



WORKING FOR A HEALTHY FUTURE

Research Report TM/06/01
March 2006

Comparing estimated risks for air pollution with risks for other health effects

BG Miller and JF Hurley

RESEARCH CONSULTING SERVICES

Multi-disciplinary specialists in Occupational and Environmental Health and Hygiene

www.iom-world.org



WORLD HEALTH ORGANISATION
COLLABORATING CENTRE
FOR OCCUPATIONAL HEALTH

Comparing estimated risks for air pollution with risks for other health effects

BG Miller and JF Hurley

It is now widely accepted that air pollution has important effects on mortality, over timescales at least long enough to impact on annual mortality rates. Given estimates of the size of these effects, it is possible to predict changes to mortality rates that might accrue following proposed reductions in pollution concentrations.

Changes in mortality rates imply changes in survival distributions, and these can be estimated using standard life-table calculations. When estimating for a whole population, it is necessary to separate the dimensions of age and calendar year, and we have developed a system of spreadsheets, IOMLIFET, to carry out and summarise the detailed calculations required. The system permits great flexibility in input assumptions and output summaries, including monetary values with or without discounting. It can be applied to changes in mortality from any cause, not only air pollution.

It is often helpful to compare the impacts of different kinds of health effects. This report investigates aspects of comparability of effects in individual birth cohorts and in mixed-age populations, based on cause-specific mortality rates for England and Wales. The predicted effects of a $10 \mu\text{g.m}^{-3}$ reduction in airborne $\text{PM}_{2.5}$ air pollution (broadly equivalent to removing all anthropogenic particles) from US cohort studies are compared with the effects of eliminating the mortality risks of passive smoking and of motor vehicle traffic accidents (MVTA).

For a single birth cohort, the impacts of eliminating passive smoking or MVTA are roughly similar in males, around 12 weeks' additional expectation of life. In females, they yield 2 months and one month respectively. In both sexes, a $10 \mu\text{g.m}^{-3}$ reduction in airborne $\text{PM}_{2.5}$ air pollution is predicted to gain some seven months' expectation of life. While these estimates are subject to uncertainty, they show that the effect of ambient air pollution on mortality is a public health issue of substantial importance. We expect similar results would be obtainable in other countries.

CONTENTS

CONTENTS	iii
1 INTRODUCTION	1
2 OBJECTIVES	3
3 METHODOLOGY	5
3.1 Life-tables	5
3.2 Impact assessments	11
3.3 Compound populations and complex scenarios	21
3.4 One-year pulses	24
3.5 Counting deaths	30
3.6 Comparing the impacts of different effects	31
4 RESULTS	33
4.1 Particulate air pollution	33
4.2 Motor Vehicle Traffic Accidents	33
4.3 Passive smoking	34
4.4 Summary of comparisons	35
5 DISCUSSION	37
5.1 Methodology for health impact assessment	37
5.2 Comparisons of impacts	38
6 REFERENCES	41

1 INTRODUCTION

Quantitative assessment of the health impacts of air pollution, and of the associated costs, has attained considerable importance in recent years, and forms a branch of health impact assessment (HIA). This is a discipline whose principal purpose is to support policy development by predicting into the unknown (unknowable) future the effects on health of current or planned policies. To do this well in the context of air pollution requires, amongst other things:

- reliable estimates of the strength of relationships between air pollution and health effects;
- assumptions regarding the distributions and characteristics of the population for whom predictions are constructed, including background levels of mortality and/or morbidity in that population;
- assumptions regarding future changes in pollution concentrations that would affect this population;
- a consistent methodology for calculating and summarising the predicted effects on mortality and morbidity of these changes in pollution, given all the above assumptions.

To date, the most comprehensive information about strength of relationships between mortality hazards (i.e. the age-specific risks of dying, conditional on having survived to that age) and long-term exposure to ambient air pollution has been available from a limited number of US cohort studies, and in particular from analyses of the American cancer society (ACS) cohort. Extensive analyses of mortality data from these studies, compared across cities, have identified relationships with levels of ambient air pollution, and especially with particulate air pollution characterised as PM_{2.5}, both for mortality generally and particularly for causes of death classified as cardiovascular or respiratory (Pope *et al*, 1995; Krewski *et al*, 2000; Pope *et al*, 2002). It is now widely recognised that risk coefficients from the ACS study are the best available for use in quantitative health impact assessment of the mortality effects of long-term exposure to ambient particles (e.g. WHO, 2006; COMEAP, 2006).

Bruneekreef (1997) used life table methods to derive the implications for life expectancy of the Dutch population of long-term exposure to ambient particles. He used a relatively simple framework which nevertheless highlighted the public health importance of ambient air pollution. The use of life table methods in the context of ambient air pollution HIA was developed and expanded considerably in work at the IOM for the European Commission's ExternE project (e.g. ExternE, 1998) and as part of a DoH-funded project about the effects of ambient particles in Britain, with a final report by Hurley *et al* (2000). The methodology for quantitative predictions, based on actuarial life-table methods, was summarised also in the report of a WHO workshop on HIA of air pollution (WHO, 2001). More recent developments at the IOM, also funded by DoH, included (i) catering for cause-specific impacts, triggered by the epidemiological observation that, in the above cohort studies, the impact of air pollution was not the same for all causes of death; and (ii) implementation of the methodology for calculation through standard spreadsheet techniques (Miller and Hurley, 2003).

There remain a number of unanswered questions that may be considered important in quantitative impact assessment, such as:

- 1) Assuming deaths are advanced by exposure to air pollution, how large is that advance, on average?
- 2) How does the amount advanced vary in the population, and what determines the variation?

- 3) What uncertainties are introduced in transferring US results for use in the UK?
- 4) How do the risks implied by exposure to air pollution compare with risks identified from other causes, e.g. road accidents or passive smoking?
- 5) To what extent is it possible and meaningful to express the mortality effects of air pollution in terms of 'attributable deaths' rather than changes in life expectancy?
- 6) Is it practicable and useful to express the effects of a sustained change in air pollution as the cumulative effect of a series of one-year 'pulse' changes in pollution?

Item 4) is needed, particularly for non-expert discussion, but has been considered difficult because risks can be and are expressed and summarised in different ways, making comparisons harder. However, if data on risks (or relative risks) from a range of explanatory factors can be found in a suitable form, they can be brought to a common base for comparison, using the relationships between hazard and survival already used in the quantification work at the IOM.

The work reported here was initiated primarily to make comparisons of the magnitude of risks from different sources, through the spreadsheet systems developed at IOM (Miller and Hurley, 2003), and recently designated IOMLIFET. The risks chosen for detailed comparison with air pollution risks were death from motor vehicle traffic accidents and risks from passive smoking; for both of these there was sufficient quantitative information in the public domain to create input to the IOMLIFET spreadsheets.

The report includes some detail regarding the performance of the method and the interpretation of its output, before moving on to the comparisons themselves.

2 OBJECTIVES

The main aim of the work reported here was to construct quantitative summaries to compare, on an equal basis, the risks of:

1. death associated with particulate air pollution;
2. death associated with passive smoking;
3. death from motor vehicle transport accident.

In order to give context for the results, we have tackled and we report results on a number of the current methodological issues noted earlier.

3 METHODOLOGY

3.1 LIFE-TABLES

Traditionally, life-tables have been a convenient way to calculate expectations of life, and patterns of predicted deaths, from published or other mortality rate data. Essentially, they are a way of tabulating mortality rates that facilitates summarising these into a survival curve.

3.1.1 Basic methodology

Table 3.1 shows the layout for a typical life-table calculation. The columns containing the mid-year population estimate and the number of deaths (by convention, excluding neonatal) are based on published data for males, in England and Wales, for the year 1999. Note that the figures for ages 90 and over are given pooled. Similar data are available for males and females for each year, with some publication delay. In addition, it is possible to obtain similar data but with a breakdown of deaths attributed to different groups of causes.

Table 3.1: Life-table calculations, using all-cause hazard rates for males, England and Wales, 1999.

Age	Mid-year population	Deaths	All-cause hazard rate	Survival probability	Cumulative survival	Deaths per 100,000 (of original cohort)
	m_i	d_i	h_i	S_{i+1}	c_{i+1}	d_i
0	322,086	688	0.002136	0.997866	0.997866	213
1	325,212	171	0.000526	0.999474	0.997342	52
2	334,966	106	0.000316	0.999684	0.997026	32
3	328,082	68	0.000207	0.999793	0.996819	21
4	334,852	60	0.000179	0.999821	0.996641	18
5	347,247	36	0.000104	0.999896	0.996538	10
6	345,470	45	0.000130	0.999870	0.996408	13
7	358,437	49	0.000137	0.999863	0.996272	14
8	362,308	43	0.000119	0.999881	0.996153	12
9	355,395	45	0.000127	0.999873	0.996027	13
10	353,289	41	0.000116	0.999884	0.995912	12
11	357,765	44	0.000123	0.999877	0.995789	12
12	346,387	59	0.000170	0.999830	0.995620	17
13	343,397	54	0.000157	0.999843	0.995463	16
14	341,263	77	0.000226	0.999774	0.995238	22
15	327,547	75	0.000229	0.999771	0.995011	23
16	330,594	132	0.000399	0.999601	0.994613	40
17	330,636	202	0.000611	0.999389	0.994006	61
18	334,892	247	0.000738	0.999263	0.993273	73
19	337,643	246	0.000729	0.999272	0.992550	72
20	326,060	246	0.000754	0.999246	0.991801	75
21	304,566	237	0.000778	0.999222	0.991030	77
22	302,390	239	0.000790	0.999210	0.990247	78
23	316,959	267	0.000842	0.999158	0.989413	83
24	332,437	281	0.000845	0.999155	0.988577	84
25	346,123	288	0.000832	0.999168	0.987755	82
26	371,460	310	0.000835	0.999166	0.986931	82
27	395,479	361	0.000913	0.999088	0.986030	90
28	414,717	356	0.000858	0.999142	0.985184	85
29	413,149	379	0.000917	0.999083	0.984281	90

Age	Mid-year population	Deaths	All-cause hazard rate	Survival probability	Cumulative survival	Deaths per 100,000 (of original cohort)
	m_i	d_i	h_i	S_{i+1}	c_{i+1}	d_i
30	424,902	398	0.000937	0.999064	0.983359	92
31	425,150	398	0.000936	0.999064	0.982439	92
32	436,256	423	0.000970	0.999031	0.981487	95
33	444,194	462	0.001040	0.998960	0.980467	102
34	451,843	472	0.001045	0.998956	0.979443	102
35	450,693	501	0.001112	0.998889	0.978355	109
36	439,778	492	0.001119	0.998882	0.977261	109
37	427,289	519	0.001215	0.998786	0.976075	119
38	416,156	529	0.001271	0.998730	0.974835	124
39	395,259	571	0.001445	0.998556	0.973427	141
40	381,349	588	0.001542	0.998459	0.971928	150
41	372,169	653	0.001755	0.998247	0.970224	170
42	359,592	657	0.001827	0.998175	0.968453	177
43	345,885	683	0.001975	0.998027	0.966542	191
44	335,213	741	0.002211	0.997792	0.964408	213
45	339,283	834	0.002458	0.997545	0.962040	237
46	336,925	959	0.002846	0.997158	0.959306	273
47	329,191	987	0.002998	0.997006	0.956434	287
48	335,750	1,045	0.003112	0.996892	0.953462	297
49	343,689	1,206	0.003509	0.996497	0.950122	334
50	355,220	1,406	0.003958	0.996050	0.946369	375
51	413,868	1,862	0.004499	0.995511	0.938137	423
53	318,956	1,801	0.005647	0.994369	0.932855	528
54	313,453	1,916	0.006113	0.993906	0.927170	568
55	314,717	2,055	0.006530	0.993492	0.921136	603
56	296,998	2,178	0.007333	0.992693	0.914405	673
57	269,973	2,365	0.008760	0.991278	0.906430	798
58	247,034	2,288	0.009262	0.990781	0.898073	836
59	260,202	2,614	0.010046	0.990004	0.889097	898
60	260,320	2,967	0.011398	0.988667	0.879020	1008
61	256,625	3,175	0.012372	0.987704	0.868212	1081
62	249,429	3,433	0.013763	0.986331	0.856344	1187
63	241,893	3,671	0.015176	0.984938	0.843446	1290
64	234,843	3,943	0.016790	0.983350	0.829402	1404
65	223,966	4,325	0.019311	0.980874	0.813539	1586
66	219,327	4,694	0.021402	0.978825	0.796312	1723
67	221,679	5,285	0.023841	0.976440	0.777551	1876
68	219,698	5,704	0.025963	0.974370	0.757622	1993
69	211,354	6,218	0.029420	0.971007	0.735656	2197
70	200,569	6,593	0.032871	0.967660	0.711865	2379
71	192,826	7,174	0.037205	0.963475	0.685864	2600
72	188,211	7,663	0.040715	0.960097	0.658496	2737
73	184,053	8,237	0.044753	0.956226	0.629672	2882
74	171,823	8,403	0.048905	0.952262	0.599612	3006
75	162,922	8,969	0.055051	0.946424	0.567488	3212
76	154,384	9,274	0.060071	0.941681	0.534392	3310
77	151,575	10,113	0.066719	0.935434	0.499889	3450
78	146,589	10,772	0.073484	0.929120	0.464457	3543
79	140,950	11,033	0.078276	0.924672	0.429470	3499
80	85,976	7,633	0.088781	0.914993	0.392962	3651

Age	Mid-year population	Deaths	All-cause hazard rate	Survival probability	Cumulative survival	Deaths per 100,000 (of original cohort)
	m_i	d_i	h_i	S_{i+1}	c_{i+1}	d_i
81	74,424	7,276	0.097764	0.906792	0.356335	3663
82	75,103	8,139	0.108371	0.897199	0.319703	3663
83	71,916	8,503	0.118235	0.888364	0.284013	3569
84	65,262	8,719	0.133600	0.874766	0.248445	3557
85	56,847	8,198	0.144212	0.865487	0.215026	3342
86	48,183	7,446	0.154536	0.856548	0.184180	3085
87	39,355	6,718	0.170703	0.842721	0.155213	2897
88	32,325	6,057	0.187378	0.828673	0.128620	2659
89	25,549	5,244	0.205253	0.813851	0.104678	2394
90+	76,277	18,674	0.244818	0.781881	0.081846	2283
Total	25,984,628	262,912			74.83	91,815

The life-table calculations involve only straightforward arithmetic. The hazard rate h_i at each age i is estimated by dividing the deaths at age i by the mid-year population at that same age. Thus, the hazard rate is the risk of dying at age i , among those males who reached their i^{th} birthday.

