2003). By adding in the 3,833 deaths from ever-hospitalised cases of heart failure in males and 4,186 in females, we can calculate the average population mortality rate of heart failure over the period. These rates (calculated by age and sex) are the inputs to a second iteration of DisMod, together with zero remission and the case-fatality rate of the first DisMod model of hospitalised heart failure cases adjusted downwards to reflect the proportion of never-hospitalised cases having 25% lower case-fatality. We continue to use the same assumptions on trends in case-fatality and incidence as in the first model. The output of the second DisMod iteration then gives us the incidence, prevalence and average durations for all heart failure, which feed into our YLD calculations. The total prevalence of heart failure in Australia in 2003 is thus estimated to be 220,000 cases.

We then identify the underlying causes for all heart failure cases – rheumatic heart disease, hypertensive heart disease, ischaemic heart disease, pulmonary heart disease, inflammatory heart disease, non-rheumatic valvular heart disease – in the Victorian linked hospital admission dataset between 1996 and 2002, if any of these were mentioned as a cause in the six years of hospital admission data. We then adjust the proportions, by age and sex, of all underlying causes so they add up to 100% to account for cases with none or more than one underlying cause identified. We use the duration, together with the incidence and prevalence estimates initially obtained from the heart failure model described above, multiplied by the proportion of heart failure cases for each of the above six underlying causes, to calculate the YLD for each of these conditions (including ischaemic heart disease).

Stroke

We model stroke in terms of the following health states: a short period of disability for those who die in the first 28 days, survival beyond 28 days with no permanent impairment at one year after onset, and survival beyond 28 days with permanent impairment. Admissions for stroke in the year 2003 are the starting point for our estimate of incidence. To get an

approximation of first-ever stroke incidence we take the ratio of hospital admission figures from the North-East Melbourne Stroke Incidence Study (NEMESIS) area during the time of the study to the reported NEMESIS first-ever incidence figures (Thrift et al. 2000) and apply this ratio to 2003 Australian hospital admissions for stroke. Next, we subtract a proportion of cases that die, using a 28-day case-fatality rate by stroke subtype as reported by Thrift and colleagues.

The case-fatality rate of stroke comes from the Western Australian linked database using a similar approach to that described above for heart failure. The case-fatality rate for DisMod is the excess mortality in prevalent cases, defined in our analyses as anyone still alive at the beginning of the follow-up period (mid 1998–mid 2004) with a mention of stroke during any admission between 1989 and 1998 as well as any new cases of admitted stroke during follow-up. Follow-up time and numbers of deaths were analysed for each 5-year age group and the overall case-fatality rate reduced by the relevant background mortality.

Analyses by Judy Katzenellenbogen in Western Australia for her PhD indicate that after the first 28 days the case-fatality rate does not vary significantly by type of stroke and that the DisMod assumption of a case-fatality hazard that varies with age but not with time since stroke is plausible.

Disability weights are derived from one-year follow-up data of stroke survivors in the Perth Community Stroke Study analysed by Judy Katzenellenbogen to compare health status information before and at 4 months and 1 year after the stroke event.

Other cardiovascular disease

Heart failure is the main disability from rheumatic heart disease, non-rheumatic valvular disease, hypertensive heart disease and the group of inflammatory heart diseases (including myocarditis, cardiomyopathy, endocarditis and pericarditis). The proportions of heart failure cases for each of these causes are derived as described above for all heart failure. For rheumatic heart disease and non-rheumatic heart disease we do a separate DisMod model based on heart failure prevalence for these causes and taking into account the remission through surgical interventions using Australian hospital data.

For aortic aneurysm, we assume the hospitalisation rate reflects incidence. For peripheral vascular disease, we assume the hospitalisation rate reflects prevalence at all ages. We derive the incidence from DisMod, assuming a relative risk of 2 and a remission rate of 0.1, which approximates the number of surgical interventions as a proportion of total prevalent cases.

For aortic aneurysm, we assume a one-month period of disability during treatment and no residual disability for those who survive treatment. Without a disability weight for this health state, we use the derived weight for laparotomy (0.349). For peripheral vascular disease, we use derived weights of 0.243 and 0.257 for men and women respectively, based on severity distributions from the 1993 Australian disability survey. Weights for amputations are from the GBD study.

2M Chronic respiratory diseases

Chronic obstructive pulmonary disease

We estimate the prevalence of chronic obstructive pulmonary disease for cases with a forced expiratory volume in one second of less than 70% of predicted (excluding those with a doctor defined diagnosis of asthma) using the 1994–95 Busselton Study (Knuiman et al. 1999). While this study sample comprises a selected rural population in Western Australia, we assume the data are representative of prevalence in all areas of Australia. We use DisMod to estimate the incidence of chronic obstructive pulmonary disease in 1994, assuming no remission and a relative risk equivalent to that calculated from death rates attributed to smoking (see section on risk factors). We include a trend of -2% per year for males and 3% per year for females based on trends in chronic obstructive pulmonary disease mortality since 1979. We calculate 2003 incidence estimates by applying age- and sex-specific trends (based on mortality) to the 1994 incidence estimates. We then use DisMod with the same assumptions about remission and relative risk of mortality to model prevalence, age of onset and duration for 2003. We derive a composite average disability weight for males (0.168) and females (0.159) using the Dutch weights for mild, moderate and severe chronic obstructive pulmonary disease and the proportionate distribution by level of severity of dyspnoea from the Busselton Study. We add the proportion of heart failure cases attributed to ‘pulmonary heart disease’ on the basis that chronic lung disease is the underlying cause.