The hazard rates can be used to derive survival probabilities. The survival probability s_{i+1} of surviving a full year, from the i^{th} birthday to the $i+1^{th}$ (conditional on first achieving the i^{th} birthday), is calculated as

$$s_{i+1} = \frac{(2 - h_i)}{(2 + h_i)}$$

The survival probabilities for individual years are then multiplied forward in chain to calculate cumulative survival probabilities c_{i+1} to the end of each year. Thus, for example, the cumulative survival to the end of the year at age 2 is $0.997866 \times 0.999474 \times 0.999684 = 0.997026$, giving the proportion of the original birth cohort expected to survive to their 3rd birthday.

By differencing the cumulative survival probabilities, we derive the unconditional probabilities d_i of dying at any age. When we think about large groups of individuals, probabilities can be interpreted as expected proportions. So, for a birth cohort of a given size, the unconditional probability d_i is the proportion of the birth cohort that is expected to die at age i . This enables us to predict the pattern of deaths over time. To aid interpretation, in Table 3.1 these are shown as deaths per 100,000 male members of the original birth cohort. As an example, the probability of dying at age two years is equal to the probability of surviving to age two minus the probability of surviving to age three: $0.997342 - 0.997026 = 0.00032$, or a rate of 32 in 100,000.

Algebraically, the cumulative survival at the end of each year is equivalent to the expected fraction of a life-year achieved in that year, because it averages over the whole cohort the predicted contributions of those who survive to and through that year and those who die in it. The total of the cumulative survivals is therefore equivalent to the expected average length of life in a population whose mortality is governed by the given set of hazard rates. The total of the cumulative survivals from the c column, 74.83 years, is the average life expectancy of any birth cohort experiencing these hazards. It is worth noting here that the size of the original

mid-year population plays no part in the life-year calculations except in providing denominators for estimating the original hazard rates. Thus the figure quoted above for the average life expectancy depends only on the hazard rates, and not on the group size.

Numerous summary aspects of the predicted mortality may be taken straight from the life-table. The total of the cumulative survivals gives the expectation of life, as noted. In addition, this column also gives estimates of the proportion of the population surviving to a given age. For example, since the cumulative survival at age 59+1 is 0.889, we expect 88.9% of males here to reach their 60th birthday. In addition, the cumulative survival values may be multiplied by a figure for the size of the birth cohort, to estimate the total life-years experienced at each age by that cohort.

Similar calculations can be done on parts of the table, e.g. to calculate expectations of future life conditional on achieving a given age. For example, to estimate the expectation of remaining life for a male reaching his 50th birthday, we need to cumulate the survival probabilities from age 50 onward and sum the cumulative probabilities. In this table we can calculate (details not shown) that a man of age 50 has an expectation of a further 28 years of life.

3.1.2 Grouped data

Within a birth cohort the total number of deaths must equal the size of the birth cohort, because each person in the cohort must die once and once only. However, the calculations as shown in Table 3.1 do not reflect accurately this necessity, because of the grouping of the 90+ age group. The cumulative survival to the end of this age is shown in Table 3.1 as just over 8%, when in a full elaboration of a life table through all possible ages of the cohort, the cumulative survival probability should ultimately be zero. However, because only grouped data are available at age 90+, the standard life table calculations omit the impact of lives entering and deaths occurring in the 91st year and thereafter. This has the effect of underestimating the life expectancy, which as noted earlier is the sum of the cumulative survivals over all ages until all the cohort has died. If we simply truncate the life-table by setting the cumulative survival at the end of year 90 to 0, the life expectancy is reduced still further.

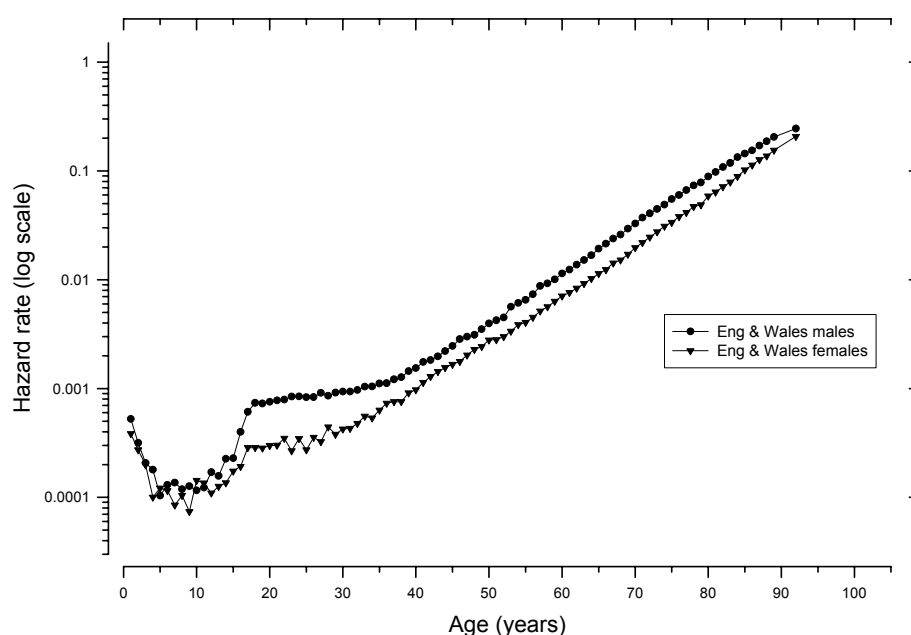


Figure 3.1: All-cause hazard rates by sex and age, England and Wales, 1999.

In order to lessen this inaccuracy, we may take some steps to extend the life-table up to some age, say 105, past which the probability of survival is sufficiently near zero to be ignored. To do this, we need to populate the ages between 90 and 105 with plausible all-cause hazard rates.

It is well established that all-cause hazard rates increase roughly exponentially in adults, as shown by the log-linear trend in Figure 3.1. It is therefore possible to extrapolate forward from the ages with known rates. By regressing the logarithms of the hazard rates on ages 70 to 89 inclusive, we can predict forward to obtain the (abbreviated) life-table in Table 3.2, which has individual hazard rates up to age 105. Performing the calculations as before, we predict a negligible probability of surviving past 105. Setting the cumulative survival after age 105 as zero, the life expectancy is now estimated as 75.05 years, which is somewhat greater than the value of 74.83 previously estimated from the grouped data, and the deaths tally with the original cohort.

Table 3.2: Life-table calculations, using all-cause hazard rates for males, England and Wales, 1999; rates for ages 90+ by extrapolation.

Age	Mid-year population	Deaths	All-cause hazard rate	Survival probability	Cumulative survival	Deaths per 100,000
	m_i	d_i	h_i	S_{i+1}	C_{i+1}	
0	322,086	688	0.002136	0.997866	0.997866	213
1	325,212	171	0.000526	0.999474	0.997342	52
2	334,966	106	0.000316	0.999684	0.997026	32
3	328,082	68	0.000207	0.999793	0.996819	21
4	334,852	60	0.000179	0.999821	0.996641	18
5	347,247	36	0.000104	0.999896	0.996538	10
6	345,470	45	0.000130	0.999870	0.996408	13
7	358,437	49	0.000137	0.999863	0.996272	14
8	362,308	43	0.000119	0.999881	0.996153	12
9	355,395	45	0.000127	0.999873	0.996027	13
10	353,289	41	0.000116	0.999884	0.995912	12
11	357,765	44	0.000123	0.999877	0.995789	12
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
88	32,325	6,057	0.187378	0.828673	0.128620	2659
89	25,549	5,244	0.205253	0.813851	0.104678	2394
90+	76,277	18,674	0.2304	0.793400	0.083051	2163
91			0.2537	0.774859	0.064353	1870
92			0.2793	0.754925	0.048582	1577
93			0.3075	0.733478	0.035634	1295
94			0.3385	0.710498	0.025318	1032
95			0.3727	0.685843	0.017364	795
96			0.4103	0.659544	0.011452	591
97			0.4517	0.631521	0.007232	422
98			0.4973	0.601730	0.004352	288
99			0.5475	0.570167	0.002481	187
100			0.6028	0.536807	0.001332	115
101			0.6637	0.501671	0.000668	66
102			0.7307	0.464826	0.000311	36
103			0.8044	0.426330	0.000132	18

Age	Mid-year population	Deaths	All-cause hazard rate	Survival probability	Cumulative survival	Deaths per 100,000
	m_i	d_i	h_i	S_{i+1}	C_{i+1}	
104			0.8857	0.386145	0.000051	8
105			0.9751	0.344493	0.000000	5
Total	25,984,628	262,912			75.05	100,000

While this is an attractive method of dealing with the grouped data, it will not necessarily be adequate in all cases; in particular, while the all-cause hazard rates allow forward prediction by regression, in the cause-specific case this is much less certain, particularly for minority causes of death, because the numbers of deaths are small and trends cannot be estimated as reliably as for all-cause mortality. This means that extrapolation may not be suitable for exercises where we seek to predict impacts only on some causes of death.

Table 3.3: Life-table calculations, using all-cause hazard rates for males, England and Wales, 1999; rates for ages 90+ constant.

Age	Mid-year population	Deaths	All-cause hazard rate	Survival probability	Cumulative survival	Deaths per 100,000
	m_i	d_i	h_i	S_{i+1}	C_{i+1}	
0	322,086	688	0.002136	0.997866	0.997866	213
1	325,212	171	0.000526	0.999474	0.997342	52
2	334,966	106	0.000316	0.999684	0.997026	32
3	328,082	68	0.000207	0.999793	0.996819	21
4	334,852	60	0.000179	0.999821	0.996641	18
5	347,247	36	0.000104	0.999896	0.996538	10
6	345,470	45	0.000130	0.999870	0.996408	13
7	358,437	49	0.000137	0.999863	0.996272	14
8	362,308	43	0.000119	0.999881	0.996153	12
9	355,395	45	0.000127	0.999873	0.996027	13
10	353,289	41	0.000116	0.999884	0.995912	12
11	357,765	44	0.000123	0.999877	0.995789	12
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
:	:	:	:	:	:	:
88	32,325	6,057	0.187378	0.828673	0.128620	2659
89	25,549	5,244	0.205253	0.813851	0.104678	2394
90+	76,277	18,674	0.244818	0.781881	0.081846	2283
91			0.244818	0.781881	0.063994	1785
92			0.244818	0.781881	0.050035	1396
93			0.244818	0.781881	0.039122	1091
94			0.244818	0.781881	0.030589	853
95			0.244818	0.781881	0.023917	667
96			0.244818	0.781881	0.018700	522
97			0.244818	0.781881	0.014621	408
98			0.244818	0.781881	0.011432	319
99			0.244818	0.781881	0.008938	249
100			0.244818	0.781881	0.006989	195

Age	Mid-year population	Deaths	All-cause hazard rate	Survival probability	Cumulative survival	Deaths per 100,000
	m_i	d_i	h_i	S_{i+1}	C_{i+1}	
101			0.244818	0.781881	0.005464	152
102			0.244818	0.781881	0.004273	119
103			0.244818	0.781881	0.003341	93
104			0.244818	0.781881	0.002612	73
105			0.244818	0.781881	0.000000	261
Total	25,984,628	262,912			75.115	100,000

Because we wished to perform cause-specific calculations, we have investigated the properties of a third method. In this, we apply the average hazard for the 90+ age group, over all the ages 90 to 105 inclusive. This is shown in Table 3.3. Here, the male life expectancy is estimated at 75.12 years.

In all cases, similar results (in terms of the effects on estimated life expectancy of different strategies for dealing with the age 90+ grouped data) were obtained for life-tables based on female hazard rates; these tables are not shown here.

Because the third method gave a close approximation, and because it can be used for cause-specific mortality impacts also, we decided to perform all our calculations on the basis of that method; that is, applying the hazard rates for ages 90+ for ages 90 to 105 inclusive. We show below (3.2.1) that, for an impact assessment typical of that used for the effect of air pollution on all-cause mortality, this choice gives satisfactorily close answers.

3.1.3 Current vs cohort life-tables

The data used in the tables so far are all from 1999. If we use them to estimate life expectancy for a cohort, then we make the strong implicit assumption that the hazard rates in the future, as the cohort ages, will be the same as those in 1999. This may not be a sensible assumption; mortality rates have been steadily falling for a long time, and we may expect that they will continue to fall as more effective treatments are found for diseases, and (hopefully) more people adopt healthier lifestyles.

The future will always remain unknown until we get there, but it is of course possible to assume future hazard rates that differ from the current. The life expectancy from such a set of assumed rates will therefore differ also; if we assume that future mortality rates will be lower, then life expectancy will increase correspondingly. UK government actuaries make this distinction, and calculate life expectancy both on current rates and on rates that they assume will reduce in line with recent trends; the two types of life-table are distinguished as “current” and “cohort” respectively.