Asthma

We estimate the prevalence of asthma for cases that have a positive airway hyper-responsiveness test and wheezing in the last 12 months from the literature (Bauman et al. 1992a; Peat et al. 1992, 1994, 1995; Toelle et al. 2004). Although these two criteria may underestimate the ‘true’ prevalence of asthma, a reliance on self-reported wheeze alone overestimates figures by up to a third (Toelle et al. 1992; Van Asperen 1995). We estimate the prevalence of asthma in children aged 1–2 years to be 5.75%, using a report of ‘wheeze’ from the US (Martinez et al. 1995) which we adjust by 42% to obtain an estimate that reflects those with wheeze having asthma (Peat et al. 1994, 1995; Toelle et al. 2004). We estimate the prevalence of asthma to be 12.3% in boys and 8.8% in girls aged 3–18 years, using an average of 3 studies from 1992 to 2002 (Peat et al. 1994, 1995; Toelle et al. 2004) and a male-to-female ratio of 1.4:1 (Gergen et al. 1988). For adults, we average the prevalence data from the early 1990 studies (Bauman et al. 1992a; Peat et al. 1992, 1994, 1995) since these were the last studies in adults to have used a positive airway hyper-responsiveness test and assume no change in the prevalence of asthma over time based on the literature and the observed trend in children. We use a male-to-female ratio of 1:1.5 (DHS 2002) to give an estimated 2003 asthma prevalence of 5% and 7.5% in male and female adults. We derive incidence estimates from DisMod assuming age-specific remission rates from a follow-up study in the US (Bronnimann & Burrows 1986), which are consistent with overall remissions reported by Australia studies (Xuan et al. 2002). From findings reported by Bauman and colleagues, we calculate that asthmatics are symptomatic 12% of the time (Bauman et al. 1992b). Rather than use the Dutch weight for this health state (0.36), which we consider to be for a more severe health state than the average for symptomatic asthmatics in the population, we use a derived weight of 0.229 based on the severity distributions found in the 1998 Australian disability survey (ABS 1998b) and the disability weight regression model. The remainder of the time

we assume is spent in a state equivalent to the Dutch weight for asthma controlled by treatment (0.03). This results in a combined weight of 0.054.

2N Diseases of the digestive system

Peptic ulcer disease

In the absence of Australian population data on the frequency of peptic ulcer disease, we assume that all incident cases of peptic ulcer disease visit a general practitioner and base our estimates on the Australian general practitioner data. We assume that 83% of cases are treated by *Helicobacter pylori* eradication therapy, which has a cure rate of 90% (Mollison et al. 1999). We model those who are cured using eradication therapy as being symptomatic for one month, with no residual disability. We assume that the remainder of those who are treated but not cured (including those receiving alternative treatments) receive relief from their treatment but remain with the condition for the GBD duration. Untreated cases we assume to be symptomatic for the same period. Because the annualised Dutch weight for peptic ulcer disease is implausible, we use derived weights from the Dutch study for both symptomatic and treated states.

Cirrhosis of the liver

The methods of deriving estimates of alcohol-related cirrhosis and the category of 'other' cirrhosis have been described in the section on hepatitis.

Inflammatory bowel disease

We model two manifestations of inflammatory bowel disease: Crohn's disease and ulcerative colitis. We estimate the incidence of inflammatory bowel disease in adults from a European study (Shivananda et al. 1996) and for children we pool estimates from a number of international studies based on a recent review (Griffiths 2004). The relative risks of mortality due to the two types of inflammatory bowel disease were based on the findings of a recent large UK study which showed that inflammatory bowel disease was associated with a small overall increase in mortality after controlling for smoking and sex (Card et al. 2003). We assume no remission and derive a composite disability weight (0.224), assuming that 20% of time is spent with active exacerbation and the remainder is in 'remission' (Griffiths 1995; Hendriksen et al. 1985; Stonnington et al. 1987).

For inflammatory bowel disease (and vascular insufficiency of the intestine, diverticulitis and intestinal obstruction), we assume that a proportion of cases have more complicated surgery involving the creation of a stoma (a surgical opening in the skin of the abdomen for excretion of faeces) that can be either permanent or temporary. We estimate the incidence of inflammatory bowel disease cases that receive a temporary or permanent stoma from Australian hospital data. We apply the ratio of stoma for Crohn's disease to stoma for ulcerative colitis from an analysis of Victorian linked hospital data. Similarly the average duration of temporary stoma was estimated from Victorian hospital data from 1998–99 to 2001–02 to determine if they were closed and, if closed, the time to closure. The duration of permanent stoma was taken to be the same as the duration of the respective condition. We

assume stomas not yet closed within this period remain open indefinitely. In the absence of a specific weight for this condition, we derive a weight (0.204) from the disability weight regression model.