In all our recent impact assessment work, we have taken a baseline assuming future rates that simply replicated the rates from the base data; thus our methods are based on extending current life-tables. We have shown previously (Hurley *et al*, 2000) that estimates of impacts from changes in hazards are relatively insensitive to variations in assumptions about future baseline rates.

3.2 IMPACT ASSESSMENTS

The life-table method predicts the patterns of survival, life-years lived and deaths for a cohort. Impact assessment uses the method on two different sets of hazard rates, and compares the

outputs from the two sets of calculations. Traditionally, we have designated these as representing “baseline” and “altered” scenarios, where the baseline represents a continuation of the status quo and we require to quantify the predicted effects of some proposed deviation from this baseline. Then summaries of the effects, i.e. the differences between the patterns of mortality in the baseline and altered scenarios, can be made in various ways, e.g. in terms of life-years in specific years or overall, or in the pattern of deaths, or in proportions surviving to specific ages, etc.

3.2.1 Changes to hazard rates

The essence of impact assessments is to assess the nature, size and time pattern of predicted changes in the distribution of survival (or its converse, mortality). As noted above, in general, we do this by setting a baseline with known or assumed hazard rates, and then quantifying the effect of changing these in known ways. Some efficiency gains can be made by noting that, for small changes, many of the effects are proportional to the size of the change, so that we need do full calculations only once, and can scale the results to other changes by simple proportion. This is particularly true for impacts quantified in life-years; it is less so for impacts summarised in other ways.

Table 3.4: Life-table calculations, using 1% reduction in all-cause hazard rates for males (age 30+ only), England and Wales, 1999; rates for ages 90+ constant.

Age	All-cause hazard rate	Survival probability	Cumulative survival	Deaths per 100,000
	h_i	S_{i+1}	C_{i+1}	
0	0.002136	0.997866	0.997866	213
1	0.000526	0.999474	0.997342	52
2	0.000316	0.999684	0.997026	32
3	0.000207	0.999793	0.996819	21
4	0.000179	0.999821	0.996641	18
5	0.000104	0.999896	0.996538	10
6	0.000130	0.999870	0.996408	13
7	0.000137	0.999863	0.996272	14
8	0.000119	0.999881	0.996153	12
9	0.000127	0.999873	0.996027	13
10	0.000116	0.999884	0.995912	12
11	0.000123	0.999877	0.995789	12
:	:	:	:	:
:	:	:	:	:
:	:	:	:	:
:	:	:	:	:
:	:	:	:	:
:	:	:	:	:
:	:	:	:	:
:	:	:	:	:
88	0.185504	0.830241	0.131271	2684
89	0.203200	0.815541	0.107057	2421
90+	0.242370	0.783827	0.083914	2314
91	0.242370	0.783827	0.065774	1814
92	0.242370	0.783827	0.051555	1422
93	0.242370	0.783827	0.040410	1114
94	0.242370	0.783827	0.031675	874
95	0.242370	0.783827	0.024828	685
96	0.242370	0.783827	0.019461	537
97	0.242370	0.783827	0.015254	421
98	0.242370	0.783827	0.011956	330
99	0.242370	0.783827	0.009372	258

Age	All-cause hazard rate	Survival probability	Cumulative survival	Deaths per 100,000
	h_i	S_{i+1}	c_{i+1}	
100	0.242370	0.783827	0.007346	203
101	0.242370	0.783827	0.005758	159
102	0.242370	0.783827	0.004513	124
103	0.242370	0.783827	0.003537	98
104	0.242370	0.783827	0.002773	76
105	0.242370	0.783827	0.000000	277
Total			75.214	100,000

In the context of impacts of long-term exposure air pollution on mortality, data on the effects of pollution come from cohort studies of adults, and are not directly informative about effects on children. It has therefore become conventional to consider the effects of changes in hazard rates only in adults. As an example, we may consider the effect of reducing adult hazard rates by 1%. We take the male hazard rates in Table 3.3, and multiply all the all-cause hazard rates at ages 30 and above by 0.99, leaving all those for ages below 30 unchanged. Then we have a new life-table, as in Table 3.4.

The life expectancy for this altered scenario is 75.214, while that for the baseline was 75.115. This implies that the gain from reducing the all-cause hazards at ages 30 and above by 1% has improved life expectancy by 0.1 years, or about one month.

For illustration, we have carried out similar impact assessments on the life-tables where the hazard rates for ages above 90 have been treated by all three methods discussed in 2.1.2: ignored (Version 1), extrapolated (Version 2) and assumed constant (Version 3). Table 3.5 shows the results of these calculations, for both males and females, and the results are instructive. We see that the different versions predict different total life expectancies for both males and females, and that Versions 2 and 3 are fairly close together and somewhat larger than Version 1. Females are predicted to live over 4 years longer than males, whichever Version we use. However, the expected days gained, for a 1% reduction in hazard rates at ages 30 and above, are similar in all three versions and are essentially the same for males and females for Versions 2 and 3.

Leksell and Rabl (2001) have shown that, given a set of hazards that are log-linear in age, the gain in life expectancy from a small proportional change in the hazards is insensitive to the level of the original hazards, and we have observed this empirically in many assessments (Miller, 2003). The very similar results by gender for Versions 2 and 3 are typical of our findings, and illustrate a very similar predicted impact, even where the underlying hazards differ quite significantly. Here, it also seems that the incomplete follow-up in Version 1 leads to some difference in the predicted gain, presumably because the truncated follow-up has proportionally bigger impact for the longer-lived females, but that both Versions 2 and 3 eliminate this. The results in Table 3.5 give a measure of reassurance that our preferred choice of Version 3 for impact assessments is unlikely to distort the predicted gains in life expectancy.

Table 3.5: Comparison of life-table results, using all-cause hazard rates for males, England and Wales, 1999, with different assumptions about ages 90+.

Version 1: Stop at age 90			
	Life expectancy (yr)		
	Baseline	Altered	Days gained
Male	74.831	74.920	32
Female	79.116	79.193	28
Version 2: Hazard rates extrapolated for ages 90 – 105			
	Life expectancy (yr)		
	Baseline	Altered	Days gained
Male	75.052	75.148	35
Female	79.736	79.834	36
Version 3: Hazard rates constant for ages 90 – 105			
	Life expectancy (yr)		
	Baseline	Altered	Days gained
Male	75.115	75.214	36
Female	79.846	79.943	35

3.2.2 Comparing life-tables

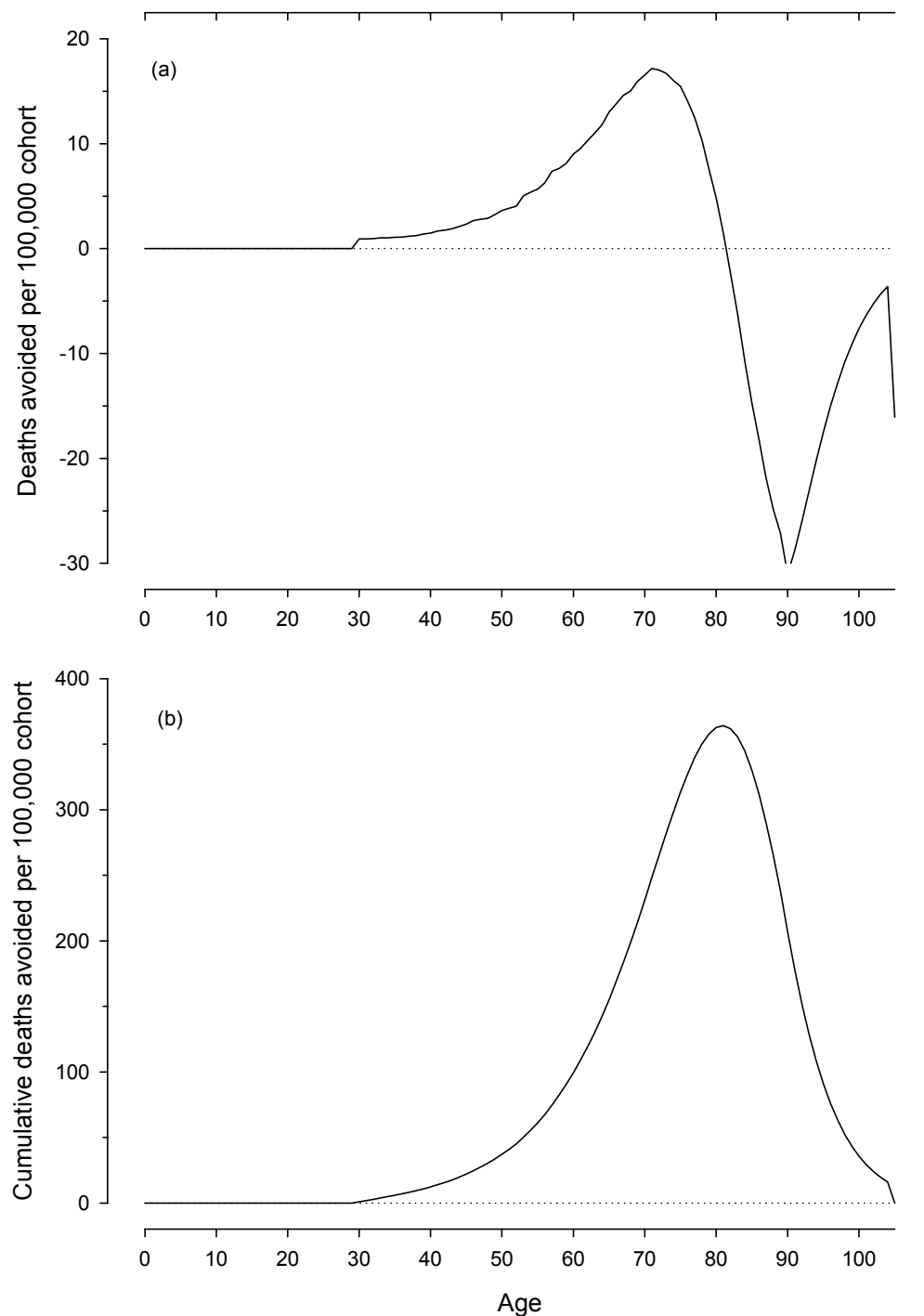


Figure 3.2: (a) Deaths avoided by 1% reduction in hazard rates, by age; (b) Cumulative deaths avoided

So far we have compared the results of life-tables, such as might be used to quantify an impact assessment, in terms only of total life expectancy. It is useful in addition to examine how this total impact is distributed over the lifetime of the cohort. This can be helped by graphical display. At each age, we can compare the number of deaths in Table 3.3 with that in the full version of Table 3.4, for the baseline and altered scenarios respectively. Figure 3.2(a) shows the pattern of these differences by age; here, the positive direction is for lives “saved” (or deaths avoided) in the altered scenario with the reduced adult hazard rates. There

is no difference between the scenarios before age 30, from which point the altered scenario begins to return fewer deaths, the difference peaking at around age 72. However, lower death rates at younger ages imply that more individuals live longer, and so at ages above 72 the larger surviving population starts to reduce the difference, until, after age 83, the reduced-hazard scenario is producing more deaths than the baseline.

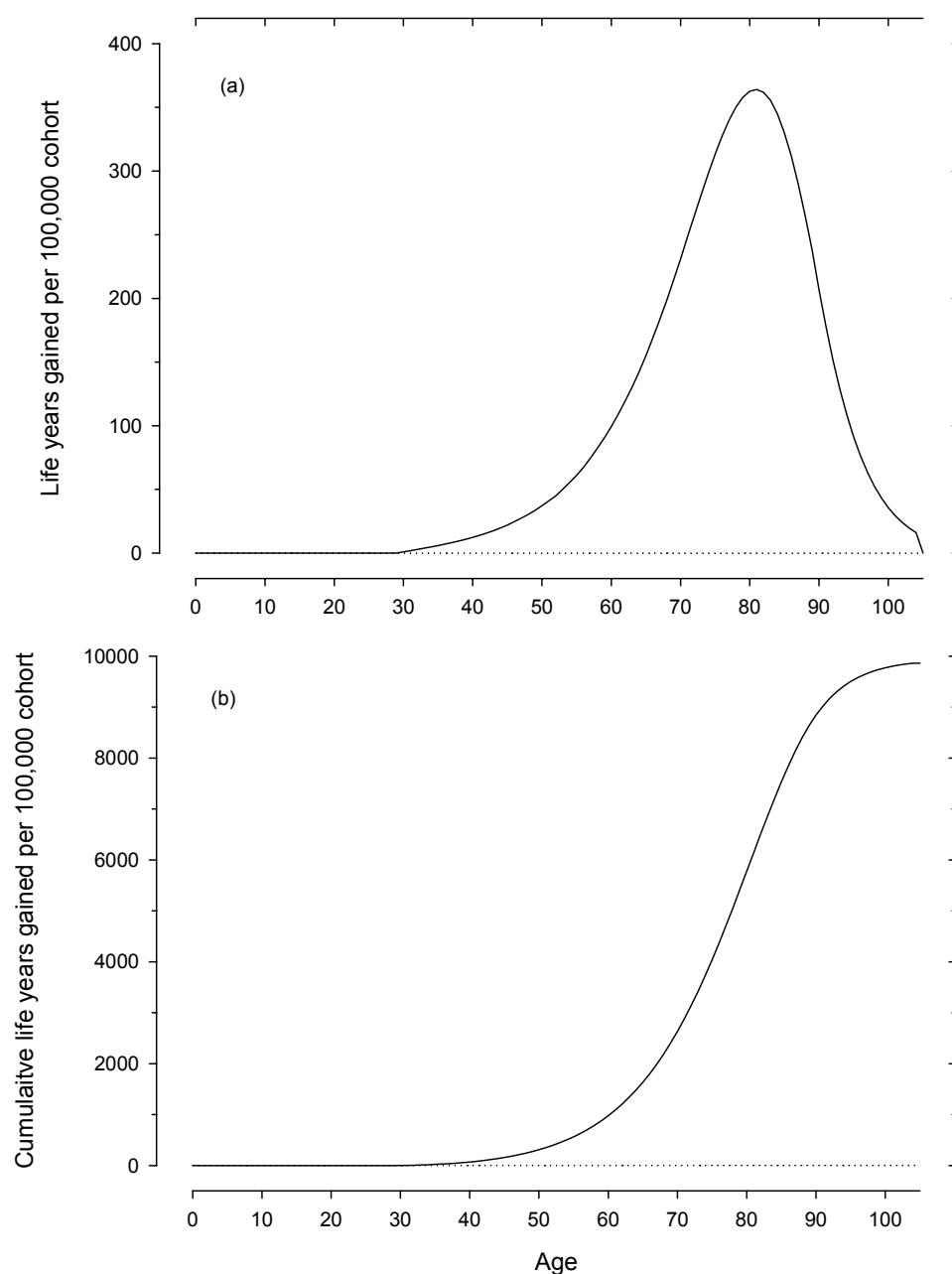


Figure 3.3: (a) Life-years gained by 1% reduction in hazard rates, by age; (b) Cumulative life-years gained.