Other diseases of the digestive system

We base the incidence estimates for appendicitis, intestinal obstruction, diverticulitis, gall bladder and bile duct disease, pancreatitis and vascular insufficiency of the intestine on the numbers of people with a relevant hospital procedure or diagnosis from Australian hospital data. With the exception of appendicitis, these conditions were not considered in either the GBD or Dutch studies. We adopt a 2-week duration for appendicitis, and a 3-week duration for gall bladder and bile duct disease, intestinal obstruction, vascular insufficiency and pancreatitis. For each of these conditions, we assume the GBD weight for appendicitis. For gall bladder and bile duct disease, we use cholecystectomies or bile duct incisions but ignore people admitted with un-operated cholelithiasis on the assumption that these people are largely asymptomatic.

20 Genitourinary diseases

Nephritis & nephrosis

We base the incidence of dialysis and transplant patients on the Australian dialysis and transplant data from which we derive durations for both categories of patients using DisMod. For dialysis patients, we use case-fatality rates to match observed deaths and remission through transplant, and apply the Dutch weight for diabetic nephropathy (0.290). In the first 6 months after transplant, we assume a health state equivalent to the Dutch weight for diabetic nephropathy (0.290). For the remaining period with the transplant, we use a weight of 0.11, which is equivalent to both the GBD weight for treated renal failure and the Dutch weight for 'uncertain prognosis'. We derive untreated end stage renal failure from the difference between dialysis or transplant deaths and total renal deaths, to which we apply an average duration of 1 year prior to death at the GBD weight for untreated renal failure (0.104). We use Australian dialysis and transplant data on underlying renal disease distribution to attribute YLD from diabetic nephropathy to diabetes, analgesic nephropathy to the injury category of medical misadventure, and congenital dysplasia and polycystic kidney disease to congenital urogenital disease, and retain only those for primary renal disease in the 'nephritis & nephrosis' category.

Benign prostatic hypertrophy

We base the incidence of benign prostatic hypertrophy on Australian hospital data. Based on expert advice we adjust the number of benign prostatic hypertrophy cases upwards to account for the proportion of cases that receive medical instead of surgical treatment. We also assume, based on expert opinion, that half of all benign prostatic hypertrophy cases receive surgical treatment, a proportion of whom experience complications or continuing symptoms following surgery (1% with lifelong incontinence at a derived weight of 0.204, 15% with lifelong impotence at the GBD weight of 0.195, and 5% with urethral stricture for 4 weeks at the GBD weight of 0.151). Of those opting for medical treatment, we assume 70%

use alpha-blocker drugs, of which half are cured. The other half may then try surgery. We assume none of those receiving drugs other than alpha-blockers are cured. We apply the GBD weight for symptomatic benign prostatic hypertrophy to each of these intervention pathways assuming the following durations: 1.5 years for surgery, 1 year for successful medical treatment, 2 years for unsuccessful medical treatment then surgery, and lifelong for unsuccessful medical treatment but no surgery.

Urinary incontinence

We derive incidence rates of incontinence from DisMod using prevalence figures reported in a review of Australian and international literature (AIHW: Lea 1993) and from Women's Health Australia. We assume that a number of diseases and injuries are associated with this condition, most of which are more prevalent at older ages, and that the underlying causes are multi-factorial and interrelated. Based on a multivariate analysis (Chiarelli et al. 1999), we assume that, while all disability from incontinence among younger men and younger and middle-aged women belongs under this category, half that experienced by middle-aged and older men and older women is already captured under other conditions either explicitly (for example, as a sequela for benign prostatic hypertrophy among men) or implicitly as part of the overall weightings for these conditions (for example, severe stroke). For unaccounted incontinence, we apply an average of the GBD weight for moderate incontinence and the derived weight for benign prostatic hypertrophy-related severe incontinence using severity distributions from the 1998 Australian disability survey.

Infertility

We estimate the prevalence of infertility from a 1988 population survey of infertility, surgical sterility and associated reproductive disability in Perth, Western Australia (Webb & Holman 1992). This survey indicates that of the 3.5% of couples with non-surgical infertility, 68% have an associated reproductive disability defined in terms of the couple being unable to achieve a desired level of reproductive function. From a review of patients at an Adelaide infertility clinic indicating that 83% of couples with reproductive disability seek assisted reproductive technologies, 30% of whom achieve a pregnancy within 2 years (Weiss et al. 1992), we derive a net prevalence of 1.02% and 0.73% for short-term reproductive disability and 0.67% and 0.48% for long-term reproductive disability in females and males respectively. The causes of infertility are derived from recent national data on assisted conception and reproduction (AIHW: Dean & Sullivan 2003; AIHW: Ford et al. 2003). For short-term cases, we assume incident cases equal prevalent cases divided by the duration, which we assume is 2 years. For long-term cases, we derive incidence and durations from DisMod assuming non-zero remission rates from ages 45 years or over to account for declining prevalence of reproductive disability reflecting adoptions and changes in reproductive goals. For women, we subtract from the total number of long-term incident cases the estimated incidence of infertility as a sequela to maternal sepsis, abortion and pelvic inflammatory disease, the disability of which is calculated under chlamydia and gonorrhoea. We determine the duration of long-term infertility by subtracting the age at onset estimated in DisMod from 45 years. GBD weights are used for both short- and long-term reproductive disability.

Other genitourinary diseases

For this residual category, we assume the application of a simple YLD to YLL ratio of one across the age groups is sufficient to capture the morbidity from other genitourinary diseases in men. This method, however, does not capture the significant burden experienced by women, particularly at younger ages. We therefore calculate separate models for menstrual disorders and hysterectomies for menorrhagia, genital prolapse and endometriosis.

We base our estimates for menstrual disorders on women who report they often have severe period pain or premenstrual tension in the last 12 months from Women's health Australia. For severe period pain we assume a duration of 1 day per month and a disability weight similar to that for caesarean section. For menstrual tension we assume a duration of 2 days each month and we use the disability weight for mild depression. We use DisMod to model the conditions, assuming no excess mortality and remission of 0.1 for ages less than 50 years.

We model disability from hysterectomies associated with menorrhagia, genital prolapse and endometriosis in terms of disability from both the procedure and the resulting infertility. We derive the number of procedures from hospital data and we assume a 2-week duration at the derived weight for laparotomy of 0.349 (compare with estimates for caesarean section). Following the findings of a survey of surgical sterility in Perth (Webb & Holman 1992), we assume the majority of women who undergo a hysterectomy have completed their reproductive objectives, and that infertility leads to disability in 3.3% of cases with endometriosis. We apply the GBD weight for infertility.

2P Skin diseases

Eczema, acne and psoriasis

We model the incidence of severe eczema (that is, an episode in the past 12 months that disrupts sleep on average one or more nights per week) using self-reported prevalence data from a study of Melbourne school children (Robertson et al. 2004) and from the National Health Survey for adults (ABS 2001c). For other skin conditions we limit our estimates to severe acne and moderate and severe psoriasis. Prevalence figures for acne are based on a study of Australian school children and a study of adults in Central Victoria (Kilkenny et al. 1998; Marks et al. 1999). Prevalence figures for psoriasis were derived from the National Health Survey and from the central Victorian study (Marks et al. 1999; Plunkett et al. 1999). We derive incidence and duration estimates from DisMod assuming no excess mortality and a remission rate of 0.1 for eczema (Thestrup-Pedersen 2003), 0.27 for acne (assuming 70% spontaneous remission after 4 to 5 years) and 0.3 for psoriasis. For eczema we derive a disability weight (0.019) from the disability weight regression model which we adjust for 3 symptomatic episodes per year lasting 6 weeks in total. For acne we use the unadjusted disability weight for eczema from the disability weight regression model (0.056) and for psoriasis we apply the GBD weight for vitiligo.

Other skin diseases

We model the disability associated with chronic leg, skin and varicose ulcers, excluding decubitus and cellulitis which we assume are captured elsewhere. We use the weighted

incident cases of skin ulcers from Australian general practitioner data to estimate the incidence of other chronic skin ulcers. YLD for diabetic foot is included within the diabetes mellitus model. To avoid double-counting diabetic foot we adjust our incident estimates for skin ulcers using Western Australian aetiological data on the proportion of leg ulceration cases that had diabetes (Baker et al. 1992). In the absence of more specific information we use the same assumptions for duration (8.9 months) and disability (0.131) as the diabetic foot model.

2Q Musculoskeletal diseases

Musculoskeletal diseases are highly prevalent in the population. The fair to good test-retest reliability of self-reported musculoskeletal diseases and the consistent correlation with pain make health survey self-reports of some use to measure musculoskeletal conditions. Although the prevalence of most musculoskeletal diseases differs substantially depending on the measurement method, with self-report showing the highest prevalence, the pattern of prevalence in men and women is often similar. A higher prevalence of herniated disc of the back and gout is found in men, whereas for most other musculoskeletal diseases the prevalence is higher among women than among men (Picavet & Hazes 2003).

Rheumatoid arthritis

Given the small numbers in Australian studies on rheumatoid arthritis and problems with proper incidence and remission measurement, we base our incidence estimates for this condition on the international literature. For juvenile chronic arthritis, we use findings from a population study during 1984–1988 in Sweden (Gare & Fasth 1992). For adults, we use results from a 40-year follow-up study of a population-based cohort in Rochester, Minnesota, USA (Doran et al. 2002). We derived durations from DisMod assuming a relative risk of mortality of 1.6 at ages 15 years or over (Pincus et al. 1994), with no increased risk for children, and a remission rate of 0.04 (Prevoe et al. 1996) indicating that, while drug treatment may slow the disease process and remission is the ultimate endpoint of treatment, most therapeutic options have fallen short of achieving this (Sesin & Bingham 2005). Because progression through the three stages of rheumatoid arthritis described by the Dutch weights is relatively rapid, we do not model this condition as progressive. Rather we apply an average of the Dutch weights using severity distributions for American adults (Hakala et al. 1994) and those relating to Swedish children (Gare & Fasth 1992).