The total number of deaths in either scenario must equal the size of the starting population, so any difference in numbers of deaths at one age must be cancelled out by other ages, and the net effect on total numbers of deaths must be zero. This is shown by Figure 3.2b, which shows the cumulative difference in the numbers of deaths increasing until age 83, then falling away to return to zero.

The picture is different when we consider life expectancy and the total number of years lived under the two scenarios. Figure 3.3(a) shows the gains in life-years at each age by reducing the hazard rates, and these are all positive. The cumulative total of these is shown in Figure 3.3(b).

Incidentally, it is no accident that Figures 2(b) and 3(a) look similar: they are in fact identical. Because the probability of death is got by differencing the cumulative survival, and because the life-years at any age equal the cumulative survival, the age-specific difference in life-years is functionally identical with the cumulative difference in number of deaths, so the graphs are the same. This is equivalent to noting that the life-years saved between baseline and altered scenarios is equivalent to the difference between the cumulative survival curves. Another aspect of this equivalence is that life expectancy, expressed as years (i.e. life-years) is exactly the same as average (predicted) age at death. The 1st and 3rd quartiles of this cumulative distribution are at about ages 71 and 83 years, implying that 50% of the total life years gained in this example are gained between these ages.

3.2.3 Insensitivity to base rates

As we have developed the methods shown here, we have noted on a number of occasions that the gains in life-years from a scenario with impacted mortality hazard rates are much more sensitive to the size and pattern of the impact factors f than to the baseline hazard rates h .

Leksell and Rabl (2001) have demonstrated a theoretical basis for this finding, based on specific distributional assumptions about the relationship between hazard and age, based on the Gompertz law in which hazard rates increase exponentially with age. An empirical demonstration is found in the results of Table 3.5, in which the gains for a 1% reduction in all-cause hazards are almost identical across the sexes, despite quite different hazard rates and a difference of several years in life expectancy.

Table 3.6 shows some additional empirical results for cohort life-tables, applying a 1% reduction in different sets of baseline hazard rates. Baseline 1 is the same baseline as before, projecting the 1999 hazard rates for each sex into each future year. Baselines 2 and 3 represent constant reductions by 10% and 30% in all the hazard rates. Baseline 4 represents a scenario in which there are future improvements in mortality rates, at a rate of 0.1% per annum; the hazard rate for age 0 is taken from current data, but future years are reduced progressively, until the hazard rate for age 105, at a point 105 years in the future, is around 10% lower than in that age group today. Baseline 5 models a faster rate of health improvement, with a reduction in hazard rates of 0.5% per annum (similar to that currently assumed by Government actuaries). This assumption would yield a hazard at age 105 reduced by 40%.

Table 3.6 demonstrates clearly that the gains in life expectancy from a 1% reduction in hazards are almost constant, at a little over a month, regardless of the size of the baseline hazards; and that this is essentially the case even when, as in Baselines 4 and 5, the underlying shape of the baseline of hazard against age is altered. The most extreme difference, between Baselines 1 and 5, is less than 10%. This implies that, while it is useful for plausibility to have up-to-date and relevant baseline rates, our results are not going to be sensitive to the choice of baseline rates.

Table 3.6: Comparison of life-table results for a 1% reduction in baseline rates, for different sets of baseline rates, all of them derived from all-cause hazard rates for males and for females, England and Wales, 1999.

Baseline 1: Standard baseline rates			
	Life expectancy (yr)		Days gained
	Baseline	Altered	
Male	75.115	75.214	36
Female	79.846	79.943	35
Baseline 2: All baseline rates reduced by 10%			
	Life expectancy (yr)		Days gained
	Baseline	Altered	
Male	76.248	76.348	36
Female	80.920	81.018	36
Baseline 3: All baseline rates reduced by 30%			
	Life expectancy (yr)		Days gained
	Baseline	Altered	
Male	78.978	79.081	38
Female	83.497	83.597	36
Baseline 4: Future baseline rates reduced by 0.1% per annum			
	Life expectancy (yr)		Days gained
	Baseline	Altered	
Male	75.803	75.903	37
Female	80.553	80.651	36
Baseline 5: Future baseline rates reduced by 0.5% per annum			
	Life expectancy (yr)		Days gained
	Baseline	Altered	
Male	78.758	78.866	39
Female	83.551	83.654	38

3.2.4 Summarising the effects

We now have a number of summary statements that can be made to compare the mortality of pollutions under different scenarios.

The graphs in Figures 2 and 3 give a clear depiction of the effects of the reductions in hazard rates on a population experiencing the same all-cause mortality (hazard) rates as the male population in England and Wales 1999. However, there is generally a need to express these

differences with some kind of summary statistic. We can make a number of statements from our calculations. Overall, no deaths are saved by reducing the hazard rates, since all the cohort must eventually die; however, the pattern of deaths is different, and on average these occur slightly later for the reduced hazards. For a 1% change in all-cause hazard rates at ages 30 and above, there is a total gain of 9873 life-years per 100,000 starting population, equivalent to 36 days per individual, in the birth cohort. By the linearity mentioned earlier, we may estimate by scaling that, for example, a reduction of 0.5% in hazard rates would have gained 18 days per cohort member.

We may make other summaries for specific purposes: for example, we may note that the baseline population has 82.9% surviving to their 65th birthday, increasing to 83.1% with the reduced adult hazards. We can also calculate, conditional on achieving a particular age, the additional life expectancy above that age. Table 3.7 shows example results: a male reaching the age of 50 can expect, on average, another 28.27 years of life based on the 1999 rates. The average age of death for these males would then be 78.27, which is over three years more than we predicted for life expectancy from birth. This is because the population is not homogeneous in risk, and we may expect the frailer individuals to die younger, on average, so that the population age 50+ contains has a larger proportion of more robust survivors.

Table 3.7: Comparison of life-table results, summarised as conditional life expectancy from age 50, with baseline using all-cause hazard rates for England and Wales, 1999, and altered having a 1% reduction on baseline, at ages 30+.

	Conditional life expectancy (yr) from age 50		
	Baseline	Altered	Days gained
Male	28.270	28.361	33
Female	32.267	32.358	33

There can be confusion about the relationship between changes in patterns of deaths and life-years, but they are as we have seen functionally related. In particular, a death avoided at a particular age, if it is a random selection from the population, may be treated as gaining the average future life expectancy appropriate to that age. Thus the total gain in life-years can also be seen as the total gain in life expectancy over the deaths whose timing has been altered. It is not uncommon for this observation to be used in simplified calculations; for example, if the expected remaining life for a 20-year old male is 56 years, and a road safety campaign avoids 10 deaths of male 20-year olds, then it might be calculated that we have saved an expected 560 life-years. This approximation ignores that in a theoretical scenario where there are ten fewer deaths at age 20, the expectation of life will be a fraction longer. In this example, the difference is clearly negligible, but in scenarios with larger differences in hazards this might not be the case. The full method comparing life-tables avoids this source of potential bias, of whatever size.

3.2.5 Weighted summaries: value, discounting and quality of life

When we total the differences in deaths or life-years, we implicitly give each equal weight. However, there are many applications where it may be desired to apply differential weights to these units; we describe some of the most common here. We consider these for life-years. Some can also be applied to deaths, perhaps with some additional summation over age-groups.

One important example is where monetary value is being assigned to a life-year, e.g. to quantify the value of benefits in a cost-benefit analysis (CBA) of proposed pollution reductions. It is legitimate to ask whether a year of life should be valued at the same amount at any stage in life, or whether years at different ages should have different values. Questions like this are controversial, and answers can depend greatly on who is making the judgments and for what purpose. If they are based on an external judgement on economic productivity, then it is likely that years at advanced ages will be valued at less than those lived during normal working ages. However, this approach raises difficult ethical implications.

An alternative approach, which has had much currency in recent years, is based on the principle of “willingness to pay” (WTP), which has the merit of being based on the opinions of the target population, rather than of politicians or economists. In this approach, a representative sample of individuals at different ages and incomes are interviewed about how much they would be willing to pay in a number of scenarios involving (for the present application) reduction in the risk of mortality, now or in the future. The data from a set of interviews can be analysed statistically to produce an average WTP-based value of a life-year at a particular age. Interestingly, a number of studies have shown that the values derived from the WTP approach do not diminish with advancing age anything like so rapidly as might be expected; this is sometimes used as justification for valuing a year of adult life at a constant value, regardless of age.

Another consideration, relevant to monetary valuation, is that money changes its value over time, through price inflation and/or interest earned or missed. For that reason, economic costs and benefits are often “discounted” by some annual amount. Over a period of years, this has an effect similar to compound interest; and even with a relatively low discount rate, discounting over several decades can reduce the future value of a life-year, viewed from the present, by a large degree. For example, with a discount rate of 3%, a life-year experienced only 25 years in the future would have a value less than 47% of its current value.

A different approach adjusts life-years to allow for the consideration that perceived quality of life may change during the ageing process. Thus, an individual with chronic health problems may value a life-year lived in poor health as having poorer quality than a year lived in good health; or a health care professional may make such an assessment on behalf of the individual. For a population followed up over a period, the life-years experienced at a particular age may be valued at a range of quality scores, from 1 (representing full quality) downwards. If the distribution of these quality scores can be estimated and totalled for each age-group, then the life-years experienced at that age can be multiplied by a factor based on the average quality score, to estimate the total “quality-adjusted life-years” (QALYs). Since the average score will be less than 1, this represents a downward weighting of the life-years. If, in addition, the average quality score decreases with increasing age, then the down-weighting will be increasingly severe with increasing age. A similar adjustment is sometimes made based on quantifying the quality of life when living with a disability, leading by the same process to “disability-adjusted life-years” (DALYs).

In any particular application, any or all, or any combination, of these weightings may be relevant. Formally, however, in terms of the life table calculations, they can be treated similarly, one at a time or in combination. For example, since they are all multiplicative factors, we can combine them to create a compound set of age-specific weights that may be applied to the life-years predicted to be saved under a proposed impact; the two-dimensional matrices in our spreadsheets allow for this if required. The ability to apply these weights, however, depends on our having access to the age-specific distribution of life-years; unless the weights are constant over age or time, we have to apply the weights before we summarise over those dimensions.

3.3 COMPOUND POPULATIONS AND COMPLEX SCENARIOS

3.3.1 Choice of cohorts

As we have seen, we can construct a life-table with future mortality hazard rates for a birth cohort, simulate some assumed pattern of impacts on those rates, and compare the patterns of expected mortality that they define over the life of cohorts experiencing the baseline and impacted rates.

Real life situations are more complex. A pollution reduction introduced today impacts not just on a single birth cohort, but on a whole population, with a distribution of current ages; and improvements to air quality made today are likely to improve the life expectancy of cohorts yet to be born. Early attempts to use life-table methods for air pollution impacts failed to take full account of the complexity this implies. Indeed, it is possible to accommodate this complexity only by clearly separating the dimensions of age and calendar time.

Once we make this separation, however, life becomes much simpler. Each age-specific cohort alive today will have its mortality experience defined by a set of age-specific hazard rates, increasing as the cohort ages. The pattern is shown in Table 3.8 (based on Miller and Hurley, 2003), which imagines a set of predictions running forward from the year 1999. Each one-year age group must get one year older with the passage of one year, which implies that one cohort's future mortality experience is defined by the rates down one diagonal of the matrix. The expected mortality experience of each cohort can therefore be calculated by performing life-table calculations down the appropriate diagonal. Results can then be cumulated across cohorts as required.