Osteoarthritis

While there are a few Australian population-based studies on self-reported osteoarthritis (Jones et al. 1995; March et al. 1998), we prefer to base our estimates for this condition on reported findings of radiographic osteoarthritis (grade 2 and above) by affected joint, age and sex from a large-scale study in Massachusetts, USA (Jones et al. 1995; March et al. 1998). We model hip and knee osteoarthritis only, given the high correlation between osteoarthritis of the hip, hand and fingers (Spector et al. 1997). We used DisMod to derive average durations, assuming a slightly increased risk of mortality (1.1) and the observed remission rate from joint replacement surgery. Because osteoarthritis is a relatively slow progressive disease, with few patients showing symptomatic progression over an 11-year period (Ahern

& Smith 1997), we apply an average of the relevant Dutch weights, assuming a severity distribution based on the Framingham study (Guccione et al. 1990).

Back pain

Back pain is a very common condition, with about 70–90% of people suffering from it in some form at some point in their lives (Hicks et al. 2002). Back pain may be viewed as running either an acute or chronic course. Acute back pain is usually considered to have a short duration and tends to resolve within days to weeks. However, recurrence of acute episodes is common and there is some contention as to the difference between recurring acute back pain and long-term chronic back pain. A duration of back pain lasting at least 3 months commonly underlies the definition of chronic back pain (NINDS 2006), and is often likely to continue indefinitely (Von Korff & Saunders 1996). Our estimates for back pain are based on self-reported prevalence of recent episodes, and long-term back pain from the 2003 Australian disability survey and the 1995 National health survey. We model recent episodes of (acute) back pain and long-term (chronic) back pain separately. Prevalence of long-term back pain resulting in at least mild disability is obtained from the Australian disability survey. Of these, the cases that were due to recent episodes of back pain were not identified separately. We therefore estimated the proportion due to recent episodes by applying the percentage of recent cases of long-term back pain from the 1995 National Health Survey. We use the Dutch weight for low back pain (0.06) as the disability weight for recent episodes of back pain, which applies to an average health state involving some problems in walking about and in usual activities, as well as moderate pain or discomfort. We assume an average duration of 4 days for painful and limiting episodes of back pain. To model chronic back pain, we use the prevalence of long-term back pain (not identified as recent episodes as described above) from the 2003 Australian disability survey. We use DisMod to derive the incidence and duration of chronic back pain, assuming a remission rate of 10% and no increased risk of mortality. For many people, there are few treatment alternatives and complete relief is rare (Atkinson 2004). We assume that 14% of long-term cases experience constant or persistent pain (Quittan 2002), and 86% experience pain 1 day per week. We use the GBD disability weight for chronic intervertebral disc pain of 0.103.

Slipped disc

Our estimates for slipped disc are based on numbers of intervertebral disc procedures from Australian hospital data. We assume only 7.5% of incident cases of disc displacement receive surgery (Deyo et al. 1990), and derive total annual episodes from this proportion. We assume on average an episode of discomfort lasts 4 weeks. For those who receive surgery, we take the median time of 224 days from onset of symptoms to recovery reported in the literature (Rasmussen 1996). In the absence of weights for both these health states, we use the Dutch weight for low back pain (0.06). Based on a 5-year follow-up study (Kurth et al. 1996), we model 14% of operated cases as going on to experience long-term chronic pain with a lifelong duration at the GBD disability weight for chronic intervertebral disc of 0.103.

Occupational overuse syndrome

Occupational overuse syndrome (formerly known as repetition strain injury) is a contentious condition with considerable disagreement within the literature about its aetiology and

pathophysiology (Byrne 1992; Cohen et al. 1992; Helme et al. 1992). Our model uses self-report prevalence data on 'repetition strain injury' from the 2003 Australian disability survey from which we derive incidence figures using DisMod assuming an average duration of 3 years and no mortality. In the absence of Dutch or GBD weights for this condition, we use sex-specific derived weights to account for the fact that all males in the 1993 Australian disability survey had mild or no handicap, whereas 26% of females had moderate handicap and 17% had severe or profound handicap.

Gout

Our estimates for gout are based on self-reported prevalence from the National Health Survey which has the same overall result as found in a general practitioner study in the UK (Mikuls et al. 2005). We assume a slight increased risk of mortality associated with gout (relative risk=1.1) and no remission, based on information that at 1 year 62%, at 2 years 78% and at 10 years 93% has had at least one repeat attack (Alamo Family Foot and Ankle Care 2005). Fitting a Weibull function to these figures gives an average time to the next episode of 2.2 years, but this is rather high because of the skewness of the function. The median time to next episode is 0.44 years. We assume that 10% has chronic symptoms and the remaining 90% has an attack of 1 week every 0.44 years. Given that people may suffer gout at varying levels, from acute attacks of a short duration to chronic gout, we assume on average one attack per 2 months lasting 1 week in 90% of people and the remaining 10% suffer chronic ongoing disease at the GBD disability weight of 0.061.

Other musculoskeletal disorders

Because mortality for musculoskeletal conditions is low and because 49% of deaths from musculoskeletal disorders do not fall within the above categories, a derivation of disability for this rest category by applying a ratio of YLD to YLL for the explicitly modelled musculoskeletal conditions is not plausible. Therefore we try to model disability from all other conditions explicitly. In the absence of detailed information, we define an 'other' category comprising both prevalent minor conditions and more serious diseases (for example joint derangement and disorders; osteopathies; chondropathies and other bone disorders; connective tissue diseases; and soft tissue problems such as rheumatism, ganglions, bunions, bursitis, cramps, tenosynovitis and tennis elbow). We base our estimates for these conditions on the prevalence of other musculoskeletal disorders that have not been accounted for in each of the musculoskeletal models described above from the 2001 National Health Survey. Based on figures from the 2003 Australian disability survey, we assume a proportion of prevalent cases report on refer to musculoskeletal sequelae of other diseases or injuries, which we account for by adjusting overall prevalence figures downwards by 50%.

For recent non-chronic cases, we assume the same duration and weight as for recent episodes of back pain. For chronic cases, we derive incidence rates and durations from DisMod assuming no excess mortality and a remission rate of 0.1. We take the proportion reporting symptoms in the 2 weeks before interview as an approximation of the proportion of time spent symptomatic and assume symptomatic chronic cases experience a health state equivalent to the weight for low back pain.

2R Congenital anomalies

Congenital heart disease

We model the disability associated with four types of congenital heart disease for live-born infants: surgically treated atrial or ventricular septal defect, surgically treated Fallot's tetralogy or transposition of great vessels, surgically treated pulmonary stenosis, and complex but not curatively operable congenital heart disease. We derive the incidence of the first three conditions from Australian hospital data by assuming that all curative procedures represent an incident case with disability. We assume a duration of 1 year before operation with disability equivalent to the Dutch weight for moderate heart failure (0.35) and post-surgery we use relevant Dutch weights and assume reduced life expectancy, except for those with septal defects (Miyamura et al. 1993; Nollert et al. 1997a, 1997b). We assume disability starts at birth and we discount YLD back to birth to account for this. We derive the incidence of other congenital heart malformations from Victorian birth defects data. Following expert advice, we assume that 50% of these cases are complex but not curatively operable. We assume that duration is half of those with surgically treatable conditions and use the relevant Dutch weight (0.72).

Digestive system malformations

We model the disability for anorectal and oesophageal atresia and other digestive system malformations. We estimate the incidence of digestive system atresia for cases surviving 28 days using Victorian birth defects data. We assume 26 weeks of disability from birth at the GBD weight for anorectal atresia (0.85). After this period, we assume that a proportion of both types of atresia cases have lifelong problems (15% and 20% respectively) and decreased life expectancy (by 10 and 5 years respectively) and disability equivalent to health state 111211 for two-thirds of the time (0.037) (Ludman & Spitz 2003). We estimate the incidence of other digestive system malformations using data from the Australian congenital malformations dataset (AIHW: Hurst et al. 2001). We assume no long-term disability, and a 1-month period of disability from birth equivalent to the GBD weight for anorectal atresia.

Renal agenesis

We estimate the incidence of unilateral and bilateral renal agenesis for cases surviving 28 days using Victorian birth defects data. For unilateral cases we assume that 20% of survivors have ongoing problems, with a life expectancy of 70 years and a disability of 0.067. For bilateral cases we assume an average duration of 3.5 days and use the GBD weight for renal agenesis (0.85). We also calculate YLD for renal failure due to renal dysplasia based on attributions from Australian dialysis and transplant data.

Other urogenital tract malformations

We model the disability associated with the following urogenital tract malformations: cystic kidney disease, obstructive defects of renal pelvis and ureter, and other urinary tract malformations. We estimate the incidence of cases of other urogenital tract malformations surviving beyond 28 days from the Victorian, Western Australian and Australian birth

defects data. We assume 30% of cases have chronic lifelong problems, with a life expectancy of 50 years and a disability weight of 0.067. YLD were also calculated for end-stage renal failure due to cystic kidney disease.

Other congenital anomalies

We estimate the incidence of anencephaly using Australian mortality data for newborns, assuming deaths are equivalent to incident cases. We assume a duration of 1 week with a disability weight of 1. For spina bifida, we estimate the average annual number of live births that survive the first 28 days from Victorian birth defect data (Riley & Halliday 2004). We derive an average disability weight (0.52) based on the Dutch weights for each level of severity combined with severity distributions from expert advice. We estimate the incidence of surgically treated cleft lip and cleft palate from Australian hospital data, assuming that all curative procedures represent a case and that all cases are treated within the first year. We assume disability equivalent to the 'treated' GBD weights (0.016, 0.015 respectively). YLD estimates for Down syndrome and 'other chromosomal anomalies' are calculated as described in the section on intellectual disability (see Section 2K).

We estimate the incidence of abdominal wall defects (exomphalos and gastroschisis) in infants surviving >28 days using 2001 Australian birth defects data (AIHW NPSU 2004) and survival data from the Victorian birth defects data. We assume a duration of 4 weeks based on Australian and international literature (Dimitriou et al. 2000; Sharp et al. 2000) and apply the GBD weight for abdominal wall defect. Based on expert advice we assume that 20% of cases have lifelong problems, a shortened life expectancy by 20 years, and disability weight of 0.200 (the Dutch weight for young adult in permanent stage after surgical repair to Fallot's tetralogy).