Table 3.8
Schematic layout showing organisation of data, and life-table calculations for prediction of mortality effects (e=entry population, b=births, h=hazard rate)

Age	Entry pop ⁿ	Year												
		1999	2000	---	2004	2005	2006	2007	----	j	----	2108	2109	2110
		Births	b ₁	---	b ₅	b ₆	b ₇	b ₈	---	b _j	---	b ₁₀₈	b ₁₀₉	b ₁₁₀
0	e ₀	h ₀	h ₀		h ₀	h ₀	h ₀	h ₀		h ₀		h ₀	h ₀	h ₀
1	e ₁	h ₁	h ₁		h ₁	h ₁	h ₁	h ₁		h ₁		h ₁	h ₁	h ₁
2	e ₂	h ₂	h ₂		h ₂	h ₂	h ₂	h ₂		h ₂		h ₂	h ₂	h ₂
⋮	⋮													
I	e _i	h _i	h _i		h _i	h _i	h _i	h _i		h _{i,j}		h _i	h _i	h _i
⋮	⋮													
103	e ₁₀₃	h ₁₀₃	h ₁₀₃		h ₁₀₃	h ₁₀₃	h ₁₀₃	h ₁₀₃		h ₁₀₃		h ₁₀₃	h ₁₀₃	h ₁₀₃
104	e ₁₀₄	h ₁₀₄	h ₁₀₄		h ₁₀₄	h ₁₀₄	h ₁₀₄	h ₁₀₄		h ₁₀₄		h ₁₀₄	h ₁₀₄	h ₁₀₄
105	e ₁₀₅	h ₁₀₅	h ₁₀₅		h ₁₀₅	h ₁₀₅	h ₁₀₅	h ₁₀₅		h ₁₀₅		h ₁₀₅	h ₁₀₅	h ₁₀₅

We have implemented these calculations in a set of spreadsheets (Miller and Hurley, 2003), which we have called the IOMLIFET system. This features

- separate calculations for males and females;
- age- and year-specific impacts on cause-specific hazard rates;
- optional age-and-year-specific weightings for life-years;

- optional monetary discounting;
- flexibility in summarising outputs across populations and cohorts.

Once populated with values appropriate to a particular impact assessment, the spreadsheets carry out all the necessary calculations.

3.3.2 Future baseline hazards

It follows that a first step in estimating the effects of proposed impacts on mortality for entire populations is to construct predictions of mortality patterns in the absence of those impacts. This requires us to populate a matrix such as in Table 3.8 with future hazard rates h_{ij} indexed both by age and by year. In work we have done to date, we have populated the first column with rates from a recent year, and assumed identical age-specific mortality rates for future years. Under this assumption, cohort life-tables and current life-tables (see 3.1.3) must be identical. However, this is merely an assumption, and it is possible to adopt alternative assumptions.

UK Government Actuaries, when calculating cohort life-tables, already assume that recent trends in mortality reduction will continue onto the future, and their predictions for life expectancy for a new-born cohort are therefore longer than would be predicted from a current life-table. However, we have shown empirically (3.1.3) that impact assessments are relatively insensitive to the levels of assumed future baseline hazard rates, so our present choice of using current rates is unlikely to be a serious distortion.

3.3.3 Age- and/or time-specific impacts

Given a set of current and future hazards laid out as in Table 3.8, it is apparent that we can construct a new version with impacted hazards h'_{ij} . We may set this up as a set of impact factors f_{ij} , so that we calculate our new hazards by multiplication:

$$h'_{ij} = f_{ij} \times h_{ij}$$

We can set up a matrix with any pattern we choose in the f_{ij} , which can thus vary by age and/or year. Any $f_{ij} = 1$ implies that the impacted hazard is identical to the baseline.

Impacts that vary by age may be based on expectations that the impacts will in fact differ depending on the age of those affected, or they may be based on lack of data. For example, the most influential of cohort studies of associations between air pollution and mortality (Pope *et al* 1995; 2002) studied only adults. While it showed no strong difference in relative risk between its older and younger subjects, it provided no information about effects in the under-30s. As a result, it has become common practice in impact assessment to model effects only at ages 30 and above. An effect in the under-30s is one of several plausible alternative assumptions that might be modelled. Indeed, there is strong evidence from another cohort study of increased mortality in infants (Woodruff *et al*, 1997), and time series studies in several countries have shown increased mortality in children following days of higher air pollution.

Year-specific impacts have particular application where impacts are expected to be staged. This allows the modelling of gains that accrue gradually after an intervention, or the effects of a series of improvements. A two-dimensional array of f_{ij} can accommodate any combination of these assumptions and many more.

3.3.4 Cause-specific impacts

We may wish to estimate the impacts of interventions that have been documented to affect only certain causes of death. For example, air pollution has been associated strongly only with cardio-respiratory causes.

Since cause-specific hazard rates are additive, we may in fact model cause-specific impacts (Miller and Hurley, 2003). This requires mortality rates by age for selected (groups of) causes of death, which are readily available for Great Britain. We may show this by introducing a third index, k , for cause. Then

$$h_{ij} = \sum_k h_{ijk}$$
$$h'_{ij} = \sum_k f_{ijk} \times h_{ijk}$$

Again, the f_{ijk} may accommodate any pattern, and any subset of f_{ijk} may be set to 1 to leave the corresponding original age-, year- and/or cause-specific hazards unchanged.

3.3.5 Length of follow-up

For a single cohort, the impact of a reduction in hazard rates can be quantified over the whole life of the cohort (which we assume to have a maximum age of 105 years). When we consider a matrix such as that in Table 3.8, and we envisage a reduction in hazards in a particular year, then all cohorts born in successive years will also benefit, and it is legitimate to predict accumulated gains over those cohorts also. However, there is usually a practical limit to how far ahead people want to quantify predicted gains. It is thus necessary to specify over what period, and what cohorts, impacts will be quantified and summarised.

Different choices here will have different implications. We may for instance choose to predict gains in only the population alive in the year when the impacts occur; then we will summarise the impact over the values in the darkened triangle of Table 3.8, noting that these require the life-table calculations plus the size of each cohort at the beginning of the follow-up. However, we may wish to include also future cohorts yet to be born. Then we need also to specify the size of each future birth cohort. However, if we stop quantifying in a particular year, represented by the right-hand margin of Table 3.8, then we must note that most later cohorts will be followed up incompletely. Alternative approaches might lead us to continue follow-up for a longer period into the future, but this would require future assumptions to be made for a longer period, and this may not be satisfactory.

In practice, this may be less of a problem than it appears. In most cost-benefit analyses, future values will be discounted, and an important effect of discounting is that it substantially reduces value after a few decades. (See 3.2.5) A discount rate of 3% produces, over 105 years, a reduction of 96%, so the discounted contribution of cohorts born many decades in the futures would be negligible. However, fashions in discounting change, and in some recent reports (IGCB, 2006) economists have added a 2% “uplift” per year to their willingness-to-pay valuations, to account for the expectation that people’s willingness to pay will rise over time with inflation, increased earnings, etc. It is clear that an uplift of 2% would cancel a discount of 2%, and reduce considerably the impact of a 3.5% discount; and that there could be valuation scenarios where events far in the future were dominant rather than negligible.

3.4 ONE-YEAR PULSES

3.4.1 Estimating the effect of a one-year pollution reduction

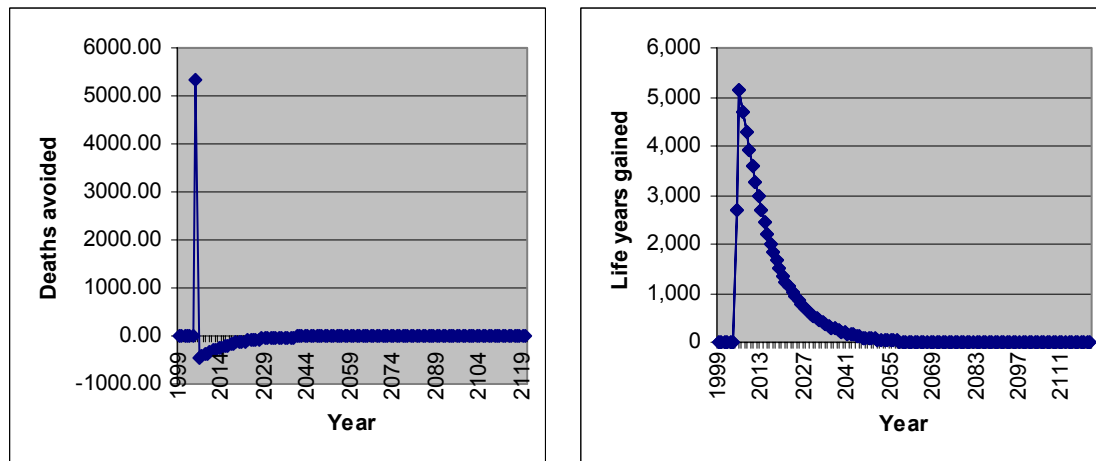


Figure 3.4: Reduction in deaths (left) and gain in life-years (right) from a one-year pulse reduction of 1% in all-cause hazard.

Some policy stakeholders have been interested in quantifying the effects of a one-year temporary reduction in pollution, sometimes referred to as a “pulse”. While it is unlikely that such a policy would be implemented, nevertheless policymakers have found the simplified pulse scenario appealing for their deliberations, and we have used it in work for ExterneE (1995; 1999) and more recently in work for DEFRA (Watkiss *et al*, 2005) and in CAFE cost-benefit analysis (Hurley *et al*, 2005).

There were two underlying motivations for this. Through the 1990s, HIA and CBA of air pollution focussed on the effects of daily variations to air pollution on health effects. These effects, quantified by time-series studies, are more-or-less immediate following changes in exposure, and it became usual to aggregate them as effects of one year’s (change in) exposure to pollution. The first motivation, then, was to try to express the effects of long-term exposure on the same basis as the effects of one year’s exposure. Second, some stakeholders found it convenient, both conceptually and from the viewpoint of calculation, to think of sustained pollution reduction as the aggregate of a series of one-year pulses. Where those pulses represented differently-sized reductions in pollution, the contributions from different years could be scaled proportionally, and summed to give total impacts.

As an example, we have calculated the impact of a one-year 1% reduction in all cause hazards in the year 2005, in an IOMLIFET life-table projected forward from 1999 populations and hazard rates. The effects can be seen in the two graphs in Figure 3.4. That on the left shows the total numbers of deaths “saved” by the impact, that is, the numbers of annual deaths fewer in the impacted scenario than in the baseline. In the year of the impact (2005), there are more than 5,000 fewer deaths, but from 2006 onwards there are more deaths per annum, until the affected cohort is extinguished after 76 years. This is because all deaths have to occur sometime, and a reduction in hazards implies a later pattern of deaths overall. Another way to see this is that if there are fewer deaths in 2005, the 2006 population is larger, and applying the same baseline rates to a larger population must produce more deaths. The effects are experienced by the population alive in the year the change occurs; a one-year change can have no effect on cohorts born in following years.

The graph on the right shows the distribution of the life-years gained from the one-year impact. Because deaths are assumed to take place throughout the year, each “saved” (actually deferred) death contributes the average 0.5 life-years. However, the 2006 population is swelled by the number of saved deaths, and most of them also live through 2006, so the gain in life-years in 2006 is almost twice that in 2005; after that, it declines steadily but always remains positive. The total life-years gained in this scenario is 29,949 for males and 29,421 for females, giving a total of 59,370 life-years.

3.4.2 Accumulating the effects of a series of pulses

Given that it is straightforward to quantify the effect of a one-year pulse change in hazards, some stakeholders have sought to extrapolate the results to more complex scenarios. In particular, it has been assumed that the effects of a change taking place and being sustained over several years could be approximated simply by multiplying the effects of a one-year pulse by the number of years over which pollution change is sustained. However, there has been no attempt to check how good is this approximation.

As we have noted already, discounting the value of life-years changes the temporal pattern of accrual of benefit; we might expect that discounting would complicate the use of a multi-pulse approximation. Since the full impact of a pulse change has to be calculated in a spreadsheet system such as IOMLIFET, it is straightforward to produce there a total impact that includes future discounting. However, the impacts of future pulses should be further discounted because they begin further in the future. On the face of it, this seems complex, but a simple algebraic result comes into play. Suppose we have a result from a one-year pulse, and that the gains in each year have been discounted at the rate of r per annum before being summed (say total = K). Then if the same gains were achieved a year later, they would need to be further discounted by r , and those in the second year discounted by r^2 and so on. Over n years, the sum would be

$$\begin{aligned}\text{Total gain} &= K(1 + r + r^2 + \dots + r^{n-1}) \\ &= K(1 - (1 - r)^n) / r\end{aligned}$$

using a standard result for a partial sum from a geometric series. (If the discount rate is expressed as $d\%$, then we set $r=d/100$).

Thus if we have a discount rate of 1.5%, the multi-pulse method should estimate the total discounted gain for 20 years as 17.4 times the gain from a one-year pulse. With a discount rate of 3.5% this factor reduces to 14.6. We have not included in these calculations any allowance for “uplift”, as discussed in 3.3.5 above.

Some stakeholders have used the method of adding together the effects of multiple pulses, suitably scaled, to estimate the effects of a series of changes in hazard, such as might be produced by introducing a series of increasingly effective pollution controls. This is straightforward in concept, although somewhat cumbersome, and is probably best done in a spreadsheet. Combining a complex scenario with discounting makes for more complex calculations, confirming the need for a spreadsheet layout. In such a case, the effort involved becomes more than that required to program the full set of changes in the IOM spreadsheets.

Nevertheless, there are those who favour the approach, so it is necessary to ask whether it gives a reasonable approximation to the fully worked life-table answer. The simple answer is that they do not give identical answers. As we have seen, the distributions by age of future hypothetical populations will be dictated by the original age distribution and by the distribution of the hazard rates. Under a different set of hazards, the age distribution must change over time in a different way. The projection forward of the results from a one-year pulse cannot take into account shifts in population, while the full life-table method does this

automatically, and so accumulation of results for a series of pulses gives only an approximation to a full life-table analysis.

We have used IOMLIFET to perform a number of prediction simulations to estimate the size of this approximation under various assumptions. These run from published population figures for England and Wales in 1999, separately for males and females, in one-year age groups. For ages above 90, the hazard rates are extrapolated to age 105 inclusive (Version 3, Table 3.5).

As a baseline, we assumed that the 1999 all-cause mortality rates would continue to apply to all years from 1999, and from these we calculated the life-years expected to be lived at each age in each calendar year into the future.