2S Oral conditions

Caries

The incidence of caries is measured by one or more new dental cavities (caries increment). The occurrence of dental caries in an individual is measured using the DMFT or DMFS index: the number of decayed (D), missing (M) and filled (F) primary or permanent teeth (T) or surfaces (S). A review of the relationship between DMFT and DMFS suggests that DMFS data should be adjusted by a factor of 1/3.5 to be consistent with DMFT data (Carvalho et al. 2004; Hopcraft & Morgan 2005; Rosen et al. 2004).

For children and adults we estimate the incidence from representative Australian caries prevalence data: the 2000 Child Dental Health Survey (AIHW: Armfield et al. 2004) and the 1987–88 National Oral Health Survey of Australia (Barnard 1993). Fitting linear regression lines to the prevalence data gives slopes in children (1–14 years) of 0.25 (AIHW: Armfield et al. 2004; Davies et al. 1997) and in adults (15–59 years) of 0.27 (Barnard 1993). For older adults (60 years or over) and nursing home residents (60 years or over) we estimate the incidence of caries from the South Australia Dental Longitudinal Study (AIHW DSRU 2002) and the 1998 Adelaide Dental Study of Nursing Homes (AIHW: Chalmers et al. 2001), respectively. Based on the 5-year increment of all new carious surfaces, the 1-year increment (assuming that the incidence of carious surfaces over the 5-year period was evenly

distributed) is 0.98 (AIHW DSRU 2002). The 1-year increment of new carious surfaces in nursing home residents is 3.5 (AIHW: Chalmers et al. 2001). We use our DMFT/DMFS adjustment factor to give annual caries increments of 0.28 and 1.0 respectively for older adults in the general population and nursing homes.

The previous Australian burden study assumed a symptomatic duration of 10 weeks based on advice from the Australian Research Centre for Population Oral Health. More recent work, based on patient self-report, by this group suggests durations in the order of 81 weeks (Brennan & Spencer 2004, 2005). However, both of these estimates refer to time spent with and without symptoms. A review of the literature shows that there is a paucity of information on symptomatic caries, specifically mean duration of symptoms and proportion of people who are symptomatic. A patient-based study in children in the UK reported that 78% of the children sampled presented within 1 month of pain onset (Mason et al. 1997) whereas a patient-based study in New Zealand observed that 67% of adults presented within 1 month of pain onset (Whyman et al. 1996). Patient-based samples are biased as they do not reflect all cases of caries in the community. Neither of these studies provided data on the mean durations for those people experiencing symptoms for greater than 1 month. We estimate the average time symptomatic for those people presenting with caries problems by fitting a lognormal distribution to the midpoint of the observed durations. This gives mean durations of symptomatic caries of 28 days in children and 55 days in adults. We base our estimate of people with symptomatic caries (32.4%) on the findings of the 1998 Australian Longitudinal Study of Dentists' Practice Activity (Brennan & Spencer 2002).

Following the first Australian burden of disease study the Australian Research Centre for Population Oral Health developed disability weights for oral disease using a patient-based sample in South Australia (Brennan & Spencer 2004, 2005). Disability weights for caries (0.044), periodontal disease (0.023) and denture problems (0.026) in this study were higher than comparable Dutch weights used in the previous Australian burden study (0.005 for caries involving a filling and 0.014 for caries involving an extraction, 0.007 for periodontal disease, and 0.004 for edentulism). We did not use these Australian-derived disability weights because patient-based samples are likely to under-represent asymptomatic people, and questions with limited response categories are likely to bias results. For instance, the duration-related question was 'During the period that you have had this dental problem, what percentage of the time (0% = none of the time, 50% = half of the time, 100% = all of the time) have you experienced the limitations listed above in relation to: mobility, self-care, usual activities, pain/discomfort, anxiety/depression, cognition?' (Brennan & Spencer 2004, 2005). Both of these limitations are likely to over-estimate the percentage of people reporting problems for each of the health dimensions as well as the duration of their symptoms.

We follow expert advice and derive a disability weight for symptomatic caries (0.057) using the disability weight regression model (health states: 20% – 111211 and 80% – 111111).

Edentulism

We estimate the prevalence of edentulism (loss of all natural teeth) for the general population and nursing home residents using the 2002 National Dental Telephone Interview Survey (AIHW: Carter & Stewart 2003) and the 1998 Adelaide Dental Study of Nursing Homes (AIHW: Chalmers et al. 2001), respectively. We derive incidence and duration using DisMod, based on these studies, assuming no remission and no excess case-fatality. We model a 2% declining time trend to reflect the observed decline of the prevalence of

edentulism from 20.5% in 1979 to 8.0% in 2002 (Sanders et al. 2004). We use the same disability weight as in the previous Australian burden study.

Periodontal disease

We estimate the prevalence of periodontal pockets larger than 6 mm using data from the 1987–88 National Oral Health Survey of Australia (Barnard 1993). Expert advice suggested that periodontal disease is a largely asymptomatic risk factor for tooth loss; pain occurs in around 1% of time when an abscess forms in a periodontal pocket; and the typical duration of periodontal disease is around 15 years. We derive incidence and duration using DisMod based on the Australian prevalence data, remission rates that reflect 15 years average duration and no case-fatality. A new disability weight for periodontal abscess was estimated (0.056) based on the disability weight regression model (health state 111211).