For the sustained 20-year change, we reduced the all-cause hazard rates for all ages over 30 by either 1% or 10%, for the years 2005 to 2024 inclusive. Hazards for the remaining years were identical to baseline.

For the one-year change, we did the same, but altering the hazards only in 2005, by 1% or 10%.

In both cases, we then ran the life-table calculation tool and summarised the differences (gains) in predicted life-years, for males and females, between the baseline and the impact scenarios investigated.

3.4.3 Comparative results

We have summarised, for males and females, the total predicted life-years gained from the changes in hazards, in a population predicted forward from 1999 all-cause hazard rates. For a 20-year change from 2005, given that these changes are applied only to those over 30, the last predicted impact for those aged 30 in 2024 is for when they are 105, in 2099.

For the one-year change, the last predicted impact is in 2080. However, if we then imagine this impact being replicated over another 19 years, it is clear that the prediction is being made for the same calendar period. It is also worth noting here that for scenarios where changes are expected to have effects on hazards for many years into the future, the final effects from repeated pulses will be very far in the future. Care is needed here when comparing results from repeated pulses and full life-table estimates, that the effects are being summed over the same periods.

The two graphs in Figure 3.5 show the effect of a 1% change in all-cause hazards sustained through the years 2005-2024 inclusive. The immediate change in death numbers is apparent, but it is also notable that the initial difference reduces fairly rapidly over the 20 years, due to the population changing its age distribution. The life-years gained accumulate at a slightly less than linear rate over the 20 years, then decline to zero after a further 75 years.

The total life-years gained under this scenario is 657,019 for males, and 618,769 for females, total 1,275,788. In contrast, multiplying the one-year value by 20 produces a total of 1,187,400, which is 93.1% of the value for the sustained change. Repeated pulses give a lower value than a full life-table analysis, because the results relate to a reduction in hazard that will produce a gradual increase in population size. The life-table method captures this effect, but the pulse method does not.

The two graphs in Figure 3.6 show the effect of discounting. They correspond to the one-year (left) and 20-year (right) impacts, and show the pattern of accumulation of discounted values of life-years. In comparison with the undiscounted versions, their shapes are similar but they approach zero more quickly in the later years.

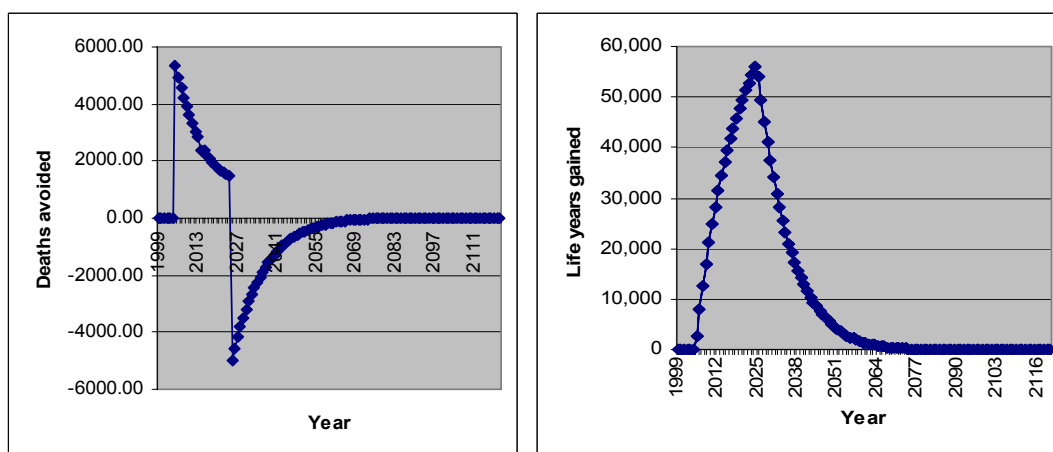


Figure 3.5: Reduction in deaths (left) and gain in life-years (right) from a 20-year sustained reduction of 1% in all-cause hazard.

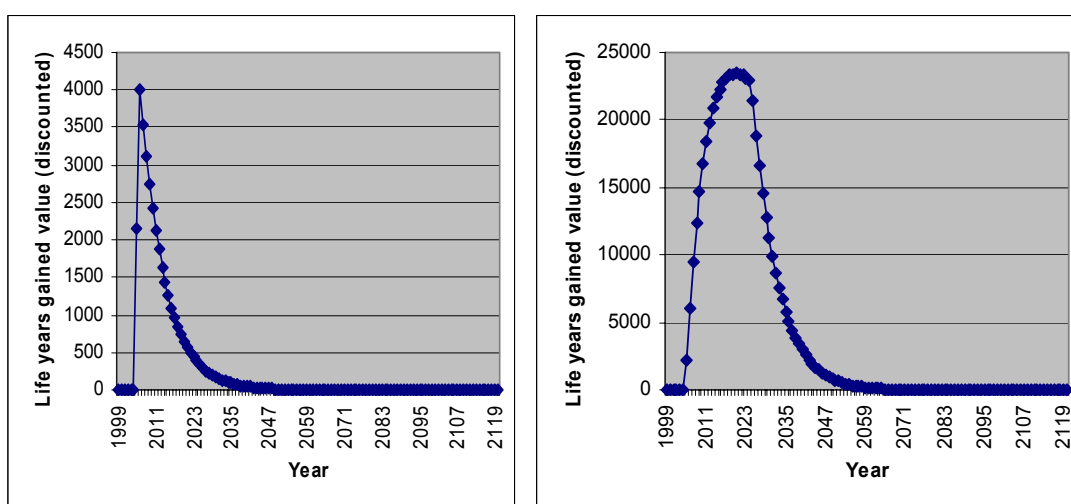


Figure 3.6 Gain in life-years, discounted at 3.5% per annum, from reduction of 1% in all-cause hazard as a one-year pulse (left) or from a 20-year sustained reduction (right).

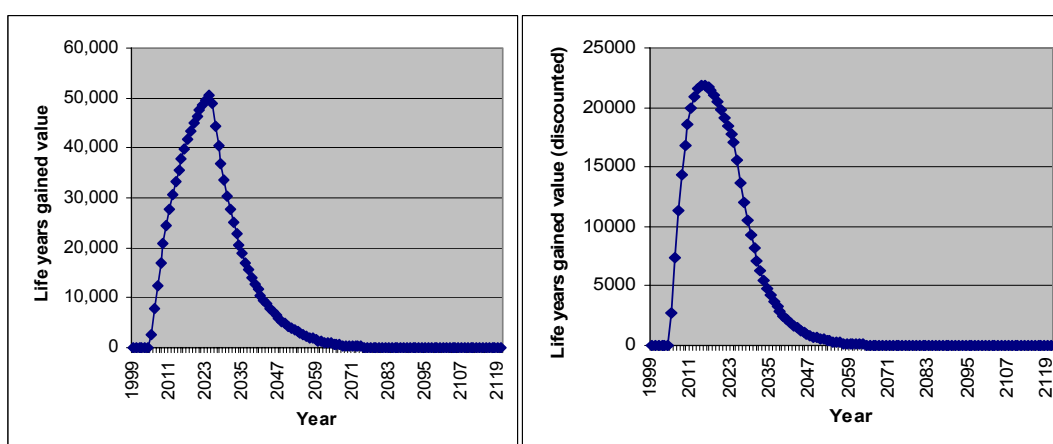


Figure 3.7 Gain in life-years, estimated as total from 20 successive pulses without (left) and with (right) discounting at 3.5% p.a.

Figure 3.7 (left) shows the pattern of the life-years gained, estimated by taking the results from a one-year pulse (as in Figure 3.6 (left)) beginning in 2005, replicating this for each of the years 2006-2024 inclusive, and adding the results to give a total life-years. In shape, this is very close to Figure 3.5 (right) for the 20-year sustained change, with the values from the repeated pulse producing slightly lower values. Figure 3.7 (right) shows the life years gained after applying an annual discount of 3.5% for each year after 2005. Again, this is quite similar to Figure 3.6 (right) for the sustained 20-year change, and here it is easier to see that, in addition to the slightly lower values overall, there is a slight difference in the year-to-year distribution of the gains making up the peak.

To give comparisons over a wider range of scenarios, we have run prediction simulations for 1% and 10% changes in hazards, either for one or 20 years, and have assessed the total life-years gained with and without discounting at 1.5% and 3.5%. Table 3.9 summarises the results, both in total and separately by sex. The second and third columns of the table hold the estimated life-years gained under the sustained and pulse scenarios, and the pulsed values are then multiplied by a value to scale them to 20 years, with discounting as appropriate.

The final column expresses the total obtained from 20 replications of the one-year results with those estimated directly from a 20-year sustained reduction.

The replicated pulse method always underestimates compared to the full life-table prediction, although never by more than 11%. The discrepancy is somewhat greater for the 10% change than for the 1%, which is because the difference between the 1% and 10% results for the sustained life-table result is greater than simple proportionality. However, a 10% reduction in all-cause mortality hazard is well outside the achievable range. In addition, there is a greater difference for males than for females. This is presumably due to the fact that male hazard rates are uniformly higher than female. The differences reduce very slightly with higher discounting rates.

In summary, we can see that for these inputs the multiple-pulse approach gives results that are similar to, but somewhat smaller than, the results from the full life-table implementations. Given that the results for a one-year pulse have to come from the life-table spreadsheet in the first place, there is no advantage to using the pulse method *ab initio* where the spreadsheets are available, since it involves more steps of calculation. However, someone who has been provided with a one-year pulse estimate as a single summary total gain could scale that to multiple years to obtain a result that would not be seriously imprecise, and could also in certain simple circumstances apply discounting. However, discounting would be harder to accommodate in scenarios including multiple changes, of different sizes, in hazard rates.

Given these results, we recommend that, wherever possible, results for impact assessments should use the full life-table method, utilising the flexibility of the two-way IOMLIFET matrix layout.

Table 3.9: Summary results for life-years gained (with discount where appropriate) for scenarios of sustained and pulse hazard reductions at 1% and 10%

Discount rate %	Sustained 20 yrs	Pulse 1 yr	20 yr discount factor	Pulse *20 discounted	%
Life-years gained for 1% reduction in all-cause hazard					
TOTAL					
0	1,275,788	59,370	20.000	1,187,400	93.1
1.5	876,678	47,009	17.391	817,529	93.3
3.5	546,472	35,140	14.560	511,643	93.6
MALE					
0	657,019	29,949	20.000	598,980	91.2
1.5	450,664	23,652	17.391	411,330	91.3
3.5	280,121	17,622	14.560	256,579	91.6
FEMALE					
0	618,769	29,421	20.000	588,420	95.1
1.5	426,014	23,357	17.391	406,199	95.3
3.5	266,351	17,518	14.560	255,064	95.8
Life-years gained for 10% reduction in all-cause hazard					
TOTAL					
0	13,099,912	594,941	20.000	11,898,820	90.8
1.5	9,008,041	471,140	17.391	8,193,551	91.0
3.5	5,617,589	352,234	14.560	5,128,575	91.3
MALE					
0	6,743,927	300,082	20.000	6,001,640	89.0
1.5	4,628,688	237,018	17.391	4,121,957	89.1
3.5	2,878,133	176,614	14.560	2,571,524	89.3
FEMALE					
0	6,355,985	294,859	20.000	5,897,180	92.8
1.5	4,379,353	234,122	17.391	4,071,593	93.0
3.5	2,739,456	175,620	14.560	2,557,051	93.3

3.5 COUNTING DEATHS

From the various discussions above, it should be clear that we favour, in general, quantifying mortality impacts in terms of years of life lost, rather than as deaths “saved” (more accurately, deferred). It is worth repeating the reasons for the preference. For a single birth cohort, a reduction in the expected future hazard rates will lead to a later pattern of deaths, but once the cohort is extinguished no deaths will have been saved. We may be interested in predictions for the whole of a population current when a change takes place. Treating this population as as a set of cohorts who have already lived parts of their lives, again future hazard reductions will alter the pattern of deaths so that they happen later on average, but in the end the number of deaths that will occur is the same as the size of the current population. Therefore, in a follow-up to extinction of the current population, no deaths are saved.

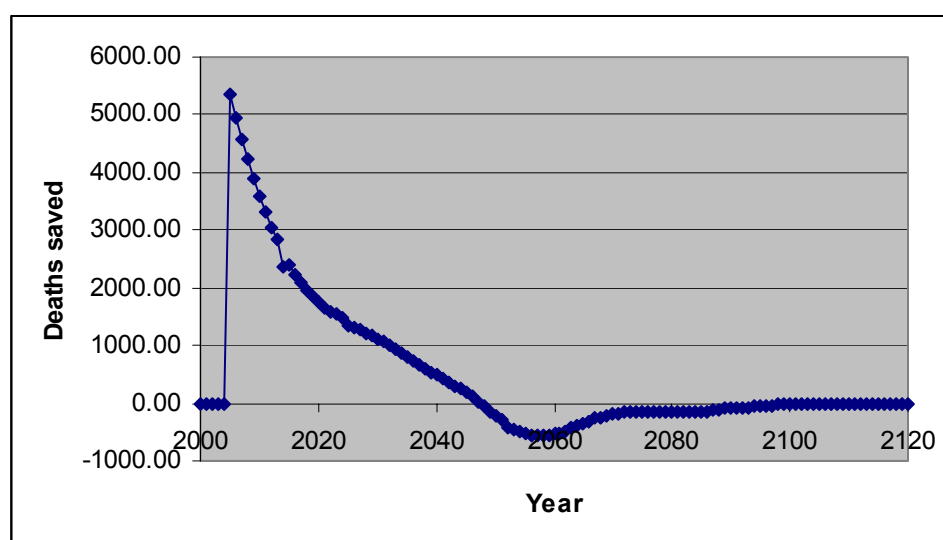


Figure 3.8: Difference in numbers of deaths from a sustained reduction of 1% in all-cause hazard; follow-up of extended population.