Pulpitis

We estimate the incidence of pulpitis using the proportion of patients sampled in the 1998 Longitudinal Study of Dentists' Practice Activity (AIHW: Spencer & Brennan 2002) who had a main diagnosis of pulpal infection and the total number of dental consultations in Australia in 2003. We assume that most people with pulpal infection will visit a dentist. We estimate the total number of dental consultations by multiplying the proportion of people who visited a dentist in the last 12 months by the mean number of dental visits per person (from the 2002 National Dental Telephone Information Survey (AIHW: Carter & Stewart 2003)) and 2003 population data (excluding the edentulous population). Expert consultation suggests that a symptomatic duration of 1 month is plausible for pulpitis with the first few weeks consisting of intermittent pain and the last week being of more severe and consistent pain. We assume that 71.3% of people with pulpitis presenting in pain, a figure which we derive from the 1998 Longitudinal Study of Dentists' Practice Activity. We use the disability weight regression model to estimate a disability weight for pulpitis assuming that 1 week is spent in moderate pain and 3 weeks are spent at a level of the disability for moderate pain for 10% of the time.

2Z Chronic fatigue syndrome

We base our model for chronic fatigue syndrome on the internationally accepted US Centers for Disease Control and Prevention criteria, which state that for a patient to receive a diagnosis of chronic fatigue syndrome, they must have severe chronic fatigue of 6 months or longer duration with other known medical conditions excluded by clinical diagnosis, and concurrently have four or more of the following symptoms: substantial impairment in short-term memory or concentration; sore throat; tender lymph nodes; muscle pain; multi-joint pain without swelling or redness; headaches of a new type, pattern or severity; unrefreshing sleep; and post-exertional malaise lasting more than 24 hours. The symptoms must have persisted or recurred during six or more consecutive months of illness and must not have predated the fatigue (Fukuda et al. 1994).

Following expert consultation we conceptualise two manifestations of chronic fatigue syndrome: (a) post-infective fatigue syndrome which constitutes between 30–40% of cases and is characterised as an acute outcome of viral and non-viral infections, has a disability starting point of moderate severity, a median duration of 12 months, and around 99%

recovery at 2 years (Hickie et al. submitted 2005; Wilson et al. 2001); and (b) protracted chronic fatigue syndrome, which constitutes the remaining 60–70% of chronic fatigue syndrome cases, where cases have an insidious onset with initially severe disability followed by cases fluctuating around 50–80% of their previous healthy state, and a median duration of around 7 years. We assume that the disability associated with post-infective fatigue syndrome is included within the disability weights and durations in the relevant infectious disease models (explicitly in the arbovirus estimates but not for other viral infections such as Q fever and Epstein-Barr virus which are subsumed in the rest of infectious disease category).

We base our estimates of prevalence for protracted chronic fatigue syndrome on the population-based study of chronic fatigue syndrome conducted in Wichita, Kansas, USA in 1997 (Reyes et al. 2003). In the previous Australian burden study we used prevalence estimates based on an Australian prevalence study of chronic fatigue syndrome (Lloyd et al. 1990). This study's applicability in the current context is limited due to the different diagnostic criteria used and the physician referral sample. The population-based study by Reyes and colleagues (2003) showed that only 16% of people identified with chronic fatigue syndrome had previously been diagnosed as such by a medical practitioner. Although it is not clear how similar the epidemiology of chronic fatigue syndrome is between the US and Australia, the findings from an international multi-centre study of the prevalence of chronic fatigue syndrome in patients lend support to the notion that the epidemiology of chronic fatigue syndrome is similar in the two countries (Wilson et al. 2001).

We model incidence and duration using DisMod, assuming no excess mortality and remission rates which gave an average duration of 7.3 years (Reyes et al. 2003). We assume that 90% of the time people with chronic fatigue syndrome are symptomatic, using findings from the 1993 Australian disability survey. In the absence of an established disability weight for chronic fatigue syndrome we use the disability weight estimated for the previous Australian burden study.

3 Injuries

We model the disability from non-fatal injuries where a person has an injury severe enough to warrant emergency department or inpatient hospital treatment but that does not lead to death. This method assumes that injuries treated outside the hospital system do not result in significant disability. We derive non-fatal incident injuries from Australian hospital data. We classify incident cases according to a matrix of 14 'external cause of injury' categories (12 unintentional and two intentional) and 32 'nature of injury' categories (for example fractures, burns, wounds, brain injury, spinal cord injury). We exclude admissions for the same ICD-10 code within 90 days, on the assumption that these are re-admissions, as well as, those resulting in death. Given that it is not uncommon for multiple sites of the body to be damaged from a single accident, we estimate disability for only the most disabling ICD-10 code associated with each incident, on the assumption that the disability for the other ICD-10 codes is captured in the weight for the more severe injury. We redistribute ill defined injuries and adjust estimates for 'amputated finger' as in the previous Australian burden of disease study. We use disability weights, durations and the risk of mortality as per the GBD study.