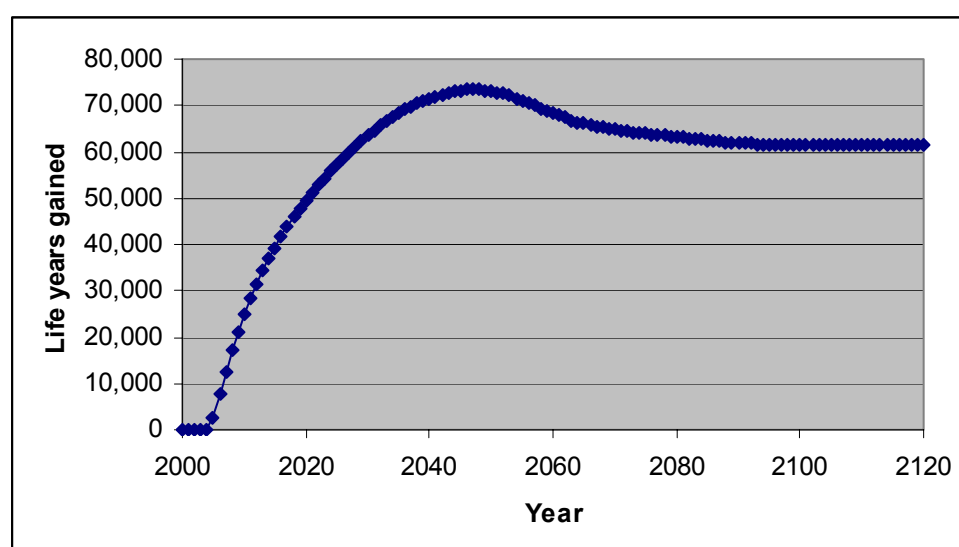


Figure 3.9: Difference in numbers of life-years from a sustained reduction of 1% in all-cause hazard; follow-up of extended population.

However, if we are calculating the predicted impact of a permanent change in pollution, which will have an effect of decreasing hazards for all years in the future, we should expect that the total number of deaths in each year will decrease. Figure 3.8 shows the impact of a 1% reduction in all-cause mortality from 2005 on the number of deaths in that and each succeeding year. (The small glitch at 15 years is an adjustment due to the approximation that cumulative survival is truncated after age 105.)

In our baseline scenario, the age-specific all-cause hazard rates are assumed to be the same in all future years, and the reduction made from 2005 onwards is a constant 1%. Yet we see that, although in 2005 we have 5000 fewer deaths, the saving in deaths reduces fairly rapidly over the next decades, and after 2047 there are actually more deaths under the altered scenario with the reduced hazards. This is because, with reduced hazards, each year sees more survivors to the next, and the population under the altered scenario is increasing more rapidly than in the baseline. After 2005, we are comparing numbers of deaths for increasingly divergent denominators between the baseline and altered scenarios.

Figure 3.9 presents the saving in life-years from the same reduction of 1% in all-cause hazards. This summary shows clearly the ongoing advantage from this reduction, increasing rapidly at first and finally levelling out to a constant annual gain once the population shape has stabilised.

The difference in the number of deaths suffers from the twin disadvantages that the population denominators are not comparable, and that it counts deaths without taking account of the age at death. Neither of these affects the life-years summary. We therefore suggest that the gain in life-years is a much truer and more reliable summary of the gains from the hazard reduction than the difference in the number of deaths.

3.6 COMPARING THE IMPACTS OF DIFFERENT EFFECTS

The methods described here have been developed to estimate and express effects in the context of long-term exposure to particulate air pollution having an impact on annual mortality rates, a sufficient but by no means necessary justification. The life-table calculations themselves require only changes in the hazard rates, and will yield the same impact predictions for the same set of assumed changes, regardless of the external drivers of those changes. Thus, while the IOMLIFET system provides a framework for comparing impacts of different patterns from one type of effect, it will also serve to make comparisons of impacts from different effects, so long as we can associate these with changes in cause-specific hazard rates.

Within the present report, we have chosen to compare the impacts on mortality of

- particulate air pollution;
- passive smoking;
- road traffic accidents.

For each risk factor, we have assumed a change in hazard in 2005, and have quantified effects in the birth cohort of that year, in the whole population alive in that year, and in the extended population that includes those alive in 2005 and all cohorts born in succeeding years, up to and including 2110, the year in which the 2005 cohort is extinguished.

The calculations, using the IOMLIFET spreadsheets, are based on predicted populations projected forward from age- and sex-specific data for England and Wales, 1999. Hazard rates were available separately for the following cause groups:

- 1 Lung cancer
- 2 All other cancers
- 3 Cardiovascular
- 4 Non-malignant respiratory
- 5 Motor vehicle traffic accidents
- 6 All other causes

4 RESULTS

4.1 PARTICULATE AIR POLLUTION

4.1.1 Input coefficients

Results are illustrated with reference to a reduction in annual average ambient $\text{PM}_{2.5}$ of $10 \mu\text{g.m}^{-3}$. It doesn't really matter what change in annual average $\text{PM}_{2.5}$ is used to illustrate the results because the main results are linear in % change in hazard and so also linear in change in concentration. (The concentration-response (C-R) function, linking concentration of $\text{PM}_{2.5}$ with % change in mortality hazard, is also linear across the effective range of concentrations to be considered). We chose $10 \mu\text{g.m}^{-3}$ to illustrate the results because many epidemiological studies and reviews report risk estimates in relation to $10 \mu\text{g.m}^{-3}$ PM. This includes Pope *et al* (2002), the paper now preferred by many expert bodies as a source of C-R functions linking long-term exposure to $\text{PM}_{2.5}$ with changes in mortality hazards. Also, though $10 \mu\text{g.m}^{-3}$ annual average $\text{PM}_{2.5}$ is a large change in terms of possible pollutant reductions in the UK, it is not so large an amount that it is unrealistic to think about impact estimates based on a difference of $10 \mu\text{g.m}^{-3}$ $\text{PM}_{2.5}$.

Having selected a difference of $10 \mu\text{g.m}^{-3}$ $\text{PM}_{2.5}$ as a basis for comparisons, it is necessary also to select an associated risk coefficient, i.e. an estimate of the percentage change in mortality hazards associated with, and arguably attributable to, a change of this magnitude in annual average concentrations of $\text{PM}_{2.5}$. Fortunately several expert groups of air pollution researchers have reached a common view about this. For some years now the World Health Organisation has recommended that HIA of outdoor air pollution should use a coefficient of 6% change in mortality hazards, per $10 \mu\text{g.m}^{-3}$ $\text{PM}_{2.5}$ (annual average). Reasons are given in several documents, including a recent WHO overview publication on particulate matter (WHO, 2006). This coefficient was also used in HIA and CBA work of the Clean Air for Europe (CAFE) Programme of the European Commission (Hurley *et al*, 2005) and in work of the European Apheis project. Most recently, following in-depth review of underlying issues, it was the estimate of relative risk recommended by COMEAP in the UK (COMEAP, 2006).

In line with our earlier work, and what seems to be conventional in HIA of air pollution, we have applied the risk reduction to the hazard rates for those aged 30+ only. It reflects the fact that the American Cancer Society cohort, whose results underlie the choice of coefficient, included only people aged 30 years or more at recruitment to the study, and an associated unwillingness of many air pollution experts to extrapolate the ACS risk estimates to younger ages. There is, however, substantial evidence from other studies that ambient air pollution measured as PM is adversely associated with mortality in children and infants and so the conventional approach is in fact a conservative one.

4.1.2 Results

A 6% reduction in all-cause hazards leads to an estimated gain of 222 days in male life expectancy, and 218 days for females. Within the population alive in 2005, we predict a gain of 14.7 mLY (million Life-Years) for males, and 14.6 mLY for females, giving a total of 29.4 mLY. Including partial follow-up for cohorts born after 2005 increases this total to 39.1 mLY.

4.2 MOTOR VEHICLE TRAFFIC ACCIDENTS

4.2.1 Input coefficients

Since we have separate hazards for motor vehicle traffic accidents, it is straightforward to set these to zero, and recalculate the total hazards as the total of cause groups (1 – 4, 6). We note

that this is in no way equivalent to proportional reductions in hazards, although it might be represented as a 100% reduction in all hazard rates for cause group 5.

4.2.2 Results

Elimination of MVTA hazards leads to an estimated gain of 81 days in male life expectancy, and 30 days for females. This is consistent with the known propensity of young males to indulge in dangerous driving behaviours. Within the population alive in 2005, we predict a gain of 2.4 mLY for males, and 0.9 mLY for females, giving a total of 3.4 mLY. Including partial follow-up for cohorts born after 2005 increases this total to 8.1 mLY.

4.3 PASSIVE SMOKING

4.3.1 Input coefficients

A recent paper by Jamrozik (2005) attempted to quantify various impacts of passive smoking on specific causes of death. The emphasis was on attributable numbers of deaths, but the paper summarises a number of facts that can help in the present case.

Studies of non-smokers exposed to environmental tobacco smoke (ETS) suggest that their risks of both lung cancer and ischaemic heart disease are increased by a factor of about 1.25. A median relative risk for strokes is quoted as 1.45. For the present analysis, we have assumed a relative risk of 1.25 for all cardiovascular causes.

Jamrozik estimates that 37% of the population 20-64 years old are exposed to ETS at home. The workforce constitutes 85% of that population, and 11% of those are exposed to ETS in the workplace. If we assume that these factors are independent, we may solve some algebra to estimate that, of the whole population 20-64, 34.9% are exposed only at home, 7.3% only at work, and 2.1% are unfortunate enough to be exposed in both environments.

If we assume that exposure at home and work leads to a compound relative risk, then the last group may have relative risks of 1.56. Over the whole population, the average relative risk over a population without ETS would be

$$RR = 0.557 + (0.349 + 0.073) \times 1.25 + 0.021 \times 1.56 = 1.117$$

For the population aged 65 and above, the prevalence of passive smoking at home is estimated at 13%, and we assume exposure at work will be negligible. For this group, therefore, the average relative risk is estimated at

$$RR = 0.87 + 0.13 \times 1.25 = 1.032$$

On these assumptions, we have estimated the gains from removal of passive smoking by multiplying hazards for lung cancer and for cardiovascular deaths by $1/1.117 = 0.895$, and for ages 65+ by $1/1.032 = 0.969$.

Jamrozik's assumptions include that the relative risks for passive smoking apply to everyone, regardless of their own smoking status. This may be unrealistic, particularly in heavy smokers; indeed, in this group, other people's smoke may make a negligible contribution to their health risks. An alternative assumption might be that the above calculations apply only to non-smokers, who constitute about 75% of the 20-64 age group. If the relative risk of lung cancer among all smokers compared with non-smokers is, say, 5.0, then the overall relative risk for passive smoking would be

$$RR = (5 \times 0.25 + 1.117 \times 0.75) / (5 \times 0.25 + 0.75) = 1.043$$

In older people the prevalence of smoking is lower, say 15%, so the equivalent calculation would become

$$RR = (5 \times 0.15 + 1.032 \times 0.85) / (5 \times 0.15 + 0.85) = 1.017$$

For cardiovascular deaths the relative risks for smoking would be less extreme, so the adjusted relative risks might lie between these and those estimated under Jamrozik's assumptions.

4.3.2 Results

The above reductions in hazard rates lead to a gain of 88 days in male life expectancy, and 62 days for females. This is consistent with the higher smoking prevalence and correspondingly higher lung cancer mortality rates in males. Within the population alive in 2005, we predict a gain of 5.5 million life-years (mLY) for males, and 3.9 mLY for females, giving a total of 9.4 mLY. Including partial follow-up for cohorts born after 2005 increases this total to 13.2 mLY.

4.4 SUMMARY OF COMPARISONS

Table 4.1 summarises the impacts that we have estimated, in terms of the expected gain in life expectancy for a single birth cohort experiencing the change. We see that the impacts from air pollution are nearly the same for males and females; that they differ more for passive smoking; and that the elimination of deaths from road traffic accidents would show a much greater gain for males than for females. To give context for these comparisons, we may note that a $10 \mu\text{g.m}^{-3}$ reduction in $\text{PM}_{2.5}$ is roughly equivalent to the elimination of all anthropogenic $\text{PM}_{2.5}$ (IGCB, 2006), so it makes an appropriate comparator for the elimination of MVTA or of passive smoking. Of course, it might be considered more realistic to compare less ambitious target improvements, say a 5% reduction in each, but we know that the results would all scale proportionally, so the comparisons would show the same relationships between effects.

If we average the effects of passive smoking across the sexes, we get a gain of 75 days, while a $10 \mu\text{g.m}^{-3}$ reduction in $\text{PM}_{2.5}$ yields a gain of around 220 days. This suggests that passive smoking has an effect approximately equivalent to that of a reduction in $\text{PM}_{2.5}$ of about $3.5 \mu\text{g.m}^{-3}$. MVTAs in males are of the same order of magnitude, while in females they are equivalent to about $1.5 \mu\text{g.m}^{-3}$.

Table 4.1: Gain (days) in life expectancy for a birth cohort

Effect	Impact	Male	Female
$10 \mu\text{g.m}^{-3}$ reduction in $\text{PM}_{2.5}$	6% all-cause reduction (from age 30)	222	218
Eliminate MVTA	Set MVTA hazard = 0	81	30
Eliminate passive smoking	Reduce Lung Ca and CV hazards by 10.5% (20-64), 3.2% (65+)	88	62

Table 4.2 shows the effect of these changes on compound populations: on the population estimated alive in 2005 when the change in hazards takes place; on new birth cohorts born between 2006 and 2110; and on the extended population that is the sum of the two.

The inclusion of sub-cohorts of different ages at the change induces different relativities from those for the birth cohort. The impact of air pollution in the current population is almost the same for both sexes, but there is more difference in the new cohorts; presumably this is because the follow-up omitted in older females is greater than in the earlier-dying males. All the populations showed a pronounced sex difference for MVTAs and for passive smoking.

Table 4.2: Total gain in life years (000s) for compound populations

Effect	Population	Male	Female	Total
10 $\mu\text{g.m}^{-3}$ reduction in $\text{PM}_{2.5}$	Current population	14,741	14,629	29,370
	New cohorts	5,389	4,299	9,688
	Extended population	20,130	18,928	39,058
Eliminate MVTA	Current population	2,427	933	3,361
	New cohorts	3,578	1,188	4,766
	Extended population	6,005	2,121	8,126
Eliminate passive smoking	Current population	5,458	3,944	9,402
	New cohorts	2,422	1,370	3,792
	Extended population	7,880	5,315	13,194

In the current population, the effect of eliminating passive smoking in males was rather greater than the MVTA effect, because a lot of the “current” males would be past the age of highest MVTA risk. For a similar reason, the reverse is true of the new cohorts. The MVTA effect in the current population was also only about 16% of that for air pollution. Elimination of passive smoking showed a reverse pattern, attributable to the fact that hazard rates for lung cancer and heart disease concentrate in older subjects.

5 DISCUSSION

5.1 METHODOLOGY FOR HEALTH IMPACT ASSESSMENT

It seems likely that needs for Health Impact Assessments will grow as more policy decisions are made on the basis of Cost-Benefit Analyses. Where the health outcome of interest is a non-fatal condition, particularly if it is treatable or reversible, a quantification methodology based on incidence predictions from exposure-response relationships will usually be satisfactory. Where the outcome is mortality, on the other hand, we meet numerous problems.

These problems begin with the observational studies that underlie the relationships. Mortality is a one-off event, and each individual can contribute an outcome to a mortality study once at most; there is no such thing as a longitudinal study of mortality, in the usual sense of the word. Instead, we are forced to derive exposure-response relationships by comparing mortality patterns across groups.

This is complicated by the knowledge that, for most causes of death, the most important determinant of risk is age. Certainly, any comparison of groups must take into account their ages, and if the age distribution differs, then some form of standardisation or adjustment for age must be employed. In the field of air pollution and its influence on mortality, the effect coefficients that are available to us are, thankfully, adjusted for age. In addition, any mortality study whose follow-up period is extensive must take into account the effect on risk of the ageing taking place during the study.

Given that the effects of age and of ageing within a mortality study are well recognised, and that standard analyses accommodate both, it is perhaps surprising that attempts to apply the results of these studies to forward predictions of mortality patterns have been slow to recognise the need to deal separately and simultaneously with age and the passage of time. However, partly due to the IOM's work on systematic and consistent methods for quantification, this is now recognised as an important principle (WHO, 2001).

The quantifications of an HIA require us to take what we know, or what studies tell us, and use it to predict what would happen under various future scenarios defined by possible actions or interventions that might be enacted. In the present context, as we are convinced that air pollution has an effect on mortality hazard rates, we envisage that a change in pollution levels will impact on future hazard rates. It is therefore of direct interest to predict and compare the patterns of life and death under various future scenarios, and these scenarios will be characterised and defined by differing patterns of age- and year-specific hazards of mortality, overall or for specific causes of death.

We have noted before (Hurley *et al*, 2000) that all such impact assessments rest on a sizeable number of assumptions about future hazard patterns, both with and without interventions. Any real-life intervention will necessarily impact on a population that includes a complete range of subjects; and interventions that permanently improve living conditions may be expected to impact positively on the health of cohorts born after the intervention. These factors have driven the development of the IOMLIFET spreadsheet system for doing the calculations, utilising the two-dimensional layout of Table 3.8, to allow separate follow-up of individual cohorts. The format and the layout also offer maximum flexibility for summarising numbers of deaths and life-years lived in almost endless combinations of cohorts affected and calendar period.

We are increasingly convinced that this spreadsheet layout should be the format of choice for HIAs that involve mortality in cohorts. For the present study, they have enabled a set of comparative calculations that give a direct comparison of the total impacts of three quite different sorts of impact. We have not shown the distribution of those impacts across ages,

but another advantage of the spreadsheets is that all the intermediate calculations are available, by age and calendar period, and could therefore be extracted and displayed with great flexibility. We will continue to recommend that these methods be used as the methodology of choice for HIAs involving mortality changes over periods of several months and more.

All of the calculations done for this report applied impacts to a single set of age-, year- and cause-specific hazards considered to represent the average risks over the appropriate sub-populations. We have previously described (Miller and Hurley, 2003) that the IOMLIFET methods can be stratified into sub-populations according to, for instance, individual frailty or susceptibility to disease. If we do not so stratify, then when we compare two scenarios, any difference in the deaths at any point of the follow-up will contribute life years, over the rest of the follow-up, equivalent to the conditional life expectancy at that point. If changes in pollution affected only a specific sub-group, then this assumption might be violated. However, we note both that we have neither markers of susceptibility, nor any direct knowledge of the distribution of frailty within any age-group; and that the coefficients extracted from cohort studies are from populations that presumably included distributions of frailty, and are therefore presumably averages over those distributions.

In this context, it is worth noting that in predicting the life-years saved by eliminating MVTAs, we have taken no account of any possible correlation between risk-taking behaviour involving young males and vehicles, and other types of risky behaviours to which they might be prone, which might lead to higher than average baseline risks for other types of accidents, or smoking- or alcohol-related diseases.

5.2 COMPARISONS OF IMPACTS

The general life-table methodology provides tool by which we can make comparisons of quite different impacts on an equal basis. These are summarised in Section 4.4, and it is clear from that table that the comparisons can look rather different, depending on how the questions are asked, and in particular what population we have in mind when we ask the questions.

In some ways, the questions and the answers are simplest when we consider the impacts on a single birth cohort. From Table 10, we see that the effect of a 6% reduction in all-cause mortality hazard is around 220 days for both males and females. We have previously noted that it is common to observe similar gains from the same changes in all-cause mortality hazard, despite the fact that the underlying hazard rates for males are consistently higher than for females; and that Leksell and Rabl (2001) have shown some theoretical justification for this.

It seems, however, that the equality of results does not always hold true. In the case of MVTA, we should not expect this: since the rates for this specific cause differ very greatly between the sexes, setting them uniformly to zero does not constitute an equal proportional reduction in hazard, and therefore need not produce an equal impact. The predicted impact of eliminating MVTA in males is in fact more than two and a half times that in females, and this fits well with our knowledge of risk taking in young adult males.

When we consider the elimination of passive smoking, again we see a discrepancy in gains between the sexes, although much smaller than for MVTA. This is understandable in terms of the sex differences in cause-specific hazard rates for cardiovascular and lung cancer rates. Since these are both higher in males, the same percentage change in those rates will lead to a higher change in all-cause mortality hazard in males than in females.

Restricting our view initially to males, we see that the predicted effects of eliminating either MVTA or passive smoking are similar, of the order of between two and three months of life expectancy. This compares with around seven months gained by a $10\mu\text{g.m}^{-3}$ reduction in

PM_{2.5} pollution. This result can be rescaled and expressed in a variety of ways: the effect of passive smoking or MVTAs in males is equivalent, on these calculations, to a reduction of 2.5µg.m⁻³ reduction in PM_{2.5}; or the effect of a 1µg.m⁻³ reduction in PM_{2.5} is about 40% of that of eliminating either MVTA or passive smoking.

When we consider predictions for populations composed of sub-cohorts of different ages, similar patterns emerge, but the relative sizes of different effects can differ somewhat. These differences can be seen to be the result of a combination of age-specific hazards and the age structure of the populations concerned. In the current male population, eliminating MVTA has much less effect than eliminating passive smoking because a large proportion are already past the age of highest MVTA risk, but are at the highest risk of ETS-related diseases. The pattern is reversed in new cohorts; however, the comparisons show different relativities from those in a complete birth cohort, because the effect of not following them up completely will omit proportionally more of the lifetime effect of ETS-related diseases than of MVTAs.

These results have arisen from applying an impact coefficient from an influential US cohort study to mortality rates for the population of England and Wales. At present, the US coefficient is generally accepted for use in other countries, but they will have different mortality rates. We expect, however, that the relativities that we have quantified, between impacts from different causes, will be broadly applicable in other countries, particularly if they have similar proportions of smokers in their populations and/or similar traffic patterns. Given suitable input data, our calculations could be repeated for other countries or other scenarios.

These details do not alter an overall conclusion, which we might summarised by saying that the effects of either MVTAs or passive smoking are equivalent to a reduction in PM_{2.5} air pollution of a few µg.m⁻³, the precise amount between 1 and 10 depending on the group in which we are predicting the effect. The inability to specify a single figure is simply a reminder that, in HIA, we must be careful to formulate our questions precisely and in a clearly stated context that includes consideration of what population is to be the base for comparison. What is clear that the effects of 10 µg.m⁻³ of PM_{2.5} pollution are estimated as considerably larger than those of passive smoking or MVTA. The effect of ambient air pollution on mortality is clearly a public health issue of substantial importance.

6 REFERENCES

COMEAP. (2006). Quantification of the effects of air pollutants on health in the UK. Interim statement, 18 January 2006. London: Committee on the Medical Effects of Air Pollutants.

<http://www.advisorybodies.doh.gov.uk/comeap/pdfs/interimlongtermeffects2006.pdf>

ExternE. (1995). DGXII (JOULE Programme) Externalities of Energy, ExternE Project, Report Number 2, Methodology. Prepared by ETSU and others.

ExternE. (1999). DGXII (JOULE Programme) Externalities of Energy, ExternE Project, Report Number 7, Methodology: Update 1998. Holland, M.R. and Forster, D. (eds.).

Hurley JF, Holland MR, Markandya A, Miller BG, Anderson HR, Ayres JG, Donnan PT, Harrison RM, King K, Stedman JR, Stevenson KJ. (2000). Towards assessing and costing the health impacts of ambient particulate air pollution in the UK. Final Research Report submitted to Dept of Health. Edinburgh: Institute of Occupational Medicine.

Hole DJ. (2004). Passive smoking and associated causes of death in Scotland. Edinburgh: NHS Health Scotland.

Hurley F, Hunt A, Cowie H, Holland M, Miller BG, Pye S, Watkiss P. (2005). Methodology for the Cost-Benefit Analysis for CAFÉ: Volume 2: Health Impact Assessment. Didcot, UK: AEA Technology Environment.

IGCB. (2006). An economic analysis to inform the Air Quality Strategy Review consultation. Third report of the Interdepartmental Group on Costs and Benefits. London: Department for Environment, Food and Rural affairs.

<http://www.defra.gov.uk/environment/airquality/strategy/igcb/index.htm>

Jamrozik, K. (2005). Estimate of deaths attributable to passive smoking among UK adults: database analysis. *BMJ*; 330(7495):812.

Krewski D, Burnett RT, Goldberg MS *et al.* (2000). Re-analysis of the Harvard Six-Cities Study and the American Cancer Society Study of Air Pollution and Mortality. Cambridge, MA: Health Effects Institute.

Leksell L, Rabl A. (2001). Air pollution and mortality: quantifications and valuation of years of life lost. *Risk Analysis*; 21(5); 843-857.

Miller BG. (2003). Impact assessment of the mortality effects of longer-term exposure to air pollution: exploring cause-specific mortality and susceptibility. Edinburgh: Institute of Occupational Medicine. (IOM Research Report TM/03/01).

Miller BG, Hurley JF. (2003). Life table methods for quantitative impact assessments in chronic mortality. *Journal of Epidemiology and Community Health*; 57: 200-206.

Pope CA, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Heath CW. (1995) Particulate air pollution as a predictor of mortality in a prospective study of US adults. *Am J Respir Crit Care Med*; 151: 669-674.

Pope CA 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA*; 287:132-141.

WHO. (2000). Quantification of the health effects of exposure to air pollution. Bonn: World Health Organization, European Centre for Environment and Health.

<http://www.euro.who.int/document/e74256.pdf>

WHO. (2006). Health risks of particulate matter from long-range transboundary air pollution. Bonn: World Health Organization, European Centre for Environment and Health.

<http://www.euro.who.int/document/e88189.pdf>

Woodruff TJ, Grillo J, Schoendorf KC. (1997). The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. Environ Health Perspect; 105: 608-612.

<http://ehp.niehs.nih.gov/members/1997/105-6/woodruff.html>

HEAD OFFICE:

Research Avenue North,
Riccarton,
Edinburgh, EH14 4AP,
United Kingdom
Telephone: +44 (0)870 850 5131
Facsimile: +44 (0)870 850 5132

Email: iom@iom-world.org

Tapton Park Innovation Centre,
Brimington Road, Tapton,
Chesterfield, Derbyshire, S41 0TZ,
United Kingdom
Telephone: +44 (0)1246 557866
Facsimile: +44 (0)1246 551212

Research House Business Centre,
Fraser Road,
Perivale, Middlesex, UB6 7AQ,
United Kingdom
Telephone: +44 (0)208 537 3491/2
Facsimile: +44 (0)208 537 3493

Brookside Business Park,
Cold Meece,
Stone, Staffs, ST15 0RZ,
United Kingdom
Telephone: +44 (0)1785 764810
Facsimile: +44 (0)1785 764